AIDS: A Darwinian Event?

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The HIV/AIDS epidemic is the biggest natural event in the history of our species for the last 500 years. Professor Roy Anderson, who has modelled the likely path of the epidemic, estimates that HIV/AIDS is a 130-year event. This, we contend, is an underestimate. HIV/AIDS has already put an indelible mark on the most affected societies, and that effect will certainly be felt for generations. In addition, Anderson's model—like all such mathematical exercises—measures what can be measured, leaving other factors as hypothetical zeros. The HIV/AIDS epidemic is a complex systemic change in human ecology. It is unleashing secondary impacts that have demographic and epidemiological consequences, which in turn create feedback loops into the dynamics of the epidemic itself.

HIV/AIDS is certainly an historic event. It may also be a 'Darwinian event'. We argue that historians have two particular responsibilities with regard to this epidemic. Firstly, history should provide us with ideas, paradigms and methodologies for understanding and responding to the disease. Secondly, there is an awful predictability about HIV/AIDS and what it has the potential to do. Historians have the experience of seeing an event of unparalleled significance unfold before their eyes. To some extent this history can be written in advance as a wake-up call as to what might happen. Certainly there must be lessons from the past that we can apply to this epidemic.

To date historians have not generally engaged with the HIV/AIDS epidemic, and we are not sure why—perhaps because thinking has been premised on the assumption that it is 'just another epidemic'. Comparisons with the Black Death abound. This can mislead. AIDS won't kill such a large proportion of the global population as the 14th-century catastrophe, nor will it kill with the same speed. In some settings, however, mortality may over time approach that experienced in parts of Europe. There are two significant differences: AIDS mortality will take place over decades, so mortality of any given cohort will exceed levels experienced in all but history's most severe demographic disasters; and AIDS selects for age and gender, the majority of those dying being between 25 and 50, with more women than men falling victim.

In this article, however, we shall focus primarily on the idea of AIDS as an event of such magnitude and with such implications that it assumes the proportions of a 'Darwinian event'. What does it mean to describe something as a Darwinian event? It is a provocative but, we hope, a Darwinian is intentional.

† Before 1845 near Birmingham peppered moths were primarily light-coloured, but some had darker wings and were called the melanistic or carbonaria forms. Before the industrial revolution birds ate the darker moths—they were easier to see. In the 1850s, about 98% of uneaten peppered moths were the light variety. Industrialisation meant that trees darkened and so birds more easily ate the light moths. By the 1950s, 98% of the peppered moths were the dark variety. Some argue this is a 'proof of evolution'; others say there can be light- peppered moths and dark-peppered moths—but they are all still peppered moths, variations within a single species.

* We are well aware of the rich debate about Darwin and Darwinism elegantly evidenced in the writings of Stephen Jay Gould, Stephen Pinker, Richard Dawkins and many others, and the existence of Darwinian discussion groups such as that at the London School of Economics (http://www.lse.ac.uk/Departments/CPSS/darwin/). Our use of the term Darwinian is intentional.

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AIDS is the acquired immunodeficiency syndrome, the name given to the grouping of diseases caused by the human immunodeficiency virus (HIV). The disease was first recognised only in 1981.

Darwin means that process of evolution by natural selection first described by Charles Darwin. Here we mean that there is a struggle for survival in each generation; there is individual variation in the population; and the variation has a hereditary component. All this means that there will be gradual variation in a population with those characteristics associated with greater survival ability dominating.

An event is shorter term than a process. In this context an event is something where we can clearly say ‘this happened and had these effects’. The classic example of a Darwinian event is the pollution arising from the industrial revolution that resulted in the dark form of peppered moths (Biston betularia) predominating over the light form.

The Darwinian framework provides a powerful set of hypotheses for the evolution of both the HIV/AIDS epidemic
and human responses to it. It raises far more issues than can possibly be addressed in a short chapter. At this point, the most that we can attempt is to put forward our ideas and hypotheses to stimulate debate, inspire disciplines new to the study of the epidemic to research the epidemic and its consequence, and postulate the value of this approach to understanding the HIV/AIDS pandemic. We hope historians will be among those who engage.

THE FRAMEWORK FOR ANALYSIS

Here we shall pursue the Darwinian hypothesis to its limits. This requires us to apply versions of universal Darwinism to social phenomena. This is not a resuscitation of social Darwinism, but a wider application of Darwinian principles, as they are currently understood in the scientific community. We recognise that this is hard to grasp in dealing with a disease like AIDS, and all the baggage it carries – as Dawkins says, ‘nature is not cruel, only pitilessly indifferent. This is one of the hardest lessons for humans to learn. We cannot admit that things may be neither good nor evil, neither cruel nor kind, but simply callous - indifferent to all suffering, lacking all purpose.” Let us begin by identifying different possible interpretations and then following through on the logic.

1. The principle of natural selection is the most famous of Darwin’s ideas, and the subsequent discovery of genetic inheritance and DNA means that this too is closely identified with genetic selection. A ‘Darwinian event’ could be one where those that pass on their genes are selected for certain genetic traits. The converse is that those who die before they breed successfully are also ‘selected’: they are selected out. This is the ‘survival of the fittest’ scenario. We will ask, below, whether HIV/AIDS fits this scenario. We would note here one important point: survival of the fittest does not mean survival of the strongest or most ‘intelligent’, it means survival of the best adapted.

2. Darwinism is commonly associated with biology’s replicator, genes. However, natural selection is a principle of evolution and not a specifically genetic theory. Any systematic encoder of information that has the traits of replicability, fecundity and durability, along with a degree of imperfection or variation in offspring, can serve as a Darwinian replicator. While genes are biology’s replicator, memes are a ‘second replicator’ operating where there are human brains that can host them. Any significant cultural or technological change (especially with regard to communications) has an impact on the replication and hence ‘evolution’ of memes. Within this vast area, we focus on memes that have a direct impact on survival and reproduction, and higher-level complex social memes such as religions and organisational systems.

3. Lastly, we have to look at Homo sapiens not just as an organism but also as part of an ecological framework.

Thus, a ‘Darwinian event’ could be a change in the ecological or evolutionary framework within which Homo sapiens exists. We exist not just as a species but as part of the web of nature, a web that includes the entire biosphere from microbes to ecosystems – and also our social environment. Under this ‘ecology change’ scenario, a ‘Darwinian event’ for humankind can be a change in that framework that affects us, leading us into a new collective adaptation. We will see that any genetic or memetic adaptation is necessarily an ecological change.

This paper will study each of these in turn. Each provides a fruitful point of departure for understanding the likely trajectory of the HIV/AIDS epidemic and assessing if it is indeed a Darwinian event.

WHY HIV?

HIV is a retrovirus, meaning that it is one of the first known viruses to transcribe DNA (deoxyribose nucleic acid) from an RNA (ribose nucleic acid) template. In order to exist, the virus has to enter a cell and insert itself into the cell’s DNA to reproduce itself. HIV is hard to transmit. The fact that heterosexual transmission is the main way in which HIV is transmitted, followed by mother-to-child transmission, may have far-reaching implications. Evolutionary theorists argue that our primary function is to reproduce and our existence is geared towards this end. We do not explore this but suggest it deserves attention.

HIV is a simple virus, replicates rapidly and so can mutate equally rapidly. People are infectious for many years, but most do not know that they are infected. A pathogen must transmit its progeny from one host to another. Success may come from taking a long time to disable the host, giving plenty of time for contact with other potential victims; surviving for a long time outside the body; and being easily transmissible. HIV falls in the first category.

AIDS will not wipe out people in the way Western diseases killed the ‘immunologically naïve’ indigenous populations of the Americas, Australasia and parts of Africa from the middle of the last millennium. Lacking defences against common European diseases such as smallpox, typhus, measles and influenza, these populations fell ill faster and diseases were more virulent. The result was massive depopulation – whole peoples disappeared, and others were so seriously depleted as to have been written out of history.

The role of disease in human history has been charted by a number of authors: initially by McNeil, who argues that disease is key, and more recently by Diamond, who sees disease as part of broader geographical determinism. The interaction of disease, famine and political crisis has also been investigated, notably with regard to the ‘late Victorian holocausts’ that overtook the emergent Third World in the last century.

* The science around the AIDS virus is clearly explained in many books and articles, one of the most accessible being Christopher Wills, Plagues (London, Flamingo, 1997).
From these experiences we can identify a nexus of complex disaster, with different factors including climate, epidemic disease, conflict and social disorder all reinforcing one another to cause a decline in life expectancy and population stagnation and, in some cases, population collapse. These are cases of populations under severe stress, but how selection pressures might operate so that we could fairly describe such events as 'Darwinian' remains speculative.

It is clear from the history of epidemics that there are some populations at sub-regional, regional or continental level that are more susceptible to infection. There are also subtypes in a population who are more likely to be infected. Does this also apply to HIV/AIDS? If it does, then the argument that AIDS is a Darwinian event is supported.

It has been suggested that at the population level those whose ancestors experienced the plague may be genetically resistant to HIV. The plague bacillus attached to the same receptor on the cell that HIV attaches to. If this were the case, then mapping the plague and movement of people whose genes carry this measure of protection would allow us to predict where the most serious HIV epidemics will and won’t be experienced. It might help explain why the genetically naïve populations of sub-Saharan Africa are experiencing such a severe epidemic.7

Recently AIDS appears to be increasingly a disease of the poor and marginalised. What this means for humankind needs to be explored. We don’t know if the disease will have a greater impact on certain groups – are entrepreneurs in business and political leadership also more likely to be entrepreneurial in their sex lives? Will HIV disproportionately affect them, and will this have any impact on our gene pool? In work done as a scenario building exercise for Shell South Africa we noted that some populations were experiencing higher levels of infection than others, with people in particular areas and particular occupation groups at high risk. Newly prosperous people are often at risk, as are sex workers, mobile workers, soldiers, miners and others in hazardous occupations, as well as the unemployed. Those who habitually think only in the short-term are at high risk. Fig. 1 was developed in a scenario planning exercise for Shell South Africa.

Is there any validity in this? Those who survive the epidemic long enough to breed successfully create the future gene pool. As Dawkins notes, ‘To the extent that differences between individuals are due to genes (which may be a large extent or a small one), natural selection can favour some quirk of embryological origami or embryological chemistry and disfavour others.’8 A characteristic influenced by genes – thinking in an entrepreneurial manner or being sexually attractive – can be favoured or disfavoured by natural selection. If it increases the chance of passing the genes on (through successful breeding), then these genes may be passed on, at the expense of others.

**THE DEMOGRAPHIC IMPACT OF HIV/AIDS**

We would argue that AIDS is a Darwinian event because of its demographic impact. It is a demographic shock. Demographic consequences are felt in a number of ways, but most immediately through increased mortality and decreased fertility. In a scientific meeting on the Demographic and Socio-
Economic Impact of AIDS, in Durban in 2003, available evidence was assembled and assessed. 'In the worst affected countries the probability of a 15-year old dying before reaching the age of 60 years has risen dramatically, from a range of 10 to 30% in the mid-1980s, to a range of 30 - 60% at the turn of the century.'

**POPULATION SIZE AND STRUCTURE**

Mortality rises among those infected. Five community-based studies in East Africa found that mortality among infected adults was 10 - 20 times higher than among uninfected adults. Child mortality rises both among HIV-positive children, infected through mother-to-child transmission (in the absence of treatment most HIV infected children die before their fifth birthday - Newell et al., quoted by Zaba et al.), and among HIV-negative children who have an infected mother. A summary of the key demographic impacts of this disease is shown in Table I. These data are taken from the normally conservative United Nations.

Only in the very worst-affected countries (the UN report specifically mentions Botswana, Lesotho and South Africa) is the population expected to start declining after 2005. This is due to increased mortality, reduced fertility and the disruption of society. Infected women are less likely to fall pregnant and carry a child to term, and premature mortality means there will be fewer women of childbearing age. For Uganda it was estimated that the number of births was reduced by approximately 700 000, corresponding with almost 5.9% of all births that would have occurred during the last two decades.

Cohort mortality may be very high. Fig. 2 looks at life expectancy for today's 15-year-old boys in a number of African countries.

According to these conservative analyses, in countries where 15% of adults are currently infected, around a third of today's 15-year-olds will die of AIDS. Where adult prevalence rates exceed 15%, the lifetime risk of dying of AIDS is much greater.

In South Africa the Medical Research Council has tracked the steady increase in deaths using the death certificates collected by the South African Department of Home Affairs. South Africa is one of the few countries where there is death registration and in 2000 over 90% of adult deaths were registered. Between 1998 and 2003 there was a 150% increase in deaths of women aged 20 - 49 years, and this is adjusting for population growth and possible improvement in registration.

An increase in mortality is also recorded for young people. In Botswana under-5 mortality is expected to have risen to 104/1 000 live births; in the absence of AIDS it was projected to decrease to 45/1 000 live births. AIDS causes the majority of these ‘extra’ deaths (pp. 2 - 15).

The structure of the population will change both in terms of age cohorts and in gender ratios. Life expectancy for women is worse affected than that for men. We know that there are more women infected than men, in sub-Saharan Africa women are 30% more likely to be infected than men, and this is even more marked at younger ages. A 15 - 24-year-old woman is 3.4 more times more likely to be infected than her male counterpart.

**Orphaning**

The increase in orphaning is a demographic impact, but it will have social and economic consequences. UNICEF estimates that by 2010 20 million children in Africa will have lost one or both parents to HIV/AIDS. However, it is not just the numbers that are important. We also need to look at affected children as a percentage of all children. UNICEF estimates that in some settings up to 25% of children may be orphaned. For over 80% of orphans in the worst-affected countries the cause will be

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**TABLE I. SUMMARY OF ESTIMATED AND PROJECTED IMPACT OF HIV/AIDS ON MORTALITY INDICATORS**

<table>
<thead>
<tr>
<th>Indicator</th>
<th>53 countries where HIV/AIDS impact included in 2002 UN estimates*</th>
<th>7 countries with prevalence &gt; 20%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of deaths (millions)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without AIDS</td>
<td>159</td>
<td>174</td>
</tr>
<tr>
<td>With AIDS</td>
<td>170</td>
<td>207</td>
</tr>
<tr>
<td>Percentage difference</td>
<td>7</td>
<td>19</td>
</tr>
<tr>
<td>Life expectancy at birth (years)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without AIDS</td>
<td>63.9</td>
<td>68.4</td>
</tr>
<tr>
<td>With AIDS</td>
<td>62.4</td>
<td>64.2</td>
</tr>
<tr>
<td>Percentage difference</td>
<td>2.4</td>
<td>6.1</td>
</tr>
<tr>
<td>Child mortality rate (per 1 000)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without AIDS</td>
<td>93.9</td>
<td>68.8</td>
</tr>
<tr>
<td>With AIDS</td>
<td>98.8</td>
<td>75.8</td>
</tr>
<tr>
<td>Percentage difference</td>
<td>5.3</td>
<td>10</td>
</tr>
</tbody>
</table>

* These countries are listed in the report and include the USA, Russian Federation, India and China.
HIV/AIDS. These children face severe stress, they are less likely to attend school, more likely to be exploited and experience premature mortality, and they also have a more pessimistic outlook on life. Life expectancy and child mortality rates have been widely used as markers for improvements in the welfare of populations. In Botswana, life expectancy at birth is now estimated to be 39 years as opposed to 71 without AIDS. We would argue that demographic impact of this magnitude is certain to have far-reaching economic, social and political consequences. Will these also be Darwinian? Perhaps; it is certainly something we need to consider, and it would merit thought by other disciplines.

Survival of the fittest?

Human beings evolved in Africa several hundreds of millennia ago. Our genome evolved in response to the constraints and opportunities of that environment. Indeed, the entire theory of evolution is premised on historical contingency and the possibility of novelty. There can be a new species, and there can also be an extinction, and even a single new species or extinction can reshuffle the whole deck of phenotypical* cards.

Could a pathogen such as HIV wipe out the entire human race? The theoretical possibility is there, but such a pathogen would have to possess remarkable microbial and epidemiological characteristics. Currently, neither HIV nor the headline-hitting Ebola and SARS fit this bill. Notable is that disease is given short shrift in Martin Rees’ doomsday prognoses for the human race. Rather, Rees sees our main collective threat as our own technology run amok, or sufficiently abusing the planet as to make it uninhabitable.

One implication of the recent character of our common ancestry is that as a species we lack genetic diversity, and hence suffer greater collective susceptibility to infectious diseases. Most epidemic infectious diseases have emerged since the invention of agriculture in the last 10 000 years, associated with population density and proximity to domestic animals. This is a very short time period for genetic selection to operate.

* The visible characteristics of an organism resulting from the interaction between its genetic makeup and the environment.
For various reasons, HIV seems especially unlikely to succumb to evolutionary pressure for lower virulence. For a pathogen to be sustainable in a population, for every currently infected host there must be a greater-than-one chance of infecting a new host. In epidemiologists' algebraic parlance, $R_0$ must be greater than one. For many infectious diseases such as measles, cholera and whooping cough, $R_0$ is in the tens. For HIV, it is only slightly above one. The disease has a high degree of lethality and the host is infectious for many years, so this low transmission ratio is sustainable. HIV also has a high rate of mutation and recombination, and uses the human immune system to replicate itself. In summary, this means that 'HIV may be evolutionarily free of constraints that reduce its virulence and increase its susceptibility'.

In theory HIV could reach saturation in a host population, infecting and killing every individual, without jeopardising its onward transmission. In such a process, in the host population there could be selection for the genetic traits that make for slow progression from HIV to AIDS. But this is unlikely to be significant because of the mode of transmission and the fact that the virus is not easily transmitted. In most populations there will be those who are not exposed to infection (don't have sex, stick to one partner who sticks to them) and those who will simply not be infected (30% of couples are discordant, meaning that one is infected and the other is not). Any selection pressures would take generations to work through.

Because of the unique way in which HIV causes immunosuppression, it also has other implications for the evolution of pathogens and their human host. A human population with a large proportion of immunocompromised individuals is a new ecosystem for infectious diseases, marked by greater ease of transmission of infectious agents, and lesser vulnerability to the human immune system. The current pandemic of TB is an example of this, but there is no reason why epidemics of other infectious agents should not also seize upon this opportunity. Given the normal functional relationship between transmissibility and virulence, this would imply that infectious agents would (mostly) evolve in the direction of higher transmission levels but lower virulence.

Concluding this tour d’horizon of the genetic evolutionary implications of HIV/AIDS, it is evident that the pandemic has intriguing, novel and potentially far-reaching impacts. Some models for the impacts of HIV/AIDS imply immense adverse outcomes, and others imply modest impacts. So far, empirical data to substantiate models are scarce. They are novel, because of the peculiar capacity of HIV to retain its lethality, which means that we face the need for a new kind of accommodation to a pathogen. They are far-reaching, because HIV/AIDS will be with us for generations, and it may change our entire disease environment.

**‘MEMETIC EVOLUTION’**

What are ‘memes’? The human brain has the capacity for receiving, encoding and transmitting information. Replicable bits of information are ‘memes’. Insofar as the packets of information have a capacity for replication (like genes themselves), they will do so, and thus become replicators along a new dimension. Having a capacity for variation and recombination, they are themselves able to evolve in a Darwinian manner. Simple examples of memes are tunes, games and the skills necessary to make shoes; examples of complex combinations of memes (or memeplexes) are religious beliefs. There is no special reason why DNA should be the sole Darwinian replicator: any information coding system can do it.

Susan Blackmore describes the gene-meme relationship as like a man walking a dog: at first the dog (memes) is on a tight leash, but increasingly it is able to steer its former master, so that it is not clear who is in charge. Memeticists describe the relationship between memes and genes as purely an analogy, but it is one that allows us to lend some analytic rigour to the interaction of society and biology in the context of HIV/AIDS. Also, insofar as we are exploring the limits of HIV/AIDS as a ‘Darwinian event’, the least we can do is give the hypothesis the best run for its money.

This section analyses two memes that are especially relevant to the case of HIV/AIDS: male circumcision and risk-taking.

**MALE CIRCUMCISION**

An instance of a meme that features prominently in the literature is male circumcision. This was cited by Richard Dawkins in his introduction to Susan Blackmore's The Meme Machine, with reference to his old school:

'[A] Martian geneticist, visiting the school during the morning cold bath ritual, would have hesitatingly diagnosed an "obvious" genetic polymorphism. About 50 per cent of the boys were circumcised and 50 per cent were not. The boys, incidentally, were highly conscious of the polymorphism and we classified ourselves in Roundheads versus Cavaliers... It is, of course, not a genetic but a memetic polymorphism. But the Martian’s mistake is completely understandable; the morphological discontinuity is of exactly the kind that one normally expects to find produced by genes.

In England at that time, infant circumcision was a medical whim, and the Roundhead/Cavalier polymorphism at my school probably owed less to longitudinal (inter-generational) transmission than to differing fashions in the various hospitals where we happened to have been born - horizontal memetic transmission, yet again. Where circumcision is religiously or traditionally based, the transmission will follow a longitudinal pattern of heredity, very similar to the pattern for true genetic transmission, and often persisting for many generations. Our Martian geneticist would have to work quite hard to discover that no genes are involved in the genesis of the roundhead phenotype.'

Unlike, for example, tattooing or navel piercing, circumcision is a meme that is less often widely transmitted horizontally.

This particular meme is important to our case because male circumcision appears to protect against HIV transmission by as much as a 40% reduction in risk. In short, it is a meme that has come to provide a very clear survival advantage to its possessors. Until now, its replicatory capacity has been related almost wholly to its attachment to certain religious and cultural memes or 'memeplexes'. But in the era of AIDS, circumcision now also provides a survival advantage.
This is of both theoretical and practical interest. The protective effect of male circumcision should logically result in populations that practise it suffering lower HIV rates than those that do not, and thus surviving better. It could also result in the meme of male circumcision itself being adopted by other populations - i.e. spread horizontally. Also relevant is to ask what determines circumcision in a society. The circumcision meme is closely associated with religious beliefs, and these may in turn be associated with other social practices that are correlated with HIV risk. Examples might include polygamy and early marriage of girls.

**RISK-TAKING**

Another memetic trait is propensity to take risks. In the field of sexual behaviour, this has been studied, concluding that there is a genetic basis to unfaithfulness to one’s partner. But it is also a gene-meme interaction, with some individuals or subgroups more prone to risk-taking than others on the basis of their genetic profile.

In the absence of HIV/AIDS or a similar STI, risk taking in the field of sexual encounters, especially numbers of sexual partners, is genetically adaptive. Men who have higher numbers of partners will have more offspring. Women who have illicit affairs are more likely to select those kinds of men as lovers, and to conceive children as a result of such casual flings, rather than through regular sexual intercourse with their long-term partners, and as many as 10% of all children have illicit affairs are more likely to select those kinds of men as lovers, and to conceive children as a result of such casual flings, rather than through regular sexual intercourse with their long-term partners, and as many as 10% of all children in Western societies are not in fact fathered by their ‘fathers’.20

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There is some historical-anthropological evidence on which to build a theory of memetic change. Many Melanesian and some native American societies were, in Marshall Sahlins’ formulation, ‘Aphrodisian’ in their traditions of sexual generosity, including routinely offering sexual favours to guests, traders and diplomats. The arrival of Western explorers, missionaries and traders unleashed catastrophic epidemics on these societies, including syphilis. The low fertility, high mortality, and the sheer disfigurement and suffering caused by the disease contributed to population plunges and a thorough-going social demoralisation.21 These societies simply could not adapt their sexual practices quickly enough to deal with threat of STIs.

In the era of AIDS, sexual risk-taking undoubtedly increases risk of HIV, and as such negatively selects for survival. This is a new phenomenon in the history of our species. As a result, we can anticipate a new dynamic between natural and sexual selection.

Christian fundamentalists are, strictly speaking, correct in advocating that the A and B of AIDS prevention (abstinence and faithfulness to one lifetime partner) are sufficient, spurning the C for condoms. In the absence of condoms or other preventive technologies, over the generations, those who faithfully pursue the A and B would survive, while the rest of humanity would become steadily less numerous. The efficacy of A and B would be strongly associated with their connection to other memetic factors, notably religious faith, implying that such a hypothetical society would be much more religious.

**IMPLICATIONS**

The cases of potential memetic ‘evolution’ in response to HIV/AIDS point to the complexity of the impacts of the epidemic. No part of human life is untouched. Some memes have clear advantages or disadvantages for survival – and may as a consequence be selected for. How long this will take, we cannot speculate. The impacts of the epidemic compel human populations to explore elements of their wired-in behavioural capacities that have not recently been in evidence (such as selfishness under energy stress). Other memes have their own autonomous logics, which may prevail despite their mal-adaptation to the realities of the epidemic.

**‘ECOLOGY CHANGE’**

The preceding discussion has necessarily veered away from simple evolutionism into examining the role of individual human host, human society and the HIV in a complex framework that encompasses all. Any change to one part of the system necessarily impacts on others. To this extent, many ‘ecological’ issues have already been addressed. Others follow. For example, as Barnett and Blaikie observed in Uganda in the late 1980s, HIV/AIDS causes changes in land use, which in turn have other consequences for agrarian society.22 This is an example of the way in which it is necessary to analyse HIV/AIDS as an historic event.

The question that remains is, how do HIV’s adaptation to its host and Homo sapiens’ response to HIV interact with one another? What future will be jointly created by our genes and memes, and HIV and its parallel and inter-linked evolution?

Let us begin with HIV, as it is in the driving seat. As mentioned, HIV has unique evolutionary features, which enable it to bypass or render ineffective the pressures for lower virulence. This occurs at various levels, from the microbial to the collectivity of the host population. It is a fractal characteristic. At the population level, the key factor is the 7 - 10-year time lag between HIV infection and the development of AIDS, which enables R to be sustained without loss of lethality.

We know that HIV is evolving. It does this by mutation and recombination, creating new strains that have differing transmissibility, virulence and vulnerability to treatments. The most likely outcome of genetic change in HIV is to increase resistance to treatment and to increase R0 by increasing the transmission rate. In populations already at saturation level (above 40% adult prevalence) the latter might be a maladaptive trait, insofar as a new variant of HIV that achieved still higher levels of prevalence among young women would actually begin to create a below-replacement fertility regimen in the host population. In evolutionary terms, this is a credible scenario.

Turning to the human response to HIV, we can identify three main types of reaction. The first is the impact of the epidemic on social functioning, including poverty, food security and social reproduction. Aspects of this have been explored above.
An additional element to the ‘new variant famine’ hypothesis, first put forward by de Waal and Whiteside, is the way in which responses to the stress of hunger and destitution become maladaptive in the context of HIV/AIDS. This is because survival strategies such as migration or commercial sex work increase the risk of HIV transmission. This is perhaps the most alarming implication of the ‘new variant famine’ hypothesis: that it has a feed-back loop into HIV transmission itself, helping to sustain high prevalence. If it occurs – and so far we have only anecdotal evidence that it does – this would be a far-reaching revision to the Roy Anderson projection of AIDS as a 130-year event.

Another potential feedback loop is the adverse impact of lower levels of girls’ education on HIV risk. Educational performance is declining because of children (especially girls) being withdrawn from school to help with sustaining the household, and because of the lower standards of schools that are stricken with high educator morbidity and mortality. Low female educational achievement is in turn associated with higher risk of HIV.

It is a commonplace of population history that a demographic shock such as a famine or war creates greater vulnerability to a second shock such as an epidemic. Disasters rarely come singly. What is different about HIV/AIDS is that it is a long-wave event, with structural implications for human ecology, which in turn implies that its secondary impacts will themselves be structural and sustained.

We could, in short, see a new viral-human ecological framework in which HIV saturation combines with poverty, hunger and social dislocation in a new kind of socio-epidemiological trap. Populations that pass a threshold of compounded distress may simply be unable to recover. In a globalised world, ambitious individuals can escape and live elsewhere, bringing up their families in richer low-HIV societies, leaving the poorest and most entrapped populations to continue a downward spiral of misery. A bifurcation of global life chances along these lines, determined in large part by HIV prevalence, cannot be ruled out.

The second human response to HIV is the medical-scientific response. In two decades, scientists have learned more about HIV than almost any other pathogen. But, in the absence of a magic bullet, what has this meant for the virus? History counsels us to be cautious about the impact of human intervention – including medical science – on the life of a pathogen.

Some of the medical responses to HIV/AIDS, such as antiretroviral therapy (ART), may in fact prove to be adaptive for the virus. This would happen if ART extends the infective period of the person living with HIV, and thereby increases the number of his or her risky sexual encounters, by a greater degree than it reduces infectivity. If the proportional increase in the life expectancy of people living with HIV/AIDS from the moment of infection on ART is A, and the proportional decrease in infectivity (through lower viral load) is B, then if A exceeds B, then R0 increases. Note that this assumes no change in sexual behaviour: simply living longer implies more sexual partners. In addition, there are some indications that the availability of ART may also increase risky behaviour, because HIV becomes seen as a treatable condition. On the other hand, the voluntary counselling and testing associated with ART provision may lead to behaviour change in the opposite direction, lowering transmission.

ART is not, therefore, a block to the odyssey of HIV. It brings many desirable outcomes, such as prolonging the lives of those living with HIV and AIDS, and potentially blunting some of the disastrous secondary impacts such as reducing the numbers of children orphaned by AIDS. But this is managing or containing the epidemic, not halting it.

The third human response is what we may call the ‘political economic’ response: the whole cluster of institutional measures put in place to manage and (optimistically) solve the problem posed by HIV/AIDS. International institutional responses have their own independent logic, driven by the need to appear to be managing the problem in such a way that the institutions themselves are protected and can reproduce themselves.

Scanning human responses to HIV/AIDS, what we see is the extent to which we have already adapted to accommodate the killer in our midst. We are sharing our habitat with HIV, and our society, institutions and even our prized science are quietly accommodating this specialist predator.

CONCLUSION

In June 2004 the Copenhagen Consensus project released their list of priorities for the ten great global challenges. These challenges, selected from a wider set of issues identified by the United Nations, are: civil conflicts; climate change; communicable diseases; education; financial stability; governance; hunger and malnutrition; migration; trade reform; and water and sanitation.23 The panel of economic experts was asked to address this and decide the best ways of advancing global welfare, especially that of developing countries, supposing an additional $50 billion of resources was available. The AIDS epidemic was identified as the first priority: ‘The panel assigned the highest priority to new measures to prevent the spread of HIV/AIDS. Spending assigned to this purpose would yield extraordinarily high benefits, averting nearly 30m new infections by 2010. Costs are substantial, estimated at $27 billion. Even so, these costs are small in relation to what stands to be gained. Moreover, the scale and urgency of the problem – especially in Africa, where AIDS threatens the collapse of entire societies – are extreme.’23

Writing in 1987, when the implications of the HIV/AIDS epidemic were first being absorbed by the scientific community, the evolutionary theorist Stephen Jay Gould wrote:

‘The evolutionary perspective is correct, but utterly inappropriate for our human scale. Yes, AIDS is a natural phenomenon, one of a recurring class of grand catastrophes, but it is a self-sustaining one, one that will not end until we end it. This is an extraordinary event in human history: the first time that we are faced with a directly transmitted pathogen that is not itself a predator of the human species, but rather a specialist predator of one particular Homo sapiens. It is simply not the case that what happens to this species will not have a profound effect on the human story. This is the first virus that has managed to get itself into the species, and the genie has been let out of the bottle.’24

Sure enough, the virus has its foot on the human species, and it is anyone’s guess how this will play out. The history of this and other epidemics teaches us that we cannot predict the outcome, and that we are likely to be surprised and often wrong. It is better to think of HIV/AIDS as a 130-year event – even if we reduce the limits of human intervention.
of pandemic diseases. Yes, AIDS may run through the entire population, and may carry off a quarter or more of us. Yes, it may make no biological difference to Homo sapiens in the long run: there will still be plenty of us left and we can start again. Evolution cares as little for its agents - organisms struggling for reproductive success - as physics cares for individual atoms of hydrogen in the sun. But we care. The atoms are our neighbors, our lovers, our children, and ourselves. AIDS is both a natural phenomenon and, potentially, the greatest natural tragedy in human history.24

Seventeen years later, we have no reason to dispute his verdict that we care, but there is evidence stacking up to suggest this may make a biological difference to Homo sapiens.

We asked if HIV/AIDS was a Darwinian event. We looked at the evidence and suggested that its demographic consequences were such that, for this reason alone, it merits this description. However, it has social and economic consequences as well.

This paper was originally presented at a historical conference. We concluded that AIDS is an event of historical significance, not least because we must respond to it in a (short) historical time frame. It is therefore high time that new disciplines become engaged in looking at the epidemic, drawing comparisons from other epidemics and demographic disasters, and applying frameworks from evolutionary science. Historians can both contribute to the debate and help us influence our future. George Orwell wrote: ‘He who controls the past commands the future. He who commands the future influences our future.’

We conclude that AIDS is an event of historical significance, especially if we consider the social and economic consequences. We suggest that historians contribute to the debate by examining comparisons from other epidemics and demographic disasters and applying frameworks from evolutionary science. This paper was originally presented at a historical conference.

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