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Sex and death: old cliché, new reality*

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Demographers have traditionally been preoccupied with human reproduction and mortality, and with the links, at different levels of aggregation, between the two. Today, when in some regions of the world sexual intercourse carries with it the risk not just of pregnancy but of fatal infection with the human immunodeficiency virus, that traditional preoccupation acquires a special urgency.

The title of this paper comes from a statement that is well known to many demographers, especially those who studied no later than the 1970s. That is why I have called it, although perhaps this is unfair, an *old* cliché. The statement's source is an account published by Hollingsworth[1] of the varied data sources that lend themselves to the study of historical demography. The statement, which introduces the book, is the first sentence of the introduction:

> Demography … has the same ingredients as all the most popular stories: sex and death.

As an apprentice demographer I was struck by Hollingsworth's statement. It made demography sound not just interesting, or even racy, but important.

Of course, even in my naiveté I realized that Hollingsworth was using 'sex' not to mean sexual activity, what Raymond Pearl, one of the founders of biometry and Heath Clark lecturer of 1937 referred to as 'the congress of the separate sexes'.[2] Rather, Hollingsworth was using 'sex' to mean fertility, which is the term demographers use for reproductive achievement (in contrast to the medical usage of reproductive potential). Even so, the subject of birth and death, and what comes in between, is a big one. Moreover, the linking of the two, sex and death, has traditionally been a major preoccupation of demographers.

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This preoccupation is formalized in the descriptive paradigm of what is called the demographic transition, the process whereby populations move from a condition of high and approximately compensating birth rates and death rates to one of low and approximately compensating birth and death rates. Both the initial and final stages are characterized by low rates of population growth. In contrast, because death rates tend to decline before birth rates, the intermediate stage is characterized by higher rates of population growth, often dramatically so. It is this intermediate stage, which is still being played out in most poor countries around the world, that has so gripped the attention of population scientists, development economists and family planners since the 1960s.

This thumbnail sketch of the historical demographic transition fails to capture at least two important elements. The first is that there was no homogeneity in the individual demographic transitions of the countries of today's industrialized world. It now appears that it was in the middle of the 18th century that mortality began to decline in Britain, France, Scandinavia and North America. The timing of fertility decline, however, was extremely variable. The earliest fertility decline, for example, took place in the latter half of the 18th century, in France; but did not begin to fall in England until another century had passed.

The second omission is any sense of what the demographic environment used to be like. In a poor, rural parish in northern Sweden, for example, mortality rates between 1750 and 1785 were such that as many as one-quarter to one-third of infants did not survive to their first birthday (one quotes infant mortality figures because they dominate the ultimate life table). By 1850, however, only one in ten infants died before their first birthday: I use the word 'only', because one dead out of ten is a dramatic improvement over two or three out of ten. Even 50 years later, at the dawn of the 20th century, very few countries recorded infant mortality that was as low. Today, of course, the world's rich countries record levels of infant mortality that are one-tenth to one-twentieth this level, between five and ten dying per thousand, and the brutal levels of a century ago — not to mention those of earlier times — are forgotten.

The crude empirical generalization of the demographic transition becomes a theory when causes are sought for the movements in death and birth rates. The classic Princetonian formulation of the 1940s singled out 'modernization' as the force driving the decline in death rates and birth rates. But in that more self-confident era the theory's formulators did not feel the need to define what was meant by 'modernization'. Presumably, it was whatever had created the economic and demographic conditions of the United States of the time. In actual fact, our understanding of what drove down mortality rates in the first place is still imperfect.

When the theory of the demographic transition was first formulated, it was felt that a declining death rate might be a necessary precondition for a declining birth

rate. Indeed, the notion that mortality decline might drive fertility decline has become almost an article of faith among demographers, even though empirical demonstrations of a causal link, rather than a merely temporal one, are startlingly rare. There are various explanations for this link. One set of explanations hinges around economic factors. For example, lower morbidity and mortality and a healthier population are likely to be associated with improved living standards, although whether improved living standards were a cause of falling mortality rates, or an effect of them, is difficult to say. In either case, some theorists would argue that improved living standards might lead to a reduced demand for children.[3]

Another set of explanations for how mortality decline may trigger fertility decline requires that couples have target numbers of surviving children that they do not want to exceed, and also that couples have the means to prevent births.[4] Then, if couples recognize that more children are surviving in general, they may be led to produce fewer children in the first place in the expectation that a greater proportion will survive; this is an argument at the aggregate level. Alternatively, at the individual level, with declining mortality couples may be able to achieve their own particular target number of surviving children with fewer numbers of births; put another way, their compulsion to 'replace' dead children may be activated less frequently. However appealing these scenarios, the assumptions on which they are predicated — the existence of fertility targets that couples do not want to exceed, and the use of birth control to prevent these targets being exceeded — are very strong.

It is sadly true that, although there is still much to learn, the quest for causal links between mortality and fertility is no longer a central part of the demographic enterprise. One contributing factor may be that contemporary demography receives more attention than historical demography, and that throughout the developing world the mortality decline that was initiated after the Second World War by mass vaccination, provision of safe water, antibiotics and so on, was easily explicable. Indeed, this second mortality decline was engineered deliberately, and rapidly, by various means, some of which played no part at all in the pre-20th century mortality transition. Fertility decline, in contrast, was far more idiosyncratic and, as a result, fertility attracted far more attention than mortality. Moreover, the emphasis on fertility reduction in the Third World may have come about simply because of the contradiction between the goals of reducing population growth rates and reducing mortality rates: reducing each of these was seen as a good, but both cannot be reduced unless fertility rates are reduced rather more than mortality rates.

It must be said, also, that demographers have tended to specialize in the area of fertility (that is, sex) or in the area of mortality (that is, death) but rarely are they equally comfortable in both. As a result, what most demographers do these days is perhaps best characterized not as 'sex and death', but 'sex or death'.

But what if we return to the title of this paper and interpret it afresh? What if we abandon Hollingsworth's shorthand for childbearing and talk about sex as sexual activity? And what if we consider the dyad of sex, interpreted literally, and death?

This takes us into the realm of sexually transmitted diseases that kill, and so into the realm of HIV/AIDS, the 'new reality' of the sex and death of my title. It is particularly poignant that nowhere has the AIDS epidemic struck with greater force than in sub-Saharan Africa. This is the global region where, in contrast to the rest of the developing world, the process of the demographic transition was long held to be stalled. True, mortality had declined in some countries: for example, in Botswana, the proportion of children dying before their fifth birthday dropped between the mid-1970s and mid-1980s from 83 to 53 per 1000.[5] Nevertheless, while such a decline is proportionately great, the later figure of 53 per 1000 is nearly ten times the level that occurs in the West. And Botswana was the success story of the region.

Only one other country in sub-Saharan Africa has ever recorded under-five mortality lower than 100 per 1000 (Zimbabwe), and many exhibit levels exceeding 200.

This is one reason for designating the region's demographic transition as stalled. The other reason is that fertility decline in the region has remained sluggish: imperceptible in some countries, merely incipient in others.

The tragedy of AIDS in Africa is that hard-won reductions in mortality have been erased or even reversed. Although their numerical estimates differ, both the UN and the US Census Bureau estimate that AIDS has significantly reduced life expectancy during the last ten years. The latter agency projects for Botswana a life expectancy in 2010 of a mere 38 years compared with 61 years in the late 1980s, and thus represents a massive decline of 23 years, that is, a decline of about one year of life expectancy for each year over the period.[6]

Before the African AIDS epidemic, sex and death had never before been linked together so alarmingly. But they have been linked before — consider syphilis. This was a comparative newcomer in the family of 'old' sexually transmitted diseases. Genital warts, for example, were known, and described in the first century AD;[7] and gonorrhoea was known to the author of the book of Leviticus, as well as to Hippocrates.[8] Syphilis, in contrast, was not clearly described, because there were confusions with leprosy,[9] until the end of the 15th century when it erupted in Naples among the soldiers of the army of Charles VIII of France.[10] As if to make up for its comparatively late arrival on the venereal-disease scene, the disease was so virulent and its symptoms so horrible that it became known as the Great Pox, banishing smallpox, itself a dreaded, disfiguring and often fatal disease, to an inferior position. Syphilis was one of the severe trials (others were being shipwrecked, hanged, cut open, lashed and sold as a galley-slave) that Voltaire

imposed on the philosopher Pangloss in order to test his belief that everything is for the best in this best of all possible worlds.

However, the real newcomer is the human immunodeficiency virus, HIV. More than 500 years have passed since the first description of the syphilitic eruptions on some Venetian foot-soldiers, but it is barely 20 years since an opportunistic pneumonia was diagnosed in five gay men in Los Angeles.

These days, most new HIV infections are spread by heterosexual intercourse. Most new syphilitic infections are spread in the same way, just as they were in the mid-18th century when Voltaire published *Candide*. In that work, the trail that ends in Pangloss — part of what we define today as a sexual network — leads back through Pacquette, a pretty serving maid, to a learned Franciscan, to an old countess, then to a cavalry officer, then to a marchioness who had it from her page, who had it from a Jesuit, who had it from one of the companions of Christopher Columbus. So, it had a largely but not entirely heterosexual transmission, plus a hint about the disease's origin as it was then understood.

HIV contrasts with syphilis in numerous ways. For example, the per-coitus probability of transmission of HIV is frequently quoted as 0.0030 from male to female and as 0.0015 from female to male[11] whereas the per-coitus transmission probability of syphilis is generally said to be 0.30,[12] fully 200 times the female-to-male HIV transmission probability. Although the estimate of per-coitus syphilis transmission probability of 0.30 is probably too high (because it was estimated on the basis of one coitus during a 30-day period), even if it is reduced by an exaggerated factor of, say, ten, it is clear that it is still orders of magnitude greater than the corresponding HIV transmission probability.

Another, and insidious, difference lies in the time from infection to the appearance of the first symptoms. On average, the first syphilitic pustule appears at the site of infection within three weeks.[13] In contrast, initial HIV infection is signalled by a febrile illness lasting several weeks that is easily confused with flu or malaria, and it is likely to be years before an HIV-infected individual begins to show the ominous outward signs of HIV infection.[14]

Once infected with HIV, individuals remain infectious until the end of their lives, although the degree of infectiousness varies with the time since infection. In contrast, even in the absence of treatment, syphilitics are infectious for no more than about 12 months. Also in contrast to HIV-infected individuals, most syphilitics develop immunity to subsequent infection.[15] Syphilis is not inevitably fatal. Indeed, only about 30% of people infected with syphilis, and not treated, will progress from the latent stage to tertiary syphilis, and mortality is hastened only in some 40% of these cases.[16] Thus, most people infected with syphilis survive their infection. (To return to *Candide* for a moment, I might mention that Pacquette, who infects Pangloss, develops no symptoms at all, and is as pretty and healthy at the end of the book as she was at its beginning.) There is, of course,

neither vaccine nor cure for HIV. For the HIV-positive with the good fortune to live in a rich country there are expensive drug therapies to stave off progression to AIDS, but for the vast bulk of the world's HIV-positive population there is not even effective palliative care. Syphilis, however, remains profoundly responsive to very simple antibiotic treatment.

As if to emphasize their historical cousinhood, sinister synergies appear to exist between the apparent newcomer among the old venereal diseases, syphilis, and the real newcomer, HIV. Early on in the current epidemic it was observed that many AIDS victims, whether American homosexuals or African heterosexuals, also suffered from other sexually transmitted infections. Syphilis was one of these, although by no means the only one. More generally, a large collection of ulcerative sexually transmitted diseases, including syphilis, and non-ulcerative ones, appeared to facilitate the spread of HIV, while their own spread appeared to be facilitated in turn by HIV. It is natural to wonder whether a major enabling factor in sub-Saharan Africa's AIDS epidemic was a high ambient level of sexually transmitted infections in the general, hence heterosexual, population.

There is a degree of ambiguity in the evidence because of the possibility of confounding by sexual activity. This is clear at the extremes. A life-long virgin will not acquire a sexually transmitted disease (STD) or HIV, or at least not through sexual intercourse. At the other extreme, a popular prostitute may acquire numerous STDs as well as HIV. For the bulk of the population, which lies between these two extremes, how can we tell that the acquisition of one disease facilitated the acquisition of the other? What we may have here is merely selection for sexual gregariousness, because engaging frequently in sexual relations, and with numerous different partners, will increase the chance of independently acquiring both an STD and HIV even if the acquisition of one infection does not, through some biological mechanism, increase the probability of acquiring another.

The problem of selectivity that bedevils the investigation of the role of STDs in the transmission of HIV was cogently addressed in 1992 by Judith Wasserheit[17] in a *Sexually Transmitted Diseases* paper that turned out subsequently to be the most cited article in the journal's 25-year history. Being concerned that selectivity did not contaminate the studies whose findings she was summarizing, Wasserheit restricted her attention to studies that made some attempt to 'control', at least in a statistical sense, sexual behaviour. Statistical assessment of such reported behaviour as the number of sex acts per year, whether the individual had ever had sex with a prostitute, the number of lifetime sex partners, age at first intercourse, and so on, are very pale reflections indeed of the richness and variety of human sexual behaviour. Even such a finer measure as the number of sexual partners during some recent period is inadequate because it obscures the possibility that infection occurred considerably earlier.

A more recent review of the evidence for a link between STDs and the

acquisition of HIV[18] draws on a new abundance of studies: 2101 to be precise, a concrete demonstration of the explosion of work in this new area. Three grounds are given for such a case: first, that it is biologically plausible; secondly, that cohort studies estimate an increased risk of HIV seroconversion in the presence of various STDs; and thirdly, that management of symptomatic STDs reduces the incidence of HIV. This last finding, from Mwanza, Tanzania[19] is particularly compelling because it cannot result, even in part, from population heterogeneity in sexual behaviour. Unfortunately, the same is not true of the second of the grounds, based on the findings of cohort studies. We are thus left with the conclusion that STDs do promote the acquisition of HIV, but that we do not know by how much. We cannot rule out the possibility that some of the observed elevation of risk merely reflects selection on the basis of sexual behaviour.

The Fleming and Wasserheit review, however, is surprisingly silent on this subject. Is it that the proliferating data of the 1990s and the duplication of the finding of an apparent epidemiological synergy between HIV and other STDs have somehow drowned out the concerns raised in Wasserheit's original review? Is it that biomedical scientists are simply more comfortable with biological explanations than behavioural ones? After all, they do tend to write more vividly about their pet pathogens than they do about people. Gonorrhoea, for example, has been called a 'fastidious and fragile bacterium'[20]; the treponeme of syphilis is described as 'eager … to adhere to cells'[21]; and as having 'great powers of survival', 'a wily and elusive foe'.[22] And they write vividly about non-human disease vectors as well. The mosquito, for example, has been called 'iridescent', 'beautiful', 'exquisite', 'hardy', 'clever', 'relentless', 'elegant', 'fascinating', 'bizarre'[23] although not in the same sentence!

Now, if it is true that biomedical scientists are more comfortable with biological than behavioural explanations, would it matter? After all, in the best of all possible worlds, of the sort advocated by Fleming and Wasserheit,[17] asymptomatic infections would be detected through frequent screening, and all STDs, symptomatic or not, would be treated promptly and effectively. The outcome would be reduced transmission not just of STDs but also of HIV.

However, we do not live in the best of all possible worlds, or at least I hope, as did Voltaire, that the one we live in is not the best we can do. Heath Clark would certainly have believed we can do better, but the scenario of universal and effective treatment of STDs is simply not, at present, an achievable goal for poor countries, such as those of sub-Saharan Africa. It has not been achieved even in rich countries.

Just as the practitioners of the biological sciences are probably most comfortable with the view from below, their gaze fixed firmly on the pathogens and vectors they know so well, the demographers probably find it most natural to take the view from above. One of our central preoccupations, underpinned by

the mathematical system known as 'stable population theory', is the quest to understand how populations work; to understand the ways in which fertility and mortality interact to change population size and age structure. This sort of demography deals with population processes at such a high level of abstraction that they are not generally apprehended by the human beings whose individual experiences go to make up the big picture. I qualified this statement with 'generally' because, as the number of funerals soars, everyone in southern and eastern Africa comprehends the appalling toll being exacted daily by AIDS.

The big picture can be a gripping one, and the language used to describe it can be correspondingly vivid. Demographers write about 'population momentum', for example, as though a population had a life of its own quite distinct from that of its constituent individuals. In a very particular sense, it does. Stable population theory tells us that, whatever a population's initial age structure, if fertility rates are held constant for long enough and mortality rates are held constant for long enough, the population will eventually assume an unchanging age structure. The form of this is determined entirely by those fixed fertility and mortality schedules, and owes nothing to the original age structure. We can say that, to quote the title of one paper, the population has forgotten its past.[24]

Such 'forgetting', however, takes time. Long after the death of the last victim of HIV/AIDS, and that death will not come soon, the populations of sub-Saharan Africa will bear the scars caused by radically depleted cohorts of young adults. These scars are more than demographic, they are economic, and social and they are psychic.

Whether our disciplinary base leads to our viewing the HIV/AIDS epidemic from below, at the level of pathogens, or from above, at the level of populations, we are united in the recognition that it is individual human beings, those who have been colonized by those pathogens, and those who, together, make a population that is the ultimate focus of our concern. Let me close with part of an overheard conversation between some ordinary citizens of Malawi, one of the countries where AIDS now rages in the general population and at least 16% of the general populations aged 15 to 49 years is estimated to be HIV positive.[25]

The conversation is between three strangers, chance-met in a bus station, a woman and two men. The woman has just come from her cousin's funeral; one of the men has been at the hospital, visiting his uncle who is being treated for TB.

First Man: There is no way we can run from the AIDS disease because nobody can control nature. Who can manage to stay with a marriage or have sex with the same person all the time? And it's impossible to use condoms all the time, especially with your wife.

Second Man: How can you use a condom with your wife? If you do so

it means that you don't want to have children in your house, and the wife herself cannot agree to have sex using a condom unless there is a great problem, like if she had an operation two or three times during delivery. Then it can be reasonable to use condoms to avoid becoming pregnant.

First Man: If you have that problem just go to the hospital and tell the doctors, and tell them to make her stop bearing [that is, undergo tubal sterilization]. But other people say that they have traditional herbs which they drink and they cannot get AIDS.

Woman: Is that true?

First Man: Maybe, but I cannot believe it. What I know is that we shall all die with AIDS, and there is nobody who will not taste AIDS.

Second Man: It seems that God wants to destroy the whole world through that disease. We shall all die with AIDS because it is now found in many diseases, like TB, malaria, headache, diarrhoea and shingles. If someone suffers from one of these diseases, you will just hear that the person has AIDS. But people were suffering from them a long time ago, before AIDS came. Today everything is AIDS, which means that everyone will die from it because nobody can deny suffering from malaria, and nobody can avoid having diarrhoea. And especially shingles, how can you avoid shingles? Let God himself do whatever he has planned to us. We are his people and he is free to do whatever he wants with us.

First Man: I think the disease is from the Government. It was purchased from somewhere because they would like few people to remain in this Malawi country.

Woman: Aah, can it be possible Sir? Purchasing a disease to kill people, why?

First Man: Why are they forcing people to get family planning? Unless they help to look after the children, they don't have a right to speak on this issue. They are just killing women deliberately in order to reduce the population, and they are killing many people through family planning injections and tablets …

Heath Clark directed that the lectures endowed in his name focus not on technical but on educational, cultural and humanistic aspects of preventive medicine and

tropical hygiene. This focus seems to me to be particularly apt in the case of sub-Saharan Africa, first with its stalled demographic transition, and now with its AIDS epidemic. We already have a technical solution to high fertility rates: efficient, modern contraception. Yet fertility in sub-Saharan Africa remains high. We already have a technical solution to the AIDS epidemic: not a vaccine, let alone a cure, but the condom. True, it would be splendid if STDs were treated, and if people were faithful to their sexual partners, and if there were widespread use of multiple drug therapies to reduce HIV transmission, relieve suffering and prolong life. But condom use is the practical key to bringing the epidemic under control.

This is where the emphasis on 'educational, cultural and humanistic aspects of preventive medicine' becomes so pertinent; we could use different words, but the intention is clear, unfortunately we do not know how to promote condom use. Specifically, we do not know how to promote condom use among people who believe that it is inappropriate to control fertility within marriage, and believe also, because of its connotation with 'outside' sex, that it is inappropriate to use a condom with one's wife. We do not know, either, how to promote condom use among people who distrust their Government. And we do not know how to promote condom use among people whose recognition of the Protean manifestations of AIDS leads them to the chilling conviction that every death is an AIDS death, and hence that HIV infection is inevitable:

> What I know is that we shall all die with AIDS, and there is nobody who will not taste AIDS.

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References

1. T. H. Hollingsworth (1969) *Historical Demography* (Ithaca, New York: Cornell University Press).
2. R. Pearl (1939) *The Natural History of Population* (London: Oxford University Press).
3. D. Kirk (1996) Demographic transition theory. *Population Studies*, **50**, 361–387.
4. S. H. Preston (ed.) (1978) *The Effects of Infant and Child Mortality on Fertility* (New York: Academic Press).
5. I. M. Timaeus (1998) Impact of the HIV epidemic on mortality in

sub-Saharan Africa: evidence from national surveys and censuses. *AIDS*, **12**, Sup 1, 15–27.
6. J. Stover and P. Way (1998) Projecting the impact of AIDS on mortality. *AIDS*, **12**, Sup 1, 29–39.
7. L. A. Koutsky and N. B. Kiviat (1999) Genital human papillomavirus. In K. K. Holmes, P.-A. Mårdh, P. F. Sparling *et al*. (eds) *Sexually Transmitted Diseases*, third edition (New York: McGraw-Hill) pp. 347–359.
8. P. F. Sparling (1999) Biology of *Neisseria gonorrhoeae*. In K. K. Holmes, P.-A. Mårdh, P. F. Sparling *et al*. (eds) *Sexually Transmitted Diseases*, third edition. New York: McGraw-Hill) pp. 433–449.
9. R. H. Kampmeier (1984) Early development of knowledge of sexually transmitted diseases. In K. K. Holmes, P.-A. Mårdh, P. F. Sparling *et al*. (eds), *Sexually Transmitted Diseases*, first edition (New York: McGraw-Hill).
10. C. Quétel (1990) *History of Syphilis* (Cambridge, MA: Polity Press).
11. R. M. Anderson. (1999) Transmission dynamics of sexually transmitted infections. In K. K. Holmes, P.-A. Mårdh, P. F. Sparling *et al*. (eds) *Sexually Transmitted Diseases*, Third edition (New York: McGraw-Hill), pp. 25–37.
12. A. L. Schroeter, R. H. Turner, J. B. Lucas and W. J. Brown (1971) Therapy for incubating syphilis. Effectiveness of gonorrhea treatment. *Journal of the American Medical Association*, **218**(5), 711–713.
13. D. M. Musher (1999) Early syphilis. In K. K. Holmes, P.-A. Mårdh, P. F. Sparling *et al.* (eds) *Sexually Transmitted Diseases*, third edition (New York: McGraw-Hill) pp. 479–485.
14. D. Low-Beer, R. Stoneburner, T. Mertens, A. Burton and S. Berkley (1998) HIV and AIDS. In C. J. L. Murray and A. D. Lopez (eds) *Health Dimensions of Sex and Reproduction* (Cambridge: Harvard University Press) pp. 297–388.
15. P. F. Sparling (1999) Natural history of syphilis. In K. K. Holmes, P.-A. Mårdh, P. F. Sparling *et al*. (eds) *Sexually Transmitted Diseases*, third edition (New York: McGraw-Hill) pp. 473–478.
16. J. Rowley and S. Berkley (1998) Sexually transmitted diseases. In C. J. L. Murray and A. D. Lopez (eds) *Health Dimensions of Sex and Reproduction* (Cambridge: Harvard University Press) pp. 19–110.
17. J. N. Wasserheit (1992) Epidemiological synergy: interrelationships between human immunodeficiency virus infection and other sexually transmitted diseases. *Sexually Transmitted Diseases*, **19**(2), 61–77.
18. D. T. Fleming and J. N. Wasserheit (1999) From epidemiological synergy to public health policy and practice: the contribution of other sexually transmitted diseases to sexual transmission of HIV infection. *Sexually Transmitted Infections*, **75**, 3–17.
19. H. Grosskurth, F. Mosha, J. Todd, E. Mwijarubi *et al.* (1995) Impact of improved treatment of sexually transmitted diseases on HIV infection in rural Tanzania: Randomised controlled trial. *The Lancet*, **346**, 530–536.
20. C. A. Hart (1998) Laboratory aspects. In O. P. Arya and C. A. Hart

(eds) *Sexually Transmitted Infections and AIDS in the Tropics* (Wallingford: CABI Publishing) pp. 51–63.
21. K. Wicher and V. Wicher (1983) Immunopathology of syphilis. In R. F. Schell and D. M. Musher (eds) *Pathogenesis and Immunology of Treponemal Infection* (New York: Marcel Dekker) pp. 139–159.
22. T. B Turner (1983) Foreword. In R. F. Schell and D. M. Musher (eds) *Pathogenesis and Immunology of Treponemal Infection* (New York: Marcel Dekker) p. v.
23. R. Horton (2001) Review of Spielman and D'Antonio. *New York Review of Books*, 9 August.
24. A. Coale and J. Trussell (1981) 'Roots', or how a population forgets its past. *Canadian Studies in Population*, **8**, 1–25.
25. www.who.int/emc-hiv/fact_sheets/pdfs/Malawi_EN.pdf

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