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Safety issues in didactic anatomical dissection in regions of high HIV prevalence

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SUMMARY

Ruggiero *et al.* (2009) have recently reviewed the importance of dissection in the training of physicians, the role played by students' fears of infection, and the evidence that those sometimes extreme fears are unwarranted even respecting HIV and AIDS, whose dangers continue to be featured prominently in popular media as though everyone were at constant risk.

It is not especially surprising that the risk of accidental infection by HIV is negligibly low in random dissections in Italy where, as in Europe generally, the prevalence of HIV is only a fraction of a percent. The question arises, however, what the risk might be in regions where the prevalence of HIV is considerably higher.

South Africa is an obvious candidate for investigating this issue since the prevalence of HIV there is among the highest reported by UNAIDS and other official bodies. Furthermore, its record-keeping system is more reliable than that of most other countries in sub-Saharan Africa, the global region that is universally regarded as the epicenter of the HIV/AIDS epidemic. In addition, South Africa has a globally recognized reputation in the teaching of human anatomy.

Perhaps surprisingly, the risks in South Africa also seem to be much less than might be anticipated on the basis of the conventional wisdom. One reason for this counter-intuitive conclusion is that estimates of HIV prevalence and of AIDS deaths issued by international bodies are significantly overblown, with some estimates being 20 times or more greater than locally recorded numbers. A second basis for the unexpected conclusion is that the possibility of false-positive HIV tests has been ignored despite the considerable range of evidence that false-positives can be a significant part, perhaps even a major part, of positive tests in certain groups or certain regions, saliently among people of African ancestry.

INTRODUCTION

Ruggiero *et al.* (2009) have recently reviewed the history of anatomical dissection in the training of medical students, its didactic importance, and the issue of fear of infection on the part of at least some students. We would add that the absolute necessity of direct, personal experience with dissection of human cadavers during medical training is brought home by the thought experiment that one would not wish any doctor's first encounter with making an incision in a human body to be in the treatment of a living patient; particularly, of course, in the case of a specialist surgeon.

The risk of accidental infection by any contagious agent depends on two factors: the probability that a cadaver is infected, and the probability that exposure to bodily fluids or tissues containing the contagious agent leads to actual infection.

There seems to be no reason why the probability that exposure leads to infection should be characteristically different in different geographic regions. Local practices might affect the probability of an accidental exposure, but there is no reason to assume that in South Africa the care taken in medical training institutions is on average less than in other places. This is particularly true considering the high reputation of South African Anatomy Departments. Thus, the Anatomical Society of Southern Africa (ASSA) was founded in 1967 and it represents anatomy departments from all tertiary institutions in Southern Africa (*i.e.* South Africa, Mozambique and Zimbabwe). It also maintains strong relations with Luanda, Angola and Lusaka, Zambia. The Society promotes excellence in research and education and has become the professional home for biomedical researchers and educators in South Africa focusing on anatomy in its broadest sense (from the web site of the Society at http://www.assa.uct.ac.za/).

So the data on the probability of seroconversion following accidental exposure adduced by Ruggiero *et al.* (2009) in that respect remains pertinent to the South African situation, a probability of at most a fraction of a percent that actual exposure leads to seroconversion.

On the other hand, the probability that a given body is HIV-positive is likely to vary from one geographical region to another since the global distribution of HIV is anything but uniform. However, the likelihood that a given cadaver is HIV-positive cannot be judged on the basis of the overall prevalence of HIV in the general population, since that likelihood is influenced characteristically by age, sex, race, history of drug abuse, and (of course) cause of death. Even in the case of anonymous cadavers, at least the first three factors can offer guidance as to the specific probability in a given case.

The greatest possible difference between the situation in Italy and circumstances in South Africa concerns the much higher estimated prevalence of HIV in South Africa. Nevertheless, this is unlikely to translate into a higher risk of infection as a result of accidents during anatomical dissection because the estimated HIV prevalence is considerably higher than the prevalence of active infection, in part be-

cause of the mathematical modeling procedures from which estimates are derived and in part because the probability of false-positive HIV tests has been seriously neglected. Evidence will be presented that the proportion of false positives is quite high, especially among Africans.

MATERIALS AND METHODS

The starting point of our review was the HIV infection rates in South Africa. We analyzed in detail the estimates and the models used to derive such data, beginning with the official ones published by the Joint United Nations Programme on HIV/AIDS (UNAIDS) and the *Morbidity and Mortality Weekly Report* (MMWR) from the US Centers for Disease Control and Prevention, because those are the information sources that the general population and the media usually refer to. We offer the web coordinates (URLs) of the data discussed, whenever they did not come from a published scientific paper. Since our interest was to determine whether HIV serophobia in body dissections is justified (in a high-incidence area such as South Africa), we tried to establish the reliability of those estimates by searching the official literature regarding the numbers actually observed in scientific epidemiological studies.

From there we moved to examine the grounds on which the estimating models are based, and to do so we referred to the available literature about HIV transmission rates in case of accidental and behavioural exposure.

Since the reliability of estimates is largely dependent on the reliability of HIV testing, we took a close look at the testing protocol in South Africa, with particular regard to the standards and the calibration procedures, and we tried to assess how much a positive HIV test actually says about active infection, as opposed to simple seroconversion without a significant trace of virus.

With such information at hand, it was finally possible to analyze the discrepancies in and between the models used for estimating HIV spread in Africa, establishing what the odds are of finding a corpse with an active HIV infection and, in that case, of contracting HIV after an accidental exposure with infected tissues.

RESULTS AND DISCUSSION

Several indications suggest that estimates of HIV infection in South Africa have been greatly exaggerated. One reason for this is reliance on mathematical models based on scanty data and a host of assumptions. The inadequacy of the models is illustrated by the stark difference between AIDS deaths recorded by Statistics South Africa and the estimates of AIDS deaths disseminated by international institutions.

A further reason for the inflated estimates is that the possibility of false-positive

HIV tests has been neglected. Most important, the cut-off value for a positive HIV-antibody test depends on a norming or calibrating procedure, and the evidence is strong that re-norming is called for in Africa. In addition, a positive antibody test does not necessarily signify active infection, so the risk of infection by accident during dissection is less than would be expected on the basis of HIV-antibody prevalence in cadavers.

Prevalence of HIV in South Africa

A widely disseminated number for HIV prevalence in South Africa in 2007 is about 18% (UNAIDS, 2008). However, there are several grounds for suggesting that the true figure is considerably lower. First, the most recent national survey in South Africa gives the prevalence as 10.6% in 2008 (Shishana *et al.*, 2009). Second, a former epidemiologist at the World Health Organization (WHO) has testified that all UNAIDS estimates for HIV/AIDS have been greatly exaggerated (Chin, 2007). Third, the estimates are arrived at by means of models based on assumptions which have not been validated independently.

Chin was aware of overestimations through his service at WHO. But further, even on the basis of his own lower, supposedly more realistic estimates, Chin calculated from generally accepted data on transmission probabilities that, to explain the magnitude of the African HIV/AIDS epidemic, one had to postulate that 20-40% of adults have "multiple concurrent relationships" – several sexual partners at the same time, changing to new partners weekly or monthly, to totals of tens of different partners over the course of each year (Chin, 2007, Table 5.1, p. 64). That level of promiscuity seems literally unsustainable. But even if it were in fact the case, then that would be obvious to the most casual observer or visitor; instead, a number of studies have failed to find evidence of high promiscuity among Africans (Bauer, 2007, pp. 77-8).

Chakraborty *et al.* (2001) also reached the conclusion that the apparent rate of sexual transmission of HIV is too low to account for the observed prevalence. Gisselquist *et al.* (2002) have made the same argument specifically for African circumstances.

Flawed models of HIV/AIDS: AIDS deaths

All models of HIV/AIDS assume that sexual transmission is the primary means by which infection is spread, but since observed rates of sexual transmission cannot explain the estimated prevalence, the present theoretical computer models cannot be relied on to make projections or estimates for purposes of policy or action. An additional reason for doubting that the role of sexual transmission is properly understood lies in several studies which have found that condom users do not have a lower rate of testing HIV-positive, for example in Africa (Rwanda, 2006; Wawer *et al.*, 2005) and Pakistan (Shah *et al.*, 2002).

The deficiency of current models of HIV/AIDS is shown not only by the inability of sexual transmission to explain plausibly the estimates of HIV prevalence generated by those models, it is further underscored by the greatly inflated estimates (from those models) of AIDS deaths in South Africa, estimates that exceed actually recorded deaths by a factor of 20 or more.

According to P. Lehohla, Statistician General for South Africa, assumptions made in the model used by the Medical Research Council (MRC) to estimate AIDS deaths are severely flawed, for example in "underplaying the role of unnatural deaths in young adults": the MRC model considered only deaths from *political* violence, which *decreased* after 1993, whereas *all* deaths from unnatural causes *increased* dramatically from 42,000 in 1994 to 61,000 in 1996; for the group aged 15 to 49, the 45,000 unnatural deaths represented 65% of all deaths. By contrast, the MRC *speculated* that in 2000, 40% of all adult deaths in South Africa might have been owing to AIDS; the Statistician General stated unequivocally that this estimate cannot be uncritically accepted (Lehohla, 2005).

Unfortunately, UNAIDS relies on the MRC estimates instead of on the actual records from Statistics South Africa. The 2008 UNAIDS Report repeats the assertion that 40% of all adult deaths were owing to AIDS, whereas Statistics South Africa (2006) gives the percentage as 2.4%, with the actual number 14,800. That number placed AIDS (or "HIV disease") only 9th among causes of death, the first three being tuberculosis (TB) (77,000), flu & pneumonia (53,000), and intestinal infections (39,000). It is worth noting that for anatomists performing dissections, TB has historically been the major threat. That these numbers are relatively reliable is underscored by the fact that the completeness of registration of deaths exceeds 50%; according to Lehohla (2005), it has fluctuated somewhat, being about 60% in 1990, 73% in the mid-1990s, and presumably no worse since then. UNAIDS, although for deaths it uses MRC estimates instead of numbers from Statistics South Africa, concedes that the death notification system "function[s] relatively well" in South Africa (UNAIDS, 2008, p. 45). Yet the UNAIDS percentage of estimated AIDS deaths is 16-17 times greater than the reported figure from Statistics South Africa.

The preference for estimates from invalid models over quite trustworthy numbers from the official South African agency has unfortunate consequences, because HIV/AIDS researchers and the popular media tend to regard UNAIDS reports as authoritative (perhaps overlooking the disclaimer, "UNAIDS does not warrant that the information contained in this publication is complete and correct and shall not be liable for any damages incurred as a result of its use" [UNAIDS, 2008]). For example, Chigwedere *et al.* (2008) accept the MRC/UNAIDS/CIA estimate of 350,000 AIDS deaths in 2007 to speculate how many deaths might have been prevented if antiretroviral drugs had been made more available earlier.

Estimates of HIV prevalence arrived at by the same models that estimate deaths are likely to be overstated by a similar amount. AIDS deaths were recorded officially as 14,800 while MRC's model estimate was 350,000, greater by a factor

of more than 20. Therefore, estimated HIV prevalence must also be overstated, perhaps by something like an order of magnitude. For that reason alone, the risk of encountering an HIV-positive cadaver is likely to be considerably less than popularly believed.

False-positive HIV tests

The unreliability of HIV/AIDS models is only one reason for questioning estimates of the prevalence of active HIV infection; another is the occurrence of false-positive HIV tests.

One possible reason for false positives is that few if any testing facilities, particularly perhaps in Africa, are able to engage in the elaborate interplay between clinical observation, medical history of patients, and laboratory work-up that are called for if HIV tests are to be used to diagnose actual infection (Weiss & Cowan, 2004). The overwhelming majority of HIV testing has been and continues to be done only via antibody tests (ELISA), which were designed and approved for screening and not for diagnosis. Those tests measure a color intensity, and "cut-off" values have to be decided to distinguish among negative, indeterminate, and positive. Cut-off values are chosen by calibrating with a control population of "true negatives", typically blood donors, and a test's performance is assessed by comparing the "true negative" color intensity with results in high-risk populations and those known to be infected (Weiss & Cowan, 2004, p. 150). Cross-reactions may still remain a problem, depending on the source of the antigens used in the tests, and could account for mistakenly high rates of apparent HIV prevalence among drug injectors, for example (Weiss & Cowan, 2004, p. 151).

Second: Antibody tests can only deliver information about the presence of antibodies, not about active infection. If one defines a genuine positive as indicative of active infection – which is the appropriate definition if one is interested in possible dangers of accidental infection during dissection – then one must take into account that seroconversion alone, the presence of antibodies, does not necessarily indicate active infection. That presumably is why the tests have been approved only for screening purposes and why the diagnosis of actual infection calls for the painstakingly elaborate procedures outlined by Weiss & Cowan (2004). The pertinent literature offers no clear explanation as to how it came about that antibody-positive is nowadays generally accepted in practice as indicating active infection.

Further evidence for false-positive HIV tests comes from the fact of apparent seroconversions in absence of exposure to HIV.

Seroconversion in absence of exposure to HIV

That some false positives arise as a result of cross reactions with non-HIV antibodies has long been known from a variety of sources, even though in practice the significance of this fact is rarely taken into account. Johnson (1996) published a sur-

vey of several dozen sources reporting cross reactions to such things as vaccinations. In trials of circumcision as a possible prevention against HIV infection, a number of cases of seroconversion, in both intervention and control groups, occurred in absence of sexual intercourse, blood transfusion, medical injections, or other possible avenues of infection (Bailey *et al.*, 2007; Gray *et al.*, 2007).

Surveys of HIV prevalence among pregnant women persistently record higher frequencies than in the general population – in Malawi (Taha, 1998); in Uganda (Gray *et al.*, 2001); in the United States (see data for pre-natal and obstetric clinics in Table 23, p. 81, and Figure 22, p. 83, in Bauer, 2007), suggesting that pregnancy itself might sometimes generate false-positive HIV results. The most direct confirmation of this was found by Gray *et al.* (2005) in a large prospective study of the incidence of HIV-positive among pregnant women: "In married pregnant women who had a sexual relationship with their male spouses, the HIV incidence rate ratio was 1.36 (0.63-2.93). In married pregnant women in HIV-discordant relationships (*i.e.*, with HIV-positive men) the incidence rate ratio was 1.76 (0.62-4.03)". Thus the rate of seroconversions in discordant relationships was very little higher than the overall rate; evidently the rate of seroconversion in non-discordant relationships was appreciable, in other words, seroconversion in absence of exposure to HIV.

In addition to the possibility of false-positive antibody tests, one must recognize that antibody-positive does not necessarily signify active infection, as demonstrated by the existence of "elite controllers" or "long-term non-progressors".

Elite controllers: What proportion of HIV-positives?

"Elite controllers" or "long-term non-progressors" persistently test "HIV-positive" yet remain healthy, and in some cases they have undetectably small viral loads, in other words, they have HIV antibodies in absence of HIV virions (Slack, 2009). By the very fact that elite controllers are healthy, there is no definitive way to determine what proportion of all HIV-positives they might constitute, short of testing every healthy person. However, several lines of evidence indicate that the proportion may be quite high.

Perhaps the most direct hard data on long-term non-progressors comes from the United States Armed Services, whose members are typically HIV-tested every two years. There are known to be 382 such individuals among 4574 who had been followed for up to 20 years, that is, 6.4% of the HIV-positives (Okulicz *et al.*, 2008).

Another approach to the question suggests an even higher proportion. About 1 million Americans have been HIV-positive in each year since the mid-1980s at least (specific sources for 1986, 1987, 1988, 1989, 1993, 2003 are cited at pp. 1-2 & 108 in Bauer, 2007). The Centers for Disease Control and Prevention continually urge universal testing because about one quarter (MMWR, 2007) or perhaps one third (Sine, 2006) of all HIV-positive people do not know that they are HIV-positive, in the United Kingdom (Carter, 2007) as well as in the United States. It is interesting

to note that this type of trend is common in all the epidemiological reports available, from regional to national data. For example, the most recent data for Italy and Tuscany (updated December 31, 2008, provided by the Center for Study, Research and Documents on Addictions and AIDS, available at www.cesda.net/downloads/AIDSARS2008.pdf) indicate that 52% of AIDS cases are found to be HIV-positive only at the time that the diagnosis of AIDS is made. This percentage rises to 63% among those who declare to have been infected through heterosexual exposure. So ever since the mid-1980s there have been one quarter or one third of the million HIV-positive Americans, in other words 250,000-333,000, who did not know they were positive, and who therefore were also not known to the authorities to be positive and were not receiving antiretroviral treatment. How many of those are "elite controllers"? In Italy for example, the Ministry of Health (Istituto Superiore di Sanità), in the annual report updated December 2008, states that only 34% AIDS patients had received antiretroviral treatment before the diagnosis of AIDS was made; 62.9% did not receive any antiretroviral treatment (and for the remaining 3.1% it is not known) (Suligoi *et al.*, 2009). This indicates that at least 62.9% HIV-positive subjects most likely did not know about their HIV status.

A recent estimate gives an annual incidence of about 55,000 new HIV-positive cases in the USA (Hall, 2008). There is no reason to imagine that 1 million HIV-positives in 1986 and later years generated fewer new cases annually than did the 1 million HIV-positives in recent years, so 1 million will have been augmented annually by 55,000 for more than two decades, for a total (beginning in 1986) of 2,155,000 by the end of 2007. On the other hand, AIDS deaths have been recorded as 583,000 through 2007 (CDC, 2009). So the 1,000,000 in 1985 should have grown by 2007 to 1.572 million (2,155,000 minus 583,000). Instead, the CDC reports 264,000 "Living with HIV infection" and 469,000 "Living with AIDS" at the end of 2007 (CDC, 2009, Table 14), a total of 733,000. The difference between the expected 1.572 million and the actual 733,000, namely 839,000, represents plausibly the number of people who, at one time or another, were HIV-positive but have never been tested nor become ill from anything that would occasion an HIV test: in other words, they are unknown or unrecognized elite controllers. The present number of elite controllers, then, is plausibly on the order of 840,000, somewhat larger than the number (730,000) of those who are currently believed to be living with HIV/AIDS. This line of reasoning suggests that more than half of all those who would test positive currently – if there were universal testing in the United States – may well be elite controllers.

There is strong evidence, then, that a significant proportion of positive HIV tests are false positive in the sense of indicating antibody-positive but not active infection. This consideration might help explain the fact that a negligible number of exposure accidents have actually led to full blown AIDS, including dissection-related incidents (Jost *et al.*, 1995; Chamberland *et al.*, 1995).

Racially biased tests

In the circumstances of South Africa, it is vital to recognize that HIV tests appear to be racially biased. African Americans test HIV-positive at far greater rates than other Americans, irrespective of economic or social class or risk group: among gay men, drug abusers, applicants for marriage licenses, military personnel, childbearing women, or prisoners, African Americans test positive at rates higher than white Americans, by factors as high as 10 or more (Bauer, 2007, Table 8, p. 51). According to the Centers for Disease Control and Prevention (CDC, 2009), the rate of new seroconversions among black Americans in recent years was 7.3 times that among whites (5.9 among males, 14.7 among females); about 10 among adults and about 20 among children <13.

Those enormous differences are typically ascribed to differences in sexual and drug-using behavior, despite the fact that other racial discrepancies make that explanation highly implausible, namely, that Native Americans test HIV-positive at rates much closer to those of whites than of blacks, and that Asian Americans typically test positive about 30% less often than whites (chapters 5 to 7 in Bauer, 2007). Moreover, as already mentioned, a number of studies have found no evidence of higher promiscuity among Africans or African Americans (Bauer, 2007, pp. 77-8).

The manner in which HIV tests are calibrated, and the possibility of cross-reactions correlated with HLA type (Weiss and Cowan, 2004, p. 151), suggest why HIV tests might be racially biased: HLA type is racially correlated (see for example, Creemers and Khan, 1998). Blood donors are typically used as low-risk "true negative" controls, yet among blood donors, African Americans tested positive 14 times more often than white Americans (Petersen and Doll, 1991; Ward, 1988). In South Africa, among regular (repeat) blood donors, blacks tested positive 23 times more often than whites or Indians (Manto, 2004). Therefore, if tests were to be calibrated separately in different racial categories with blood donors as controls, apparent HIV prevalence among Africans and people of recent African ancestry would be much lower than present estimates.

One cannot, of course, assume linear relationships and reduce estimated HIV prevalence among Africans by a factor of 23, but it does seem evident that the actual rate of HIV infection in South Africa is considerably less than the UNAIDS/CIA figure of 18% for HIV (antibody) prevalence or the Statistics South Africa figure of 10.6% (Shishana *et al.*, 2009); according to the 2001 census cited by the CIA Fact Book, blacks make up 79% of the South African population, whites less than 10%, colored about 9%, Indians or Asians about 2.5%, so the overall South African prevalence is determined overwhelmingly by the rate among black Africans.

Probability of seroconversion after accidental exposure to HIV during anatomical dissection

The foregoing data establish that the rate of HIV-antibody-positives among

South Africans is significantly less than the official estimates of 18% or 10.6%, owing in the first case to flawed models and in both cases to racially biased HIV tests. Therefore the relative risk in South Africa compared to Italy cannot be judged on the basis of national differences in apparent HIV prevalence.

Yet another reason why HIV-antibody-positive cadavers may not be actively infected is that trauma, perhaps death itself, can apparently produce substances that cross-react with HIV tests – seroprevalence was found to correlate with degree of critical illness (MMWR, 1987), it was higher in emergency rooms (Kelen *et al.*, 1989), and it seemed to correlate with penetrative trauma (Kelen *et al.*, 1988). In autopsies, HIV prevalence was higher among homicide victims than among those who died accidentally or of natural causes (Coleman *et al.*, 1986; Resnick *et al.*, 1991).

Ruggiero *et al.* (2009) cited a number of sources that suggest a very low probability of seroconversion after accidental contact with HIV-contaminated tissues, fluids, or objects. There are further indications that the probability may in fact be negligibly small. For example, studies at a clinic for drug abusers found, opposite to expectation, a much higher prevalence of HIV (34%) among those who did not share needles than among those who did share (19%) (Krueger *et al.*, 1990). Similarly, clean needles were associated with a ten-fold *increase* in the odds of seroconverting to HIV-positive even though transmission of hepatitis B was less (Bruneau *et al.*, 1997). Furthermore, medical service in the military encompasses high probability of accidental exposure to infected fluids and tissues, yet medical personnel in military service are at no occupational risk of seroconversion to HIV-positive (Cowan et al., 1991; Kelley *et al.*, 1990).

CONCLUSIONS

In the early 1980s, sheer panic was aroused by the appearance of an apparently quite new disease, originally designated GRID but later AIDS, that was invariably fatal and without cure. When the cause was said to be a sexually transmitted virus, now designated HIV, the panic became a world-wide phenomenon. That HIV/AIDS constitutes a clear and present danger to everyone everywhere has become an entrenched part of the conventional wisdom, even as experience has shown that the dreaded disease has not spread, as was initially feared, into general populations in most of the world. There is now a remarkable contrast between the continuing popular belief about the dangers of contracting HIV and the actual fact that the incidence of HIV has nowhere outside sub-Saharan Africa constituted a threat except to the groups in which it was first identified, promiscuous drug-abusing gay men and other drug abusers.

Ruggiero *et al.* (2009) pointed out that medical students, like the rest of the population, believe that any contact with an HIV-positive person or tissue is very risky, and that this belief brings anxiety particularly with the didactically necessary training in anatomical dissection. In the context of Italian circumstances, where the

population-average prevalence of HIV is quite low (0.4%), it was shown that students' anxiety is ill founded because of the very low probability of seroconversion after exposure to HIV.

That conclusion can be plausibly extended to all other regions where HIV prevalence is comparable, but the question arises whether it can be extended to high-prevalence areas like sub-Saharan Africa.

A cumulation of evidence from a variety of directions indicates that students' anxiety can be assuaged even in so high-prevalence a region as South Africa. In the first place, the estimates of HIV prevalence are based on surveys using tests calibrated under non-African conditions, and it seems clear from comparative data on repeat blood donors, who are "true negative" controls for assessing HIV tests, that the most widely used tests are racially biased in a manner that greatly overstates HIV prevalence in people of African ancestry. Second, the ubiquity of false-positive tests should not be ignored, given the copious reports of antibody-positives in absence of active infection.

Thus the reassurance by Ruggiero *et al.* (2009) to medical students concerning didactic dissection can be generalized even to regions where UNAIDS and other international institutions posit the highest observed prevalence of HIV antibodies.

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