THE CAUSES AND MANAGEMENT OF HIV/AIDS AND RELATED PROBLEMS: REASONS FOR QUESTIONS AND CONCERN. Ref AIDS/drugs 82-04.

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ABSTRACT: *Although AIDS has been since 1984 featured as the greatest medical disaster since mediaeval plagues, many physicians know nothing about it and have seldom if ever seen a case. This is mainly because, in developed countries, AIDS is an uncommon disease, restricted now, as in 1981 when it first appeared in the USA, to groups of homosexual men with multiple partners, users of addictive drugs and their consorts. Official predictions that AIDS would spread uncontrollably to general populations by heterosexual transmission of HIV were shown in 1992 and subsequently to be wrong in the UK, probably in all developed countries and in some communities in developing countries. But, in sub-Saharan Africa and some other developing regions, registrations of AIDS exploded in totally different clinical and epidemiologic patterns, with considerable but unmeasured overlaps with concurrent endemic diseases classifiable as HIV/AIDS under the permissive Bangui definition. This new concatenation of morbidity raised fears that affliction would become global with a dimension second only to that of terrorism. The public health profession, as the medical arm of national and international security, was slow in responding to the challenge of AIDS, and left a gap. Hence, in the critical period of exponential increase in registrations in1985-89, there was a lack of local, professional wisdom to correct errors and exaggerations in projections of incidence and distribution. The gap widened into a failure internationally to define opportunities for primary prevention by ABC (Abstinnce, Be faithful, Condoms) and control by strategic antimicrobial and other therapy. Instead, wisdom was credited in 1984 to an upstart hypothesis that positive indirect tests for a new retrovirus HIV meant AIDS and then death, with no options. Immediate and uncritical acceptance of this hypothesis became consensual in official quarters, as in the London Declaration of 1988, reinforced later by academics internationally in their dogmatic millennial Durban Declaration on HIV/AIDS. This imposed a blanket of censorship on any scientific dissent, which intensified as registrations of HIV/AIDS increased, mainly in countries in Africa and the Americas with no capability for validation of diagnosis and routes of transmission. The need for ABC to counter this deficiency was recognised by the African National Congress and other grass root groups.*

The consensual HIV hypothesis of causation and transmission of AIDS prioritised instead the medical construct, and continued to insist on drugs and vaccines for control of HIV, despite twenty-three years of limited achievement and stultifying pessimism The present Statement questions again the validity of this hypothesis, rejects its apocalyptic implications and suggests an aetiology linked primarily to risk behaviours in life styles leading to overloads of unmanageable infections with exhaustion of immunity and vitality. But it fully endorses fears of uncontrolled threats to survival of economically-active adults and of children in backward communities, not only in developing countries, because there are large overlaps elsewhere with deprivations of elementary human rights, runaway epidemics of drug misuse, hepatitis and all of the common sexually-transmissible, traditional and opportunistic infections. These are aggravated by adversities and abuses experienced disproportionately by women and no longer denied by the Consensus. There is therefore common ground for rationalisation of dissent and convergence of efforts to develop simpler, more effective and affordable domestic as well as biomedical measures to control the assortment of diseases registrable medically as HIV/AIDS, and many more which are preventible by explicit information to men, women, children and communities about how they should avoid or desist from risk behaviours and situations.

I INTRODUCTION ***(Nullius in verba).*** The concern expressed in this Statement is based entirely on data, facts and observations which are verifiable in the period 1982 – 2004 (Refs 1a – e). During this period, new registrations of HIV/AIDS in the UK rose until 1994 and then began to decrease almost symmetrically (Figure 1). This happened just after a Gaussian rise and fall in New York City (1b), the original epicentre, where the incidence of AIDS is still higher by orders of magnitude (Figure 2) than in London and the West Midlands of the UK, all with comparable urban populations . It began before the introduction of highly active anti-retroviral treatment (HAART), though previous improvements in combined antimicrobial and other therapy had already contributed, along with safe sex and the ABC (*Abstinence, Be faithful, Condoms*) of common sense to decreases in severity and mortality. Since then, total registrations of the various diseases officially classifiable as AIDS have decreased further in the resident population of the UK (1a). The Scottish Centre for monitoring HIV/AIDS made a characteristically subdued bur historic declaration by reporting in March 2004 (1c) that their data clearly demonstrate that HIV transmission in the indigenous heterosexual population in Scotland including injecting drug users is relatively rare; indeed, "*HIV is now almost exclusively confined to gay men and persons from abroad, especially African countries.*" This admits for the first time anywhere officially since 1985 that AIDS is not, repeat *not*, being spread by heterosexual transmission of HIV. The same trend has been maintained through 2004. Current data (1a) from the Health Protection Agency (Former PHLS) confirm that *the same can be said of England, Wales and Northern Ireland. This is true also in New York (1b), California and other conurbations in the USA (1d)* where registrations since 1985 show that AIDS is confined to original risk groups and to deprived or backward minority Black-Hispanic communities where it is as prevalent as in some developing countries, despite unprecedented efforts and unparalleled expenditure from public, private, charitable and industrial resources for detection and control

This difference is part of the global problem of the distribution of diseases and deprivations.. The pattern of AIDS, as registered maximally in the sub-Sahara and other parts of the developing world, is entirely different in epidemiology, clinical diversity, transmission and scale from diseases registered officially under the same heading (ICD/WHO 10th revision, B20-24) in developed countries. AIDS appeared, only in the USA, in 1981 as a new disease acquired by some homo- and bi-sexual men and drug addicts in affluent urban communities with preferred life-styles (1,2-4) that exposed them to overloads of sexually-transmissible and other infections which exhausted their natural immunity quickly and almost always fatally. Behaviours, especially ingestion of instant euphoriant drugs (Poppers) associated with these life-styles (4) were identifiable as markers and primary determinants of AIDS *which could therefore be almost entirely avoided or prevented by understanding this, as many people otherwise at risk soon did. This is why there was and still is no appreciable heterosexual or vertical spread in these countries.* In developing countries, in contrast, AIDS was not recognised until years later when it began to occur as a nondescript disease spreading explosively in underprivileged and unaware communities in whom it appears to be highly transmissible heterosexually and congenitally, though reliable data are still difficult to find (3). *The only common feature, in the large, arbitrary compendia of diseases in the ICD that are officially registrable anywhere as AIDS , is the assumption since 1984, still unproven, that a positive serotest for HIV mandates a diagnosis of AIDS.* In developing countries, this diagnosis is often made on symptomatic grounds under the Bangui definition of 1985 without

any tests (1e). Underlying this is the assumption that HIV has been proved scientifically to be the unique, necessary and sufficient common cause.of AIDS everywhere.

These assumptions arose firstly from medical presumptions that seropositivity to antigens from co-cultures with lympho-***pathic*** properties confirmed a diagnosis of AIDS in all its forms in anyone anywhere and, secondly, that onset and severity were governed by viral load, as measured in blood indirectly by amplifications of RNA presumed to come from a lympho-***tropic*** virus HTLV 3, later designated as HIV. Neither of these assumptions are upheld by the manufacturers who have adopted or been assigned responsibility for preparing test kits from co-cultires. The first assumptions were made and scientifically accepted as being valid on suggestive but minimal evidence in 1984, as described in para XX and references below. Both assumptions became an overnight consensus, fortified subsequently by an unprecedented volume of detail and rationalisation in over 100,000 publications in peer-reviewed journals and by *four arbitrary but official reclassifications (5)* expanding criteria for diagnosis, re-designating AIDS as HIV/AIDS, and leading immediately (Figure 2) to enormous increases in official registrations.in the USA and third world locations. In western Europe, symptomatic AIDS was actually diminishing and is now uncommon. This is what it always was (Figure 1, Table 2) everywhere in the UK except in limited locations in London and a few other cities (1a,e) in which many homosexual and bisexual men still choose to engage in high-risk behaviour, as described below. The same is true in western Europe (1e) but the incidence in the USA where AIDS began is, by comparison, enormous (Figure 2). In eastern Europe, including Russia and Ukraine, registrations of HIV/AIDS are increasing in parallel with the availability of new addictive drugs, spread of STD's and recreational risk behaviour (1d,e) in a latter day version of the urban plagues of contemporary society..

II The consensual HIV Hypothesis denies this and asserts that everyone everywhere is at risk of AIDS by heterosexual transmission of HIV. The arbitrary reclassifications of AIDS (5) promoting this view have been repeatedly upheld by WHO, UN Global AIDS, the US National Institutes of Health (NIH), the Royal Society of London, national health authorities everywhere and Statesmen in successive Declarations of Commitment, for instance in Washington DC 1984, London (1988), Durban (2000) and New York (2002). These Declarations - *denying any possibility of uncertainty* and denouncing any dissent, are justified by insistence that, although there is no curative drug or effective vaccine and, indeed "No end in sight" (6), any questions about why this is so are harmful, even criminal. In its contemptuous denials of factual evidence, willingness to deceive and *zero tolerance of alternative views*, never questioned in professional transactions or mainstream Media, this consensus is a wilful subversion of the ethic of medicine and soul of science. It has produced a defeatist impasse, and made reasonable doubt a stopper for discussion and effective action. It is surprising that so few professionals feel, and hardly any express, concern about this deception and blatant opposition to open debate. The Journal of Bioscience has however amplified earlier expressions of dissent in the Lancet (5) and Nature (6) by publishing recently a comprehensive review by Duesberg, Rasnick and Koehnlein (7). The present article amplifies and endorses most of their arguments but is critical of their epidemiology and misunderstanding of reasons for medical intervention and recovery.

III ANTECEDENTS of AIDS: In USA, Europe and Eurasia, attitudes and behaviour in

sexual matters became mildly and then extremely liberal and permissive in the 1960's, especially with the availability of the contraceptive pill. Homosexuality came out of the closet. Euphoriant drugs were ingested, injected and inhaled on a sociopathic scale. These changes, starting in the age group 20 –30, extended to teenagers and young middle-aged people of both sexes through the 1970's (2,4). A new age of risk behaviour linked paradoxically to expectations of Health for All was dawning. So was AIDS, along with a massive increase in traditional sexually transmitted infections which are continuing almost everywhere while AIDS is diminishing in the UK and other developed countries except in communities engaging in or subjected to continuation of high-risk behaviour (1 a-e,2-5).

IV ONSET: The communicable character and clinical features of the new syndrome had been quickly and accurately described by US Health Authorities (1b,d) in a few dozen young men in New York City and California in 1980 – 81. Since then, cases have been assiduously monitored and validated by agreed criteria, as below, in all developed countries. Through 1984, *3600* cases of AIDS were registered in New York City and *1400* in California. In both situations, it was a new, self-defining syndrome which young homo- and bisexual men acquired uniquely by engaging in sexual behaviour that exposed them to an insufferable load of traditional sexually-transmissible and other alimentary and respiratory infections that could easily spread by intimate contacts in bath houses, sex markets, bars and communal dwellings (2). The syndrome became fatal when immunity was exhausted by these habits, by secondary, opportunistic superinfections some of which became AIDS-defining. Many of these men had Kaposi's sarcoma which is associated with herpes viruses though still registrable as AIDS-defining. *More often than not, immunity was profoundly weakened also by inhalation of volatile erotogenic nitrites (poppers, which were by themselves rapidly and often irreversibly immuno-suppressive, or by shared injections of heroin and other toxic and addictive psycho-active drugs* deemed to be recreational (2,7). Female partners of bisexual men or drug addicts developed AIDS, and some transmitted this together with drug-dependence to infants congenitally or perinatally. This was and is relatively uncommon, even in New York City except in black-hispanic minorities (1b). In the resident population of the UK, perinatal transmission of AIDS is very rare (1a), except in drug addicts, certain immigrant groups and visitors (Table 1) many of whom are affected by tuberculosis and other infections.

V COURSE: From 1982 onward, AIDS was registered in smaller numbers of identical cases in the same risk groups in some major cities in the Americas, Europe and Australasia (1). By 1985, *6921* cases (*32 per million*) were registered in the USA but only *107 (1.8 per million)* in the UK (Fig 1) and *762 (2 per million)* in all of Europe. From 1982 through 2004, over a million validated cases were registered cumulatively in the USA, but only about 20,000 (Table 1) in the UK.. Globally, UN AIDS expects 29 million new cases by 2010 out of the global denominator of 6 billion. How the huge residue of 5,71 billion in theAsiatic denominator will escape is not considered in the arithmetic or logic of these projections.

VI MICROBIOLOGY. It is necessary to reconsider more antecedents, as follows. In 1970 in the USA, Baltimore (8) with Temin identified the enzyme reverse transcriptase (**RT**) in cellular cultures of classical RNA viruses, claiming that it came from, and characterised these as retroviruses because they transformed their RNA into nuclear DNA. Baltimore and Temin were awarded Nobel prizes for this undoubted advance in biochemistry, *though it was*

known at the time that RT was produced, independently of the viruses, by the mammalian cells in their complex cultures (9). Since then, it has been assumed that ***RT*** specifies the presence of live retrovirus. This claim has never been corrected by the Journals which published it, and are now firmly closed to correspondence on this basic deception and related issues - which include the facts that, independently of AIDS, endogenous retrovirus sequences might occur naturally in the human genome (10); and that the activity of RT contradicts the central dogma of the Watson-Crick hypothesis that RNA is released only from DNA as a messenger..

RT became a main issue in 1983 in Paris when it was identified in cellular co-cultures implanted with additional cells from an enlarged lymph gland excised from one well man considered to be at risk of AIDS. *This co-culture, reinforced successively with human and other mammalian cells, also yielded a cyto/lymphopathic agent designated Lymphadenoma–related Virus (LAV) because it was assumed that the RT came from a retrovirus (11).* The discoverers, Luc Montagnier and colleagues in the Pasteur Institute, sent the culture for confirmation of this to Robert Gallo at the National Cancer Institute (NCI) in Bethesda, USA. Gallo confirmed that a retrovirus was present in the co-culture but said that it was essentially the same as a lymphotropic retrovirus HTLV3 which he and his colleagues had already isolated from homosexual men with AIDS, but not from well men, in USA (12). The Pasteur Institute and NCI both claimed in patent applications that specific antibodies to crude protein antigens in the said co-cultures could be detected in sera from homosexual men sick with AIDS but not in sera from other, well men. The NCI in USA, the Pasteur Institute in France, the Institute for Cancer Research in London, named scientists and pharmaceutical firms were awarded patents for discovery and commercial exploitation of these claims, including production of antigens for diagnostic kits. Since then, detection of these antibodies in anyone anywhere mandates a diagnosis of AIDS, reclassified as HIV disease, with no ifs or buts or doubts. The word "Seropositive", which legitimately confirms specificity in routine diagnoses of many other communicable infections, entered dictionaries, lexicons, manuals on health education and all the Media as an exclusive synonym defining HIV/AIDS in all its forms everywhere as a fatal illness transmissible by sexual intercourse, needle-sharing, blood transfusiona and tissue grafts..

On this basis, it was presumed also that the lymphopathic agent LAV/HTLV3 in the co-cultures described above was the discovery of a transfection to humans of an ancient simian retrovirus.(19) which was declared in 1984, without direct isolation from patients by standard virological techniques or other corroborative research, to be "*Probably the cause of AIDS (12)*" Almost immediately, this agent, renamed HIV-1 in 1986, was accepted by the medical profession, scientists and health authorities everywhere as the unique, necessary and sufficient cause of AIDS. Levy and Jaffe in USA, Weiss et al in UK and many others then reported independent isolations of the same virus in cell cultures implanted with blood, semen, cervical and other secretions from patients with AIDS. These claims were shown later to be open to question (10) because later work confirmed that RT was produced also in cells and co-cultures of hepatitis C and other viruses, and that other antibodies cross-reacted with antigens presumed to be specific for HIV (11-14)..There were and still are no confirmed reports of isolates of HIV directly from patients or of electron micrographs of retrovirus free from co-culture material (15). Although cytopathic effects and syncytia in secondary

subcultures, and inclusions resembling viral or proviral particles are observed in hosts' mononuclear cells in various body fluids and tissues, these do not amount to proof of infection unless the virus itself is isolated. The same applies to a protein factor *Vif* and other biologically active products like *tat* and *gag* in complex cell cultures (12).
There was therefore plenty of room for doubt about HIV but nothing surfaced until 1987 when PN Magee, the Editor of Cancer Research, invited Peter Duesberg, a pioneer in retrovirology, to review the pathogenicity of these indeterminate viruses. Duesberg's work on viruses as oncogenes had him to doubt their pathogenicity. In his first review (7) he claimed that HIV was a latent virus devoid of pathogenicity; but he went further and reasoned that AIDS was caused by ingestion or injection of addictive drugs or was a recurrence of other diseases. This review, together with a further paper in 1989, marks the beginning of scholarly dissent from the HIV hypothesis. This has continued (13-19) with corroboration from other quarters. In 1990, Montagnier and his colleagues at the Pasteur Institute reported that co-cultures of HIV (LAV) were not cyto- or lympho-pathic unless a contaminant, a *Mycoplasma*, was present (16). Lo *et al.* in the US Army Laboratories (18) claimed that a *Mycoplasma* in their co-cultures was similar to that isolated by Montagnier *et al,* and suggested that this could be the cytopathic agent in AIDS. These findings also were ignored. So was the fact that – except for repeated expansions by reclassification (5) which mandated all seropositives, low CD4 counts and additional conditions in non-risk groups as HIV Disease - registrations of symptomatic AIDS diminished before anti-retroviral therapy was used routinely in the 1990's.in USA and UK (Figures 1and 2), as in other developed countries. From then onward, complicated schedules of anti-bacterial, anti-fungal and anti-protozoal and anti-viral drugs, described deceptively as "Highly active ant-retroviral treatment" (HAART), were used to combat and sometimes cure the various opportunistic and concomitant infections, including sexually transmitted diseases (STD's), which contributed, arguably along with HIV, to the complex and variable pathology of AIDS.

VII SEROLOGY: Prior to 1984, routine tests had shown that many, probably all men registered as AIDS had serological evidence of infections with hepatitis, herpes, varicella-zoster, cytomegalo- and other viruses (1,20). Since 1984, routine diagnostic tests to differentiate these infections in patients at risk of AIDS were discontinued and often superceded by serological tests for the HIV antigens described above which *are often indeterminate, or cross-react with antibodies in the blood of patients with about sixty other diseases or conditions (7,14-16.)* For this reason, the fluorescent ELISA.test was abandoned for diagnosis of AIDS by the Harvard Group in Africa (15) The confirmatory (sic) chromatographic test (Western blot) was discarded in 1994 by the Public Health Laboratory Service in the UK but is still widely used elsewhere, Seropositive results with ELISA do correlate well (8, 20) with appropriate symptomatology and risk behaviour but the antibodies detected are markers which do not signify specificity or immunity, or differentiate between former and current infection, or between treated and untreated cases. The four-fold rise in titre necessary for confirmation of active infection with other viruses is not required in diagnosis of HIV/AIDS. Reversions of positivity are not recorded. Despite these failings, routine tests by ELISA are used unreservedly, not only for diagnosis and registration of new and cumulative cases but also to track historical and palaeological origins of infection in humans from simians (19)

VIII SYMPTOMATOLOGY: In resident populations of industrialised countries, 80% or more of patients with AIDS are homosexual or bisexual men, drug addicts and their female consorts (1 a-e).Onset may be silent, or a feverish illness, chronic cough and pneumonitis due mainly to *Pneumocystis carinii*, stomatitis and oesophagitis due to *Candida albicans*, infectious enteritis with continuing diarrhoea due to various pathogens or none, anorexia, wasting and asthenia. Illness deepens, with collapse of immunity, susceptibility to opportunistic infections, asthenia and depression which worsen unless risk behaviour with sex and drugs is reduced or stopped (20). Failing this, and especially when volatile nitrites are inhaled (2,7,20), illness becomes desperate In 1980 - 82, fatality usually occurred within a year of diagnosis but awareness of the risks plus symptomatic treatment for the oral, respiratory, alimentary and opportunistic infections, and for malnutrition, delayed deaths for months and recently, with better treatment, for years. Much depends upon the pattern of behaviour and recognition of risks (1,2,14,20). Even so, the slogan in Berkeley USA in 1993 was "Die young and beautiful" until, a year later, improvements in combined therapy seemed to offer respite with a more protracted exit except that, in some communities, life-styles remained perilous because they embraced oro-anal, oro-genital, traumatic genital and other parasexual practises that caused oral, genital, colonic and rectal lesions, gross sepsis, fistulae, malodorous decay, exacerbation of infections with varicella-zoster, herpes, pyogenic and intestinal organisms: a burden of sickness which can still defy available and dedicated medical and surgical care - but remits in those who desist from risk behaviour (24)..
In developed countries, this sad and often visible picture of AIDS suddenly afflicting gay men, adolescents and pubertal boys, combined with outings of muted illnesses in celebrities, especially in showbiz, aroused popular and demonstrative compassion which attracted publicity for research and funding, endowing AIDS with a newsworthy glamour that *never presented it as what it was and is in enlightened communities: an illness which is largely self-induced and avoidable (24-26)*. In less sophisticated societies, AIDS and seropositivity to HIV are stigmatising, grounds for expulsion, rejection and death, often by mistake. It also attracts funding, often to the detriment of other exigencies, lethal diseases and elementary necessities like safe and adequate supplies of water (3). In such areas, in sub-Saharan Africa and elsewhere, AIDS is *a collage of disaster with no resemblance to the scene originally depicted in New York and California*, and then acquired in copy-cat fashion by risk groups elsewhere (1,2 14,20).

IX BIOMATHEMATICAL PREDICTIONS. Official projections (1) and mathematical models by designated experts, notably in the UK (6,22), envisaged exponential epidemics of tens or hundreds of thousands of fatal cases of AIDS by 1992 in the general populations of North America and Europe. But when the incidence and moving average of new cases registered as above or with lesser severity as AIDS since 1982, and as HIV/AIDS since 1984 in the UK, were plotted on a time scale, both showed a much lower, strictly linear increase in risk groups correlating ($\rho > 0.9$) with time, with minimal residual variance through 1992 (20,22). The trend from 1982 to 1989 accurately predicted new cases to within 10% of actual registrations.of new cases and cumulative distribution in risk groups through 1992 (Table 2). Shorter term prediction in homosexual men was similarly accurate, not only in the UK but also in New York City (24). This was consistent with continuation of risk behaviour within defined groups but not with external heterosexual or other transmissions of an infection caused by a single pathogen in general populations. Deaths, incompletely registered, followed

the same trend (Figure 1). Cases in women in the UK and NYC were few or nil except in risk groups through 1992. There has never been proof anywhere of spread of, or deaths from AIDS in general populations by heterosexual transmission of HIV except as part of a multifactorial aetiology which includes CMV, hepatitis, VZ and other virus infections, plus traditional STD's, immuno-supprssive drugs and needle-sharing (1-4,20,24)

All of this was predictable and was predicted (22,24). After 1993, realistic prediction became impossible because successive reclassifications by the US Center for Disease Control (CDC) and WHO expanded section B20-24 in the ICD to make 28 pre-existing diseases eligible for diagnosis and registration as AIDS-defining HIV Diseases (5,17). These included, for example, women with tuberculosis, cervicitis, carcinoma of the cervix, children with pneumonia or toxoplasmosis, asymptomatic seropositives, persons with low CD4 counts, and non-specific viral loads. Unsurprisingly, this unprecedented, unscientific and unethical manoeuvre did lead immediately (1b,d) to a predictable (5), doubling or threefold increase in cases and deaths registrable as AIDS (1b): a self-fulfilling prophecy which was then claimed to be an endorsement of prior claims that the outbreak was spreading by heterosexual transmission of HIV . *In fact, the passage of time since 1982 has shown no evidence of spread to general populations of developed countries (1a-e)) except in black-hispanic ethnic minorities in the USA, and in immigrants and visitors to the USA and UK from countries where AIDS and related STD's are prevalent (Tables 1 and 3).*. In the resident white population of the UK, cases of AIDS in women and infants are infrequent and data are insufficient for detailed analysis. In New York City, fuller and more informative data are available in continuous surveillance of seropositive women and perinatal AIDS since 1982. These data show (1b,20,24,25), as in the UK, low or near-zero frequency in the resident white population and in Asiatic communities - in whom the prevalence of HIV/AIDS is proportionally lower than in the resident white population - even allowing for the increase in registration produced by expansions of criteria for diagnosis of AIDS as HIV Disease in the 10th (1993) revision of the ICD and otherwise, as described above. This and other differences in the epidemiological patterns of AIDS are ignored by UN AIDS and the consensus in their projections of imminent and uncontrolled spread of AIDS in India, China and other countries in Asia.

X ASSOCIATION WITH TRANSFUSIONS: In 1983, it was noticed by the US CDC that a few patients showed signs of AIDS and seroconversion after blood transfusions (1d). Haematological changes similar to these associated with seroconversion occur also with transfusions of uninfected blood and blood products (24). This happened also, with much higher frequency, in men and boys with haemophilia type A who had received concentrates of anti-haemophiliac Factor VIII, and ceased when concentrates or fractions were heated to 60C or treated with detergents. The improvement is usually attributed to the elimination of HIV but this is questionable because pooled blood was and, in many countries, still is as likely or more likely to contain hepatitis and other readily-transmissible viruses (20). It is not certain that any of these viruses can survive after fractionation for factor VIII. Spouses of seropositive men show seroconversion but very few succumb to AIDS. There is no evidence of onward transmission to progeny or independent contacts. Data from years of experience are unavailable (See Table 1) Most patients who received infected blood or blood products before 1985 did not seroconvert or develop AIDS (7) but many showed immune responses to

other viruses, especially hepatitis B and C. Fundamental questions about technical, medical, domestic and legal aspects of seroconversion-related AIDS are overdue for attention (23,25). Meanwhile, it is assumed that seroconversion means that live HIV has been transmitted, that AIDS will inevitably develop and that anyone who is seropositive or develops any of the signs suggestive of AIDS after a blood transfusion or injection of blood products before 1985 can claim compensation. In some countries, this is held to justify prosecution of doctors, nurses and health authorities for negligence, assault, homicide or even murder..

X ANTIMICROBIAL CHEMOTHERAPY. Despite reclassification, new registrations of AIDS in the USA and UK dropped from 1994 onwards. It is claimed by the consensus that this fall was due to reduction of blood-borne load of HIV by specific treatment with antiretroviral drugs. This cannot be the explanation in the UK where annual incidence dropped from a peak of 1600 in 1994-95 to 600 in 2000-01 before there was time for treatment to reduce infectivity and where, despite safe-sex and condom programmes, STD's double or trebled after 1995. Antimicrobial drugs used before and since then in combined therapy are active also or more so against the collateral and opportunistic infections which form the AIDS syndrome. The same applies in sub-Saharan Africa where registrations of AIDS are immensely higher; but so also are overlapping STD's along with tuberculosis, enteritis, recurrent malaria and other lethal infections (3,13,14,20,24). Under the Bangui definition (1e,f, 3,13) almost any serious non-surgical illness can be legitimately registered as AIDS which therefore shows maximal incidence in seriously deprived tribal and shanty communities who are told that they are all are at risk of AIDS and that it is incurable without drugs or a vaccine, neither of which are available on a scale sufficient to treat the millions of cases registered by WHO and UN AIDS, and enlarged further by panic stations in the Media, activist quarters and health authorities..Duesberg and his colleagues among others have offered evidence (17) that some or even all of the nucleoside and other drugs used as anti-retroviral agents are intrinsically cytotoxic. Biologically, they belong to a rapidly growing range of xenotoxic substances. It is accepted even by the consensus that they can now be used only in reduced doses for perinatal prevention or HAART. Although beneficial results are claimed, it is not certain that these can be attributed solely to specific ARV effects. Registrations of AIDS were decreasing (Figures 1 & 2) before 1994 when the newer nucleosides began to be used (1a-d) .

XII SURVIVAL OF PERSONS DIAGNOSED AS SEROPOSITIVE:

In many countries including the UK, AIDS is not notifiable, testing is optional, permissive, surrogate and often indeterminate, contacts are not identified nor traced, surveillance is anonymous, biassed by re-classifications and absurdly incomplete; for instance in ignoring transmission to contacts. Deaths are usually registered under other headings. Surveillance in the UK by the Health Protection Agency show that, between 1985 and 1994, when annual incidence reached a peak of 1851 new cases, 43 – 76% of patients died of AIDS within 12 months of diagnosis, falling to 40% of 430 new cases in 2001. From 1982 to 2004 inclusive, 64599 persons were identified as seropositive of whom 20501 (32%) developed AIDS with 12937 deaths attributed to this, 2845 to other causes, and 48817 (74%) surviving. It is common knowledge everywhere that a considerable proportion – probably a majority - of asymptomatic seropositive men and women have survived for more than ten years from the

time of testing 17). Many homosexual men and female partners of bisexual men with AIDS experience the Lazarus-like remission of symptoms when they desist from, but relapse if they resume risk behaviour. It would appear that most female spouses or sex partners of seropositive haemophiliac men also remain well even if they seroconvert and that there are *few if any reports of tertiary transmission to progeny*. But there is in the UK no disclosure and, it would seem, no availability of any systematic follow-up of this important eventuality, nor of the incidence of seropositivity in wives, partners and offspring of military personnel known to have been exposed to AIDS while serving in areas of high prevalence. Nationally and internationally, access to raw data of incidence and relevant variables in this field seems to be denied: a unique exclusion from health statistics and freedom of information (27).

XII IMMIGRANTS AND VISITORS: Again, exact figures are seldom available. In the data base of theUK (Table 1),. the best estimate is probably obtainable from the numbers of seropositive and seroconverting women attending ante-natal clinics.or seeking attention for other reasons. Of 930 such patients in 1995-96, 619 (67%) were black African. This number rose to 5503 in 2002-3, of whom 3793 (69%) were black African – a highly significant excess, just as the number of 29 (0,5%) in Asiatic women was a much more highly significant deficit. The same disproportion exists in the cumulative totals since 1982 of registrations of AIDS (N=3364) in all women: 1993 (59%) in black Africans but only 62 (1.8%) in Asiatics. In New York City (1), with a cumulative total (1982-2001) of 27750 cases of AIDS in females, 15010 (54%) were black as were 1110 (56%) of N=1975 paediatric cases. There is therefore, for whatever reason, a disproportionate excess of HIV/AIDS in certain black populations, not only in sub-Saharan Africa. By the same token, it should be recognised that there are also black communities which do not show and justifiably deny this excess, and that in many populous urban and rural locations in west and especially in North Africa, HIV/AIDS is very much lower in frequency, morbidity and mortality than the releases of UNAIDS or even Sentinel Surveillance by the WHO would suggest.

XIII GLOBAL ESTIMATES. UN AIDS estimates that there were at the end of 2004 almost 40 million (35.9 – 44.3) adults and children living with HIV, 9 million (4.3 – 6.4) new cases more than 3 million deaths..This excess occurs almost entirely in developing countries, especially to sub-Saharan Africa but UN AIDS is careful in stating that "The AIDS epidemics coursing through this region are highly varied….. ….between and within subregions".. This admission verifies earlier references (3) to uncountable or uncertain numbers of cases, estimated by the elastic Bangui definition in unidentified backward areas without facilities for diagnosis, and extrapolations to general populations of surveillance data in unrepresentative samples of populations (5,13,14,24) Discrepancies between estimates have been reduced by Sentinel surveillance of returns but even those are often unvalidated. Nevertheless, UN AIDS and therefore the medical and lay Press of the USA and Europe insist that AIDS is more prevalent in South Africa than anywhere else because of heterosexual and vertical transmission of HIV. It is estimated that there are now 5.3 million psople living there with AIDS of whom about 2.9m are now women, with a sero-prevalence in pregnant women rising from 25% in 2001 to 28% in 2003. Closer analysis reveals significant regional variation, with three provinces reaching about 38%, more than twice that in the remainder. This is the position throughout the sub-Sahara where the excess prevalence is attributable to high rates in shanty or dislocated townships and former tribal territories. An

explosive increase recently in Botswana, where surveillance, education and facilities are or were good, and diamond mining is prosperous, suggests concurrent invasion by new infections, conceivably a bandit virus, perhaps related to HIV which is regarded by the Consensus as interminably mutable. All of this awaits explanation and rational intervention in the setting of life-styles, deprivations and environmental conditions as well as of serological testing, symptomatology and strategic treatment of all the diseases eligible for registration throughout Africa simply as HIV/AIDS, despite UN AIDS admission in their update of 2004 that "There is no single African epidemic"

UN AIDS says that the spread of AIDS by heterosexual and vertical transmission threatens the survival of economically-active adults in these countries. Their gloomy projections are taken at face value by economists (28) no less than by politicians, philanthropists and publicists seeking outlets for mega-million unaccountable donations in hard currencies. There is no doubt that shattering outbreaks registrable as HIV/AIDS are occurring. But vital statistics issued, also by the UN, show that three- or four-fold increases in populations of the affected sub-Saharan countries since the 1960's are continuing (Table 4), that the frequency of AIDS varies considerably, that easy money is seldom well-spent and often wasted for unaccountable reasons.. There are enormous increases also in all forms of STD's and other infections which overlap symptomatically, and in local perceptions and anxieties with AIDS. It is obvious from this and from the preceding paragraph that, in the USA, UK and in the sub-Sahara, black populations are exceptionally susceptible to such illness. Yet one finds parallel, propinquitous communities in black as well as non-black populations throughout Africa where this is not so, even where deprivations are comparable as in northern States. Locations, deprivations, life-styles, perceptions and behaviours require investigation as prime determinants.of AIDS and STD's. Claims (and donations) made without attention to these variables and to tribes, location, migrations, wars, malnutrition, collateral infections and inordinate increases in populations are shallow, deceptive, dangerous and often slanderous to young Africans who are repeatedly astounding richer countries with their athletic proficiency, cheerfulness, vitality and survival. The same can be said of India, China and Asia generally.

These virtues in all populations negate official pessimism and predictions of indiscrimuinate, uncontrolled spread of AIDS by heterosexual and perinatal transmission of HIV. But they may be insufficient to prevent STD's, AIDS, mutilation and other hardships in women in deprived communities, shanty towns and tribal areas. Dr Piot, Director of UN AIDS, estimates that women in sub-Sahara are 2.5 times more likely than men to contract all STD's, that this links with abuses of women, and accounts for most of the increase in AIDS (29). But it is linked also to fourfold increases in population, poverty , malnutrition, breakdown of hygiene and recurrence of endemic communicable diseases, all of which explains (30) why modern medicine cannot control multifactorial diseases like HIV/AIDS by focussing on a putative virus without comparable attention to other socio-biological factors and ABC. The foreseeable decline in AIDS in the UK as described in section I above, is offset by huge increases in chlamydia, gonorrhoea, hepatitis and genital herpes, a return of syphilis on a global scale and a huge overlap with HIV/AIDS. But there is a difference, concealed by reputable health authorities (1), standard texts like the Harvard Guide(32), and the HIV/AIDS hypothesis: AIDS is now decreasing in resident populations of developed countries (Figures 1 & 2) and is not an appreciable risk except in the original high risk groups of homo- and

bisexual men, drug addicts and their consorts who persistently engage in the sexual and other behaviours which led in the late 1970's to outbreaks of unmanageable overloads of various infections which exhausted their immunity and vitality, correctly labelled as Gay-related Immune Deficiency (GRID) by US Centers for Disease Control (1d) Despite arbitrary reclassifications, false claims and concealments by authorities, AIDS failed to spread by heterosexual transmission of HIV in general populations.in whom it is still an extremely rare or non-existent disease In routine practice, the reality and danger of this essential fact is that individuals presenting themselves for assessment or fear of STD's are often and in some clinics always denied a test for HIV in Europe and North America if they are not homosexuals or in other risk groups,

Nevertheless, it is now predicted by UN AIDS that India and China are verging on disasters due to HIV/AIDS, linked at present largely to the other main risk in life style, namely a rapid and communicable misuse of euphoriant drugs. There are certainly grounds for deep concern and, in the absence of verifiable microbiolgical and epidemiological data, it cannot be denied that there might be a spread since 1985 of a new highly infectious agent or a constitutional susceptibility. At present, there are discrepancies and differences between local, national and international situations and assessments, and an astounding dearth of critical and verifiable epidemiological analysis of distribution and risk factors (See section I). In contrast, *the virtual absence of HIV/ AIDS in all communities of Asiatic immigrants in UK and USA should be noted, especially since UN AIDS, WHO and the prevailing Consensus are unwilling or unable to differentiate between these communities, or between social strata and locations, or sentinel and Bangui surveillance, or validated and unvalidated data.* They prefer to rely, with the Consensus (6), on authoritarian declarations based on extrapolations and unreal mathematical models (Table 2) which were discredited but are still used even though the assumptions on which they are based are falsified by continuing errors, inconsistencies (1, 5,7,13,14,16,17,20,27,30-32) and demographic data (Table 3) In its construct of HIV/AIDS, this Consensus ignores verifiable data, plain facts and uncertainties and seldom discloses conflicts of interest (27,31). In this situation, claims of infallibility and zero tolerance of alternative viewpoints by the Consensus (6), editors, learned societies and allied pressure groups are absurd but formidable barriers to investigation and rational prevention because they do not hesitate to misuse medical wisdom to fuel an epidemic of fear that denies facts, rewards deception, impedes correction and disgraces medical science..

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