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This *Journal*, founded by the Medical Society for the Study of the Venereal Diseases, publishes original work on the investigation and treatment of genitourinary and allied disorders, and review articles, correspondence, and abstracts.

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(3) *The title* of the paper should be as brief as possible.

(4) *The number of authors* should be kept to the minimum, and only their initials and family names used.

(5) *Only the institution(s)* where work was done by each author should be stated.

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(9) *Figures* should be numbered in the order in which they are first mentioned, referred to in the text, and provided with captions typed on a separate sheet. (*Diagrams:* use thick, white paper and insert lettering lightly in pencil. *Photographs:* should be marked lightly on the back with the author's name and indicating the top, and should not be attached by paper clips or pins. They should be trimmed to include only the relevant section (sizes 2¼" or 5¼" wide, maximum 5¼" × 7") to eliminate the need for reduction. Photomicrographs must have internal scale markers. *X* ray films should be submitted as photographic prints, carefully prepared so that they bring out the exact point to be illustrated.

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AIDS were positive for antibodies to HIV-I compared with only 1/27 wives of seronegative controls.[2] Another study undertaken in Kinshasa showed that of 3000 couples tested, in 80 cases the male partner was infected, and in only 20/80 (25%) cases were both spouses infected.[3] The high rate of infectivity in the first Zaïrian study was probably because all the men had AIDS, whereas in the second study, as well as in ours, the subjects were clinically healthy.[1]

The incidence of heterosexual transmission that we report in the present survey is higher than in wives of men infected through blood products. Several hypotheses may explain this finding. In Africa, chronic stimulation of the immune system is more common than in American heterosexuals, thus possibly increasing the number of target cells that the virus can infect.[4] More specifically, the high prevalence of sexually transmitted diseases in Africa, particularly genital ulcers, may facilitate the transmission of HIV.[1,5] Finally, young African women may be more sexually active and thus at increased risk of infection.

This study highlights the need for prospective studies to assess more accurately the efficiency of heterosexual transmission of HIV and the possible role of cofactors in the spread of the disease.

Yours faithfully,

L Bélec
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1 Padian NS. Heterosexual transmission of acquired immunodeficiency syndrome: international perspectives and national projections. *Rev Infect Dis* 1987;**9**:947–60.
2 Mann JM, Quinn TC, Francis H, *et al.* Prevalence of HTLV-III/LAV in household contacts of patients with confirmed AIDS and controls in Kinshasa, Zaire. *JAMA* 1986;**256**:721–4.
3 Africa: vaginal sex inefficient in transmitting HIV. *CDC Aids Weekly* 1988; April 4:4.
4 Quinn TC, Piot P, McCormick JB, *et al.* Serologic and immunologic studies in patients with AIDS in north America and Africa. The potential role of infectious agents as cofactors in human immunodeficiency virus infection. *JAMA* 1987;**257**:2617–21.
5 Kreiss JK, Koech D, Plummer FA, *et al.* AIDS virus infection in Nairobi prostitutes: spread of the epidemic to east Africa. *N Engl J Med* 1986;**314**:414–8.

Notices

First congress of the European Academy of Dermatology and Venereology

The first congress of the European Academy of Dermatology and Venereology, for continuing education, will be held on 25–28 September 1989.

Topics will include: AIDS and dermatologists, what's new in treatment, dermatological surgery, warts and viruses, cutaneous histopathology, cutaneous immunopathology, and contact dermatitis.

For further information, please contact Centro Servizio Segreteria, EADV, Via Lapini 1, 50136 Florence, Italy.

Conference on vaccines for sexually transmitted diseases

A conference on vaccines for sexually transmitted diseases will be held on 5–7 April 1989 at Oxford University. It is sponsored by the journal, *Vaccine*, and the World Health Organisation.

For further information, please contact DE Cogan, Vaccines for Sexually Transmitted Diseases, Butterworth Scientific Ltd, PO Box 63, Westbury House, Bury Street, Guildford, Surrey, GU2 5BH (Tel. 0483 300966).

8th Meeting of the International Society for Sexually Transmitted Disease Research (ISSTDR)

The 8th meeting of the ISSTDR will be held on 10–13 September 1989 in Copenhagen, Denmark.

For further information please contact the meeting organisers: DIS Congress Service, Linde Allé 48, DK-2720 Vanløse, Copenhagen, Denmark or the scientific secretariat (Mrs Sandra Hyman), Statens Serum-institut, Neisseria Department, DK-2300 Copenhagen S, Denmark.

Correction

Detecting *Chlamydia trachomatis* by direct immunofluorescence using a Cytobrush sampling technique (August 1988;**64**:245–6)

We regret that an error occurred in the above paper. The second sentence of the first paragraph should have read:
"Direct immunofluorescence is adequately sensitive and specific compared with conventional culture."

Genitourin Med 1989;**65**:64–69

List of current publications

Selected abstracts and titles from recent reports published worldwide are arranged in the following sections:

Syphilis and other treponematoses

Isolation and characterization of recombinant *Escherichia coli* clones secreting a 24-Kilodalton antigen of *Treponema pallidum*

PEI-LING HSU, MINDE QIN, SJ NORRIS, S SELL (Houston, USA). *Infect Immun* 1988; **56**:1135–43.

***Treponema pallidum* invades intercellular junctions of endothelial cell monolayers**

DD THOMAS, M NAVAS, DA HAAKE, AM FOGELMAN, JN MILLER, MA LOVETT (Los Angeles, USA). *Proc Natl Acad Sci USA* 1988;**85**:3608–12.

Competitive enzyme-linked immunosorbent assay for *Treponema pallidum* antibodies

AA CODD, MS SPROTT, HK NARANG, PB CRONE, RH TURNER (Newcastle, England). *J Med Microbiol* 1988;**26**:153–7.

Recognition of *Treponema pallidum* antigens by IgM and IgG antibodies in congenitally infected newborns and their mothers

SRM DOBSON, LH TABER, RE BAUGHN (Houston, USA). *J Infect Dis* 1988;**157**: 903–10.

Western blotting was used to detect antigens of *Treponema pallidum* recognised by IgM, IgG, and IgG subclass antibodies in sera from six infants with congenital infection. Five of the infants had symptomatic infection, whereas one, born to a mother treated one month before delivery, was symptomless. The antibody response of the infected infants was compared with that of their mothers. Controls comprised sera from five infants born to mothers with negative cardiolipin antigen test results and no history of syphilis; serum pairs were taken from infants and mothers with either biologically false positive reactions (three pairs) or persisting reactivity in cardiolipin antigen tests (serofast) after adequate treatment of a previous infection (five pairs). The sera of infected infants was also examined for the presence of 7S and 19S rheumatoid factor.

Sera from infected infants contained IgM antibodies capable of reacting with 15 distinct treponemal polypeptides on western blotting. None of the cord sera from the five healthy infants gave an IgM reaction with any of the *T pallidum* polypeptides, but two of the five patients born to serofast mothers gave IgM reactions with the 190, 83, and 61 kilodalton treponemal proteins. Sera from infants born to mothers with biologically false positive reactions showed faint but detectable bands to several proteins. Eight of the 15 reactions between IgM and *T pallidum* were exclusive to the infected infants' sera, and were not present on blots reactive with sera from control infants. Only two, however, those with the 47 and 37 kilodalton polypeptides, were detected in each of the infected infants' sera. The serum response of the infant with symptomless infection was limited to the 47 kilodalton antigen only. In general, the number of IgM specific reactions in the infected infants exceeded the more limited response noted by their respective mothers who, on the basis of medical history, were in the early latent stage of disease. IgM rheumatoid factor was found in half of the sera from infected infants. Removal of the IgG substrate from the infants' sera did not affect the number of antigen reactions between IgM and *T pallidum* in western blotting.

The infected pairs of infants and mothers had IgG reactions to a wide range of proteins. IgG reactions in the infants were exclusively of the IgG1 and IgG3 subgroups and mirrored those of the mothers, except for IgG1 and IgG3 reactions to the 83 kilodalton protein, which occurred almost exclusively in the infants' sera. Although the infected pairs yielded more intense IgG bands to a wider range of antigens than did the serofast and biologically false positive pairs, no reactions were exclusive to the infected group.

As IgM antibodies to the 47 and 37 kilodalton protein were found only in sera from infants with symptomatic infection, detecting these antibodies at birth might form the basis of a more reliable test for congenital syphilis. Further study is required to evaluate the presence of these antibodies in patients with symptomless infection.

Hugh Young

Gonorrhoea

The production and characterization of monoclonal antibodies against the protein III of *Neisseria gonorrhoeae*

CG COPLEY, S MACFARLANE (Cambridge, England). *J Gen Microbiol* 1988;**134**: 1005–8.

Antibodies to the C epitope of *Neisseria gonorrhoeae* are present in patients with gonorrhoea and absent in normal sera

RDO HORMAECHE, H JESSOP, C BUNDELL (Cambridge, England). *J Gen Microbiol* 1988;**134**:1289–97.

Evaluation of a fluorescent monoclonal antibody reagent for identification of cultured *Neisseria gonorrhoeae*
CA ISON, A TANNA, CSF EASMON (London, England). *J Med Microbiol* 1988;**26**:121–3.

Successful treatment of gonococcal endocarditis with ceftriaxone
JR BLACK, JM BRINT, CA REICHART (Maryland, USA). *J Infect Dis* 1988;**157**:1281–2.

Non-specific genital infection and related disorders (chlamydial infections)

Severe *Chlamydia trachomatis* pneumonia in a patient with no immune deficiency.
Z SAMRA, A PIK, A GUIDETTISHARON, DB YAAKOV (Tel Aviv, Israel). *Arch Intern Med* 1988;**148**:1345–6.

Non-specific genital infection and related disorders (general)

Value of the gram-stained urethral smear in the management of men with urethritis
SJ LANDIS, IO STEWART, MA CHERNESKY, *et al* (Hamilton, Canada). *Sex Transm Dis* 1988;**15**:78–84.

Oral sex as a risk factor for chlamydia-negative, ureaplasma-negative nongonococcal urethritis
I HERNANDEZ-AGUADO, C ALVAREZ-DARDET, M GILI, EJ PEREA, F CAMACHO (Seville, Spain). *Sex Transm Dis* 1988;**15**:100–2.

Trichomoniasis

Treatment failure in *Trichomonas vaginalis* infections in females. II. In-vitro estimation of the sensitivity of the organism to metronidazole
WHR LUMSDEN, C HARRISON, DHH ROBERTSON (Edinburgh, Scotland). *J Antimicrob Chemother* 1988;**21**:555–64.

Candidiasis

Current perspectives in candidal vulvovaginitis
EG FRIEDRICH Jr (Florida, USA). *Am J Obstet Gynecol* 1988;**158**:985–1013.

Candidiasis in women fitted with an intrauterine contraceptive device
W PAREWIJK, G CLAEYS, M THIERY, H van KETS (Gent, Belgium). *Br J Obstet Gynaecol* 1988;**95**:408–10.

Genital herpes

Detection of herpes simplex virus DNA from genital lesions by in situ hybridization
A LANGENBERG, D SMITH, CL BRAKEL, *et al* (Seattle, USA). *J Clin Microbiol* 1988;**26**:933–7.

Evaluation of a new latex agglutination method for detection of antibody to herpes simplex virus
PC DEGIROLAMI, J DAKOS, K EICHELBERGER, S BIANO (Boston, USA). *J Clin Microbiol* 1988;**26**:1024–5.

Haemorrhagic cystitis due to herpes simplex virus as a marker of disseminated herpes infection
DA DEHERTOGH, LR BRETTMAN (Conneticut, USA). *Am J Med* 1988;**84**:532–5.

Oral acyclovir for treatment of first-episode herpes simplex virus proctitis
AM ROMPALO, GJ MERTZ, LG DAVIS, *et al* (Seattle, USA). *JAMA* 1988;**259**:2879–81.

The use of in-vitro sensitivity testing to predict clinical response of recurrent herpes simplex to suppressive oral acyclovir
DW SMITH, CS GOODWIN, (Perth, Australia). *J Antimicrob Chemother* 1988;**21**:657–64.

Virological screening for herpes simplex virus during pregnancy
PD WOOLLEY, CA BOWMAN, DA HICKS, GR KINGHORN (Sheffield, England). *Br Med J* 1988;**296**:1642.

Screening pregnant women for genital herpes
O CARNEY, A MINDEL (London, England). *Br Med J* 1988;**296**:1643.

Management of genital herpes in pregnancy
RS GIBBS, MS AMSTEY, RL SWEET, PB MEAD, JL SEVER (San Antonio, USA). *Obstet Gynecol* 1988;**71**:779–80.

Increased efficacy of phosphonoformate and phosphonacetate inhibition of herpes simplex virus type 2 replication of encapsulation in liposomes
FC SZOKA, C-J CHU (San Francisco, USA). *Antimicrob Agents Chemother* 1988;**32**:858–64.

Immunization with a vaccina virus recombinant expressing herpes simplex virus type 1 glycoprotein D: long-term protection and effect of revaccination
JF ROONEY, C WOHLENBERG, KJ CREMER, B MOSS, AL NOTKINS (Bethesda, USA). *J Virol* 1988;**62**:1530–4.

Genital warts

Sequence duplication and internal deletion in the integrated human papilloma virus type 16 genome clone from a cervical carcinoma
K-B CHOO, H-H LEE, C-C PAN, *et al* (Taipei, Taiwan). *J Virol* 1988;**62**:1659–66.

Human papillomavirus, herpes simplex virus and cervical cancer incidence in Greenland and Denmark. A population based cross-section study
SJ KJAER, EM DEVILLIERS, BJ AUGAARD, *et al* (Copenhagen, Denmark). *Int J Cancer* 1988;**41**:518–24.

Anogenital papillomavirus infection in children
JD ORIEL (London, England). *Br Med J* 1988;**296**:1484–5.

Evaluation of different DNA-DNA hybridisation techniques in detection of HPV 16 DNA in cervical smears and biopsies
MTE CORNELISSON, KJ van der VELDEN, JMM WALBOOMERS, *et al* (Amsterdam, Netherlands). *J Med Virol* 1988;**25**:105–14.

Detection of human papillomavirus capsid antigens in various squamous epithelial lesions using antibodies directed against the L1 and L2 open reading frames
JM FIRZLAFF, NB KIVIAT, AM BECKMANN, SA JENISON, DA GALLOWAY (Seattle, USA). *Virology* 1988;**164**:467–77.

Comparison of the cytobrush and cotton swabs in sampling cervical cells for filter in situ hybridization detection of human papillomavirus types 16 and 18 DNA
H-Q PENG, P ROTH, D CAUSSY, W RAWLS (Ontario, Canada). *Acta Cytol* 1988;**31**:311–3.

Urethral cytology of cytobrush specimens: a new technique for detecting subclinical human papillomavirus infection in men
S CECCHINI, I CIPPARRONE, M CONFORTINI, A SCUDERI, L MEINI, G PIAZZESI (Florence, Italy). *Acta Cytol* 1988;**32**:314–7.

A colposcopical lesion of the uterine cervix frequently associated with papillomavirus type 16 as detected by in situ and Southern blot hybridization: a cytohistological correlation study
C MORIN, C BOUCHARD, M FORTIER, R LEVESQUE, A MEISELS (Quebec, Canada). *Int J Cancer* 1988;**41**:531–6.

Human papillomavirus and herpes simplex virus in vulvar squamous cell carcinoma in situ
RH KAUFMAN, J BORNSTEIN, E ADAM, J BUREK, B TESSIN, K ADLER-STORTHZ (Houston, USA). *Am J Obstet Gynecol* 1988;**158**:862–71.

In this study the lesions of 62 patients with vulvar carcinoma in situ (CIN), and when possible the adjacent normal skin, were examined for the presence of human papilloma virus (HPV) DNA and herpes simplex virus (HSV) type 2 related antigens. Of the 62 biopsy specimens, 16 were unsuitable. Of the remaining 46 patients, three had recurrences biopsied, and 34 specimens of normal skin were also taken from 1·5 cm away from the margin of CIN.

Each specimen was examined by: a direct immunoperoxidase method to detect HSV type 2 induced antigen, infected cell specific polypeptides (ICSP) 34/35; DNA hybridisation to HPV types 6b, 11, 16, 18, and 31 under both stringent and non-stringent conditions; the avidin-biotin peroxidase method to detect HPV antigen using bovine papilloma virus type 1; and blind microscopy of adjacent sections of material stained with haematoxylin and eosin to confirm the diagnosis of CIN. The blood of 23 patients with grade III CIN was also tested for HSV specific antibodies by microsolid phase radioimmunoassay.

The results show that 83% (38/46) of the specimens contained HPV, and 48% (22/46) of the specimens contained HPV types 16, 18, or 31, either alone or in combination with types 6 or 11. However, 26% (12/46) of the specimens contained HPV types 6 and 11, either alone or in combination with other types. Testing for HPV antigen was not helpful as 22% (10/46) of specimens were positive. Half (23/46) of the specimens were positive for HSV type 2, and 41% (19/46) were positive for both HPV and HSV. Only 9% (4/46) were positive for HSV alone. Of the 34 specimens of normal adjacent skin, 18% (six) contained HPV DNA, which in each case was of the same type as that found in the CIN lesions. In only one patient was HSV type 2 related antigen found in the adjacent normal skin. Of the 23 blood samples tested for HSV antibodies, 74% (17) were found to be positive for type 2 and 65% (15) for type 1. Three samples gave negative results despite HSV type 2 antigen being found in the vulval skin.

The ages of patients in the study ranged from 21 to 79, and evaluation of the data in relation to age showed that unifocal disease was commonest in women over 35, which was in keeping with the finding that invasive carcinoma of the vulva is commoner in older women and with the unproven suspicion that invasive carcinoma arises more often in unifocal than multifocal lesions. No other variables differed with age, although HPV types 16, 18, and 31 were more (but not significantly) prevalent in the older age group. Local recurrences, which occurred in 9/43 patients followed up for a year, did not depend on the types of HPV present or the presence of HPV in normal adjacent skin, although recurrences were commoner (in 3/6) in those who originally had HPV DNA in adjacent normal skin than in those (6/27) who did not. The authors did not give data on other variables, such as multiple sexual partners or age of first intercourse, for these patients.

The paper contributes to the debate as to whether these viruses occur in association with genital neoplasia or are causative factors. The authors felt that the two viruses might act synergistically as a causative factor. Although the high percentage of specimens positive for HPV DNA might lend support to this as a causative agent, the lower figures for HSV type 2 antigen in the specimens do not. It would be interesting to know how many specimens were positive for HSV type 1 antigen as well. The paper also shows that, although 48% of the specimens were positive for types 16, 18, or 31 viral DNA, 37% were positive for types 6 or 11 viral DNA. This contrasts with the usual findings in CIN, but supports the findings of other workers who have found that HPV DNA types 6 and 11 were prominent in squamous carcinoma of the vulva.

M Weir

Verrucous carcinoma of the vulva containing human papillomavirus-11. Case report.
ME CROWTHER, JH SHEPHERD, C FISHER (London, England). *Br J Obstet Gynaecol* 1988;**95**:414–8.

Selective suppression of human papillomavirus transcription in non-tumorigenic cells by 5-azacytidine
F RÖSL, M DÜRST, H zur HAUSEN (Heidelberg, Germany). *EMBO J* 1988;**7**:1321–8.

Treatment of condyloma acuminatum with three different interferons administered intralesionally: a double-blind, placebo controlled trial
RC REICHMAN, D OAKES, W BONNEZ, *et al* (New York, USA). *Ann Intern Med* 1988;**108**:675–9.

Acquired immune deficiency syndrome

Molecular pathogenesis of human immunodeficiency virus infection
AB RABSON, S KOENIG, DF DAUGHERTY, HE GENDELMAN (Bethesda, USA). *Gene Anal Techn* 1988;**5**:41–53.

HIV/HTLV gene nomenclature
R GALLO, F WONG-STAAL, L MONTAGNIER, W HASELTINE, M YOSHIDA (Bethesda, USA). *Nature* 1988;**333**:504.

Activation of human immunodeficiency virus type 1 by DNA damage in human cells
K VALERIE, A DELERS, C BRUCK, *et al* (Pennsylvania, USA). *Nature* 1988;**333**:78–81.

Tests for infection with HIV: slandered goods
PP MORTIMER (London, England). *Br Med J* 1988;**296**:1615.

HIV isolation and antigen detection in infected individuals and their seronegative sexual partners
J ALBERT, PO PETERSON, S SCHULMAN, *et al* (Stockholm, Sweden). *AIDS* 1988;**2**:107–11.

Passively acquired human immunodeficiency virus seropositivity in a neonate after hepatitis-B immunoglobulin
SG ALBERSHEIM, JA SMYTH, A SOLIMANO, D COOK (Vancouver, Canada). *J Pediatr* 1988;**112**:915.

Loss of human immunodeficiency virus type 1 (HIV-1) antibodies with evidence of viral infection in asymptomatic homosexual men: a report from the multicenter AIDS cohort study
H FARZADEGAN, MA POLIS, SM WOLINSKY, *et al* (Bethesda, USA). *Ann Intern Med* 1988;**108**:785–90.

It has become evident this year that the

method of gene segment amplification using the polymerase chain reaction (PCR) adds another arm to the diagnosis of infection with human immunodeficiency virus (HIV). Instead of relying on the detection of the HIV antibody response, with minor additional contributions made by antigen detection and virus culture, it is now possible to seek previously defined segments of HIV proviral DNA in a specimen, amplify by many times the number of copies of the segment that are present, and then to probe for the chosen segment by hybridisation.

Farzadegan *et al* have used this approach to review the cases of four asymptomatic homosexual men who, out of 1000 who were seropositive, have been found to have reverted to a seronegative state. Case 1 was HIV antibody positive on his first two six monthly visits and negative on the third, fourth, and fifth visits. Proviral HIV DNA, however, was shown by PCR on visits 1, 2, 3, and 4. Case 2 was broadly HIV antibody positive on visit 1, but lost bands in western blot on subsequent visits and was HIV antibody negative by ELISA throughout. He was PCR positive on visits 2, 3, and 4. Case 3 lost western blot HIV antibody bands after his first visit and became negative on ELISA. He was PCR positive on visits 1 and 2, but negative on visits 3 and 4. Case 4 seronverted (by ELISA and western blot) at visit 2, became antibody negative at visits 3 and 4, and became positive again at visit 5. PCR gave positive results at visit 1 and 3, but negative at visits 4 and 5.

Plasma protein profiles and human leucocyte antigen (HLA) typing (again using PCR, to amplify HLA gene segments) showed beyond reasonable doubt that specimens had not been mixed up, thus excluding one possible explanation for the observed reversions to a seronegative state. Other explanations considered by the authors were: false positive serological reactions; low level contamination, for example by splash from an adjacent specimen; passive antibody, for instance from immunoglobulin injection; and immunisation with non-infectious HIV particles. They concluded, however, that the few people who lost antibody to HIV probably remained infected, as evidenced by the presence of proviral DNA.

Gene amplification has been so little used in diagnostic virology so far that its specificity is not known. Moreover, for HIV, showing HIV RNA (which can be achieved by the same technique as for HIV DNA) may be a clearer proof of infection and infectiousness as it implies HIV expression and virion production. It is also noteworthy that in case 4, PCR gave negative results after initially being positive. The interpretation of the findings is further complicated by the possibility of re-exposure and reinfection in this cohort, so that our understanding of the mechanisms of exposure to, infection with, and serological response to HIV has still to be considerably refined.

From a diagnostic point of view a few HIV infected people may plainly revert to seronegativity, and in some circumstances PCR may define their infectivity and also detect other HIV infected but seronegative people. PCR will be applied more widely in HIV diagnosis in the coming months, but it is already evident from this and some other current studies that, as these authors write, "HIV 1 infection [may be] undetectable by available serologic technology [adding] further, however minor, uncertainty to the control of HIV infection".

Philip Mortimer

Comparison of 10 enzyme immunoassays for detection of antibody to human immunodeficiency virus type 2 in west African sera
F DENIS, G LEONARD, A SANGARE, *et al* (Limoges, France). *J Clin Microbiol* 1988;**26**:1000–4.

Characterization of T lymphocyte responses during primary infection with human immunodeficiency virus
DA COOPER, B TINDALL, EJ WILSON, AA IMRIE, R PENNY (Sydney, Australia). *J Infect Dis* 1988;**157**:889–96.

Differential syncytium-inducing capacity of human immunodeficiency virus isolates: frequent detection of syncytium-inducing isolates in patients with acquired immunodeficiency syndrome (AIDS) and AIDS-related complex
M TERSMETTE, REY de GOEDE, BJM AL, *et al* (Amsterdam, Netherlands). *J Virol* 1988;**62**:2026–32.

Prognostic value of Langerhans cells in the epidermis of HIV patients
B DRENO, B MILPIED, JS BIGNON, JF STALDER, P LITOUX (Nantes, France). *Br J Dermatol* 1988;**118**:481–6.

Evidence for a cytotoxic T-lymphocyte alveolitis in human immunodeficiency virus infected patients
B AUTRAN, CM MAYAUD, M RAPHAEL, *et al* (Paris, France). *AIDS* 1988;**2**:179–83.

The transmission of AIDS: the case of the infected cell
JA LEVY (San Francisco, USA). *JAMA* 1988;**259**:3037–8.

The group-specific protein marker: a possible indicator of syphilis, not human immunodeficiency virus infection
DR POLLARD, P GILL, A DAY (Ottawa, Canada). *Can Med Assoc J* 1988;**138**:1013–6.

HLA haplotype A1 B8 DR3 as a risk factor for HIV-related disease
CM STEEL, CA LUDLAM, D BEATSON, *et al* (Edinburgh, Scotland). *Lancet* 1988;i: 1185–7.

Human immunodeficiency virus transmission among heterosexual couples in central Africa
M CARAEL, PH van de PERRE, PH LEPAGE, *et al* (Brussels, Belgium). *AIDS* 1988;**2**:201–5.

Epidemiological parameters of HIV transmission
RM ANDERSON, RM MAY (London, England). *Nature* 1988;**333**:514–9.

Unrecognised human immunodeficiency virus infection in emergency department patients
GD KELEN, S FRITZ, B QAQISH, *et al* (Baltimore, USA). *N Engl J Med* 1988;**318**:1645–50.

Clinical, haematologic, and immunologic cross-section evaluation of individuals exposed to human immunodeficiency virus type-2 (HIV-2)
RG MARLINK, D RICARD, S M'BOUP. *et al* (Boston, USA). *AIDS Research and Human Retroviruses* 1988;**4**:137–48.

This paper is a collaborative attempt by three groups of workers from Senegal, France, and the USA to document accurately the effects of HIV-2 on a cohort of seropositive prostitutes in Dakar, Senegal, using the variables mentioned in the title. A statistical comparison was then made with prostitutes and patients undergoing minor surgery who were HIV-2 seronegative using the western blot technique. Concomitant HIV-1 infection was excluded before subjects were enrolled in the study.

In total, 18 prostitutes were identified who were HIV-2 positive. No difference in incidence of concurrent sexually transmitted diseases (STD) was noted between the two prostitute groups; neither was there a difference on serological testing to a wide variety of other viruses (Epstein-Barr, herpes simplex types 1 and 2, cytomegalovirus, and hepatitis B) between the HIV-2 group and the control group.

A thorough clinical assessment of the two groups was then performed. No differences were noted on clinical examination, abdominopelvic ultrasonography, or skin testing for delayed hypersensitivity using standard antigens.

Analysis of the respective haematological variables showed no differences in haemoglobin concentrations, platelet counts, absolute white cell counts, or total lymphocyte counts. Subset analysis, however, did show fewer T4 cells in infected prostitutes than in controls, but this difference failed to reach significance at the 5% level. The lower T4 counts were attributed to the fact that the HIV-2 population was slightly older than the control group (45 *v* 34); multivariate linear regression analysis had already shown that age was the most important predictor of total T cell and T4 counts. T8 counts, on the other hand, were significantly higher in the seropositive group, which gave rise to a low T4/T8 ratio. Concentrations of β microglobulin and immunoglobulin G were increased, though those of IgM and IgA were not.

The authors concluded that HIV-2, a sexually transmitted retrovirus more akin to the simian T-lymphotrophic virus type 3 than to HIV-1, did produce a pattern of disease different from that of HIV-1, particularly in clinical presentation in their study group, but that long term follow up of their cohort was required. The last point is probably worth further comment. The study was only started in 1985, and therefore only three years of follow up have taken place. Recent studies using similar virological tests have shown that HIV-2 alone can cause full blown AIDS, but that the incubation period can be a decade or more. HIV-2 is possibly less "virulent" than HIV-1 and may produce a more heterogeneous disease pattern that will become clearer with time.

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