

LETTERS TO THE EDITOR

Carbamazepine in Reiter's syndrome

EDITOR,—A psoriatic spectrum with Reiter's syndrome as the most severe manifestation occurs with greater frequency in HIV infected individuals.¹ Immunosuppressive therapies for RS are associated with a poor response and increased morbidity.² We describe a case where carbamazepine showed an excellent response in an HIV infected patient with Reiter's syndrome.

A 30 year old married man presented with erythematous papules and plaques of 2 months' duration covered with hard limpet-like scales on face, body, and both extremities (fig 1). Palms and soles showed keratoderma blenorrhagicum and subungual hyperkeratosis with distal onycholysis. Both knees and wrists had painful swelling with restriction of movements. With this clinical presentation Reiter's syndrome was inferred. All routine investigations were normal except a raised erythrocyte sedimentation rate of 100 mm in the first hour. *x* Rays of the affected joints were normal. ELISA for HIV-1 and HIV-2 was positive with two kits (Immunocomb, Tri-dot) and confirmed with western blotting technique (Speciality Ranbaxy Limited). The absolute helper T lymphocyte count was 435 cells $\times 10^9/l$. Human leucocyte antigen B27 and rheumatoid factor were negative. The patient was commenced on prednisolone by mouth 60 mg daily and indomethacin by mouth 25 mg three times daily without any concomitant antiretroviral therapy. New erythematous papules and plaques appeared with no relief in joint pain and swelling.

In seeking an effective treatment, we serendipitously came across the efficacy of carbamazepine in an HIV infected patient with psoriatic erythroderma.³ We started carbamazepine 200 mg daily in two divided doses in addition to above. The erythema cleared rapidly within 7 days. To confirm the effect of carbamazepine, it was stopped. New lesions similar to the old ones appeared within 3-4 days. Carbamazepine was then reintroduced in the same dose. Erythema cleared again within 7 days followed by scaling and joint swelling and pain. New lesions stopped appearing. Prednisolone was then tapered off rapidly and analgesics were stopped. Carbamazepine was continued in the same dose for 6 months. On follow up at 1 year, the patient showed no recurrence of

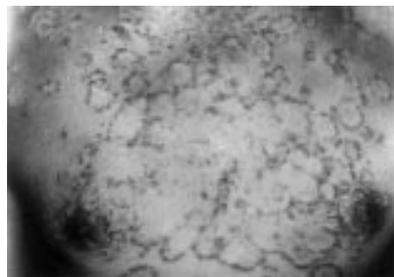


Figure 1 Close view of erythematous annular papules and plaques on chest before carbamazepine therapy.

skin lesions and synovitis, no change in liver and renal function tests, with no further deterioration in his overall health and no opportunistic infections.

It has been proposed that in genetically predisposed people, the release of neuropeptides like substance P, calcitonin gene related peptide, vasoactive intestinal peptide, and the inflammatory leucotriene B4 from cutaneous sensory nerves causes local inflammatory responses that trigger psoriasis.⁴ Stimulated mast cells secrete a number of proinflammatory cytokines and proteases that act similarly.^{4,5}

Carbamazepine significantly inhibits the uptake of noradrenaline (norepinephrine) and blocks a cyclic AMP mediated calcium influx that is associated with neuropeptide release and control of a slow potassium current.⁶

The rapid clearing of erythema, secondary to raised levels of neuropeptides, with carbamazepine may have been mediated through inhibition of these neuropeptides and by inhibition of uptake of noradrenaline. The exacerbation and subsequent resolution of lesions on withdrawal and reinstatement of carbamazepine respectively proves its efficacy in our patient. Also, the clinical remission maintained for 1 year after stopping carbamazepine confirms its therapeutic role in Reiter's syndrome. The therapeutic response seen in our patient conforms to that seen in the HIV-1 positive patient of Smith *et al.*³

This apparent success adds carbamazepine to the armamentarium against Reiter's syndrome in an HIV infected patient. This is the first reported case and an evaluation of long term carbamazepine therapy is warranted.

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Condoms and warts

EDITOR,—Wen *et al.*¹ should be applauded for their attempt to address the key question of whether or not condoms protect people from genital warts. However, some of the major study variables need clarifying, as they did not match up with my knowledge of the Sydney Sexual Health Centre (SSHC) database.

The article discussed the issue of "acquisition of genital warts" and was presented as an incidence study. Cases were defined as: "All

patients with a new diagnosis of macroscopic genital warts who attended SSHC [in] 1996." However, many of these patients had been previously diagnosed with genital warts elsewhere while others had recurrent lesions. In Australia, most genital warts are managed by general practitioners.² Consequently, the experience of specialist services is biased towards recurrent and difficult cases. "New diagnosis" in this situation means new to the clinic but not necessarily new to the patient. This means that the main outcome measure was a mixture of incident, prevalent, and recurrent cases, with the possibility that the warts may have affected the behaviour of many of the study subjects.

The SSHC database does document whether a person has previously been diagnosed with HPV infection. To me, the study would have had more validity if patients with a past history had been excluded.

The diagnostic grouping for warts at SSHC does not distinguish between genital and anal lesions. The readers of the journal need to know that many of these male "genital wart" cases would have been homosexually active men with anal warts. This is important as risk factors for penile and anal warts may differ, potentially confusing the results of the present study.

Originally developed as an HIV risk measure, the condom use variable at SSHC only refers to the previous 3 months or since the last registration/disease episode. Wen *et al.*'s article¹ failed to mention that this variable was time limited. As 3 months is the median duration before the appearance of exophytic warts,³ up to half of the relevant sexual behaviour may have been overlooked.

Finally, the referent group in the table describing condom use deemed as "Not applicable, no sex" should have been more accurately described as "No vaginal or anal sex in the previous 3 months." Many of these people would have practised oral sex or other sexual acts during those 3 months. Others may have ceased practising vaginal or anal intercourse up to 3 months earlier because of their persistent or recurrent warts.

Large relational quality assured clinical databases can be powerful tools for health service evaluation, surveillance, and the generation of research questions. It may be prudent for researchers to engage the people responsible for designing and maintaining those databases to minimise errors of interpretation.

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Reply

EDITOR,—We are grateful to Dr Dayan for her helpful and constructive comments. The major criticism of our paper relates to the selection of cases, and the possible inclusion

of prevalent and recurrent cases as well incident cases. However, our concern with this possible bias at the outset of the study led us to exclude all patients with a history of previous genital warts. This included those previously diagnosed at SSHC, and those who gave a history of having their warts managed elsewhere. Consequently, when we state a new diagnosis of genital warts, this is precisely what we mean.

With regard to the conduct of the study, this was performed with the assistance of the current data manager responsible for the SSHC data base, whose help and assistance were duly acknowledged.

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Photosensitivity reaction to efavirenz

EDITOR,—The non-nucleoside reverse transcriptase inhibitor (NNRTI) efavirenz is a recent addition to the armamentarium available to physicians in the treatment of HIV infection. However, at present the known side effect profile of this new agent is still in its infancy. We would like to report a case of photosensitivity associated with efavirenz.

A 27 year old white homosexual man was commenced on combivir (zidovudine/lamivudine) and efavirenz in March of 1999. One month later he reported that he was well and had no major side effects associated with his new combination. However, 4 weeks further into treatment he represented with an itchy rash affecting his arms and hands. On examination there was a maculopapular rash over the affected area but there was no oral ulceration, conjunctivitis, or fever. A drug reaction was diagnosed and he was prescribed antihistamines and asked to continue with his medication. One week later the rash had subsided. Then having spent a day outside in the sun he had a florid recurrence of the rash over the exposed areas (arms, back of neck, face, and ears). The rash was significantly worse over his elbows where there was obvious blistering and oedema. His medication was stopped and 3 weeks later the rash had completely resolved. Hepatitis C antibody and porphyria screening were negative. This man had been diagnosed as HIV antibody positive in June 1997. In March 1998 his viral load was 356 790 copies/ml (Roche PCR) and his CD 4 count was 512×10^6 cells/l, he was commenced on dual antiretroviral therapy with stavudine and didanosine (patient choice). Initially he did very well as the viral load became undetectable (<400 copies/ml). However, after 9 months on this combination his viral load began to rebound (5192 copies/ml) and a change in antiretroviral therapy was initiated to combivir and nevirapine which he initiated in the normal way (dose escalation at 2 weeks of nevirapine). He was started on this combination as he wished to take a protease sparing regimen. However, 1 week later he developed a rash affecting his entire body, especially his trunk and arms, associated with enlarged lymph nodes and constitutional symptoms, fever, and lethargy. In view of the constitutional symptoms it was decided to stop this present combination. One month later, the

rash had settled, he then commenced combivir and efavirenz.

Photosensitivity in the context of HIV has been reported as a presenting sign of underlying HIV infection in a number of cases.¹⁻³ In addition to this porphyria cutanea tarda (PCT) has been reported in the context of HIV infection and has been associated with concomitant hepatitis C infection⁴; however, screening for both these conditions was negative. Switching from nevirapine to efavirenz in this context may have been regarded as unwise; however, of 19 patients who have been intolerant of nevirapine secondary to the development of rash, who have switched to efavirenz only nine have developed a mild to moderate rash, of which only two needed to discontinue therapy.⁵ Photosensitivity in the context of HIV infection may not only be a presenting condition but also secondary to concomitant treatment.

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HIV associated cytomegalovirus retinitis in Melbourne, Australia

EDITOR,—We report the results of a 12 year review of human immunodeficiency virus (HIV) associated cytomegalovirus (CMV) retinitis in Melbourne, Australia.

We conducted a retrospective review of all HIV infected patients diagnosed with CMV retinitis at Fairfield Hospital and the Alfred Hospital between 1984 and 1996, aiming to identify factors at diagnosis of CMV retinitis which were predictive of outcome. Both hospitals had the same protocol for the treatment of CMV retinitis and employed 3 monthly ophthalmological screening of all HIV infected patients with CD4 counts of less than $50 \times 10^6/l$.

The study outcomes were visual loss and death. Moderate visual loss was defined as a visual acuity of less than 6/12 in the better eye, and severe visual loss as visual acuity of less than 6/60 in the better eye (this is legal blindness in Australia).

CMV retinitis was diagnosed in 212 of 1281 patients (16.5%) with AIDS over the study period. As of June 1998, 193 (93%) had died, at a median time of 36 weeks (range 0-192) from CMV diagnosis. Seventy four patients (35%) developed moderate visual loss at a median time of 23 weeks (range 0-163) and 30 patients (14%) developed severe visual loss at a median time of 35 weeks (range 0-120) from diagnosis of CMV retinitis.

The presence of visual symptoms at diagnosis of CMV retinitis was predictive of the development of moderate visual loss (relative risk 2.1, 95% confidence interval 1.1-4.2). Fifty eight of 138 patients (42%) with visual symptoms at diagnosis developed moderate visual loss, compared with 16 of 64 patients (25%) who were asymptomatic at diagnosis ($p=0.02$). The presence of visual symptoms at diagnosis was not predictive of the development of severe visual loss, or early death ($p>0.2$). Other factors measured at diagnosis of CMV retinitis included the patients' age, CD4 count, weight, visual acuity, and the presence of any previous AIDS defining condition. None of these was associated with the development of visual loss or early death ($p>0.1$).

The advent of highly active antiretroviral therapy (HAART) has resulted in a reduction in the incidence of new diagnoses of opportunistic infections. Prolonged survival times with CMV retinitis have been demonstrated in patients who achieve immunological recovery with HAART.² The ability to predict those patients who are at highest risk of visual loss may assist in advising those who may reasonably cease maintenance therapy for CMV retinitis following immune restoration. An understanding of the natural history of CMV retinitis in the pre-HAART years remains important in managing patients who are failing HIV therapy.

The only factor measurable at diagnosis of CMV retinitis that was predictive of outcome was the presence of visual symptoms. The use of routine ophthalmological screening in HIV infected individuals with low CD4 counts aims to detect CMV retinitis before visual symptoms occur. It is possible that visual loss may be prevented by detecting disease before retinal damage occurs. A prospective evaluation is needed to confirm this finding.

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Azithromycin v oxytetracycline for the treatment of non-specific urethritis

EDITOR.—Single dose azithromycin 1 g rather than multidose tetracyclines or erythromycin over several days for the treatment of chlamydial infections is becoming more widespread as patient acceptability and improved compliance outweigh cost considerations. However, in men, treatment is often initiated on the basis of microscopic evidence of urethritis before the chlamydial result is available. Relatively few studies report the efficacy of azithromycin in the treatment of non-gonococcal non-chlamydial urethritis (NSU),^{1,3} but recently published evidence based guidelines for the management of NSU recommend either doxycycline 100 mg twice daily for 7 days or azithromycin 1 g immediately.⁴

In this genitourinary medicine clinic azithromycin became first line treatment for all proved or suspected chlamydial infections from 1 April 1998. This retrospective study assessed the efficacy of azithromycin for the treatment of NSU compared with oxytetracycline 250 mg four times daily for 7 days, the previous first line treatment regimen for men with microscopic urethritis in whom no Gram negative diplococci were evident.

The outcome of all men with NSU diagnosed between 1 April 1998 and 30 September 1998 (treated with azithromycin) was compared with those diagnosed between 1 April 1997 and 30 September 1997 (treated with oxytetracycline).

NSU was defined as the presence of at least five polymorphonuclear leucocytes (PMNL) in five or more fields on microscopy of a urethral smear, negative culture of *Neisseria gonorrhoea* after direct plating onto modified New York culture medium and negative chlamydial screen on ELISA testing (Syva) of a urethral swab.

"Cure" was defined as either resolution of symptoms or clearing of previously positive two glass urine. A repeat urethral smear was not examined routinely.

"Treatment failure" was defined as persistent PMNL on microscopy of a urethral smear taken because of ongoing symptoms or persistent positive two glass urine test, with possibility of reinfection denied.

The results (see table 1) demonstrate that azithromycin is as effective as oxytetracycline in curing NSU, and produces fewer treatment failures, possibly owing to better compliance with single dose therapy. Compliance with multidose regimens might be expected to be less good in asymptomatic patients, but with no satisfactory "test of cure" this was difficult to ascertain. Overall, there was a 25% non-attendance rate for follow up, biased towards the asymptomatic patients and those treated with oxytetracycline.

Table 1 Comparative age, symptoms, and response to treatment of the two groups

	1997, oxytetracycline	1998, azithromycin
Number treated	76	52
Median age (range)	28 (18–63)	25 (16–54)
No with symptoms (%)	35 (46)	25 (48)
No cured (%)	29 (38)	27 (52)
No treatment failures (%)	6 (8)	0
Outcome uncertain*	41 (54)	25 (48)
Symptomatic dna	8/35 (23)	4/25 (16)
Asymptomatic dna	13/41 (32)	7/27 (26)

*Originally asymptomatic with clear two glass urine; did not reattend (dna); possibly reinfecting.

The results of the two glass urine test did not differ significantly between the two groups but overall was positive in 70% of symptomatic patients compared with only 47% asymptomatic ($p < 0.01$). Its low sensitivity and specificity⁴ are likely to be even lower in asymptomatic patients. Default from follow up occurred more frequently in the asymptomatic patients, but was less evident in the azithromycin treated group, who had a lower default rate overall, as previously reported.⁵

In conclusion, although the numbers are small, it would appear that azithromycin is an effective treatment for NSU, and can be given at the time of initial diagnosis, pending the chlamydial result. Financial considerations preclude the use of azithromycin as first line treatment for NSU in many centres, but better compliance resulting in fewer treatment failures, and fewer wasted appointments from defaults may counter the economic argument.⁵

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Sexually transmitted infections in elderly people

EDITOR.—Jaleel *et al* recently presented the incidence of sexually transmitted infections and other conditions among elderly people attending a genitourinary medicine clinic.¹

We, in our genitourinary medicine department at Royal Berkshire Hospital, Reading, studied the reasons for attendance of elderly people and compared them with the younger age group. Data were collected from patients aged 60 and above who attended the clinic between January 1998 and December 1998. Randomly selected sex matched people aged 20–35 years are taken for comparison.

A total of 68 elderly people attended the clinic. The mean age was 66.5 years (range 60–83); 61 (90%) were male and seven (10%) were female. Forty one (60%) attended for STI screening and 27 (40%) attended for non-STI management. In the younger age group 60 (88%) attended for STI screening and eight (12%) attended for non-STI management ($p < 0.001$). Sixteen (24%) older attendees had an STI compared with 35 (51%) in the younger age group (see table 1). Of the 16 older attendees with suspected STIs 11 (68%) waited over 2 weeks between symptom recognition and clinic attendance. Of 31 symptomatic attendees in the younger age group 10 (32%) waited over 2 weeks for symptom recognition and clinic attendance ($p < 0.001$).

Table 1 Diagnoses of older and younger clinic attendees

	Older clinic	Younger clinic
(No of patients)		
STIs		
NSU	7	21
Latent syphilis	3	
Genital herpes	2	1
Genital warts	1	11
Gonorrhoea	1	2
<i>Trichomonas vaginalis</i>	1	
HIV	1	
Other conditions		
Erectile dysfunction	15	1
Balanitis	9	1
Lichen sclerosus	1	
Zoon's balanitis	1	
Genital psoriasis	1	1
Genital ectopic sebaceous glands		1
Genital skin tag		1
Inguinal hernia		1
Genital sebaceous cyst		1
Miscellaneous (hepatitis B vaccination)		1

Many elderly people maintain heterosexual and homosexual activity. Therefore this age group is at a risk of all sexually transmitted infections.² In our study, a smaller percentage of older attendees had STIs compared with previous studies.^{1,3} However, the number of older patients who attended for non-STI management are comparable. The delay between symptom recognition and healthcare presentation is a feature of STI related illness behaviour. The delay behaviour among individuals with suspected STIs is age specific, with longer latency periods experienced by people over the age of 50.⁴ This finding was seen in our study as well.

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Tertiary syphilis

EDITOR.—I read Dr Reed's letter on tertiary syphilis¹ with interest.

The regimen he describes for the treatment of early syphilis—arsenic, bismuth, and round the clock aqueous penicillin, was used in our hospital from 1946–8² although daily penicillin in beeswax was also used. It was unclear how much inactive penicillin K was in the commercial product used. The penicillin regimen used here was higher than in Lincoln (40 000–75 000 units 3–4 hourly). There were 10 treatment failures (re-infections) out of 275 patients described.²

Treponema pallidum remains viable in the CSF even after adequate clinical treatment.^{3,4}

The old adage that we achieve clinical but not microbiological cure of syphilis with antibiotics is probably true.

It is likely that most people in developed countries nowadays who have untreated syphilis have received treponemoidal antibiotics for other intercurrent infections, so that any neurosyphilis that developed would either be modified⁵ with few physical signs or would be completely treated and clinically cured. However, others disagree with this.⁶

But, to answer Dr Reed's question, we haven't seen anyone treated since the second world war who has developed neurosyphilis in subsequent years.

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BOOK REVIEWS

Infectious Diseases. By Donald Armstrong and Jonathan Cohen. Pp 2000; £250 (two volumes). London: Mosby, 1999. ISBN 0723 423288.

The most striking first impression of these two volumes is the lavish production with marvellous illustrations, photographs, and tables. It has many excellent features. The text is well set out and easy on the eye. The experience of the authors in approaching various diseases and clinical syndromes comes through strongly. The sections comprehensively cover infectious disease from basic science to clinical management. The clinical microbiology section is an important anchor and could be a short textbook in itself. I very much enjoyed the numerous practice points, which are oriented towards clinicians faced with funding solutions to problems. These consist of short essays with tables or illustrations and tackle particular clinical problems such as "the diagnosis of HIV in newborns," "what is the treatment of a positive toxoplasma titre in pregnancy?" or are in a debating style—for example, "how long should osteomyelitis be treated?"

Each section is colour coded and although the American numbering system takes a few minutes to get used to one can easily navigate around the book. The contributors are all internationally famous in their fields and, with so many of them, I am quite impressed by how up to date the book is. They must have been chased hard to get their contribution in on time. One of the few criticism

would be that there could have been more on hepatitis C and its interaction with HIV.

However, if you can't find what you want in this book, there is a comprehensive list of websites, which are of interest to infectious disease and other physicians. There is a free CD ROM which creates a direct internet link to these sites. The other important resource is a slide library, which comes on the same CD ROM. In all, 1500 tables and clinical and other photographs are stored and can be made up into personalised presentations; these can then be used as a teaching resource via computer generated images. The high quality of these images will impress anyone involved in producing material for teaching. However, it is a shame some of the useful tables have not made it from the text to the CD ROM.

Although this book is expensive, I would recommend it to anyone interested in infectious diseases especially those who have to teach at any level, undergraduate or post-graduate.

With the rise of the internet the big textbook might soon be heading for extinction. Thankfully this book delays the time when I will be downloading information from the super highway rather than turning over the pages of a well produced book. If I need to use my computer there is always that free CD ROM.....

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Review of ABC of Sexual Health. Ed John Tomlinson. Pp 60; £14.95. London: BMJ Books, 1999. ISBN 0-7279-1373-5.

I was delighted when the editor sent me this book and asked me to review it. I had looked forward with anticipation to the original series that were published in the *BMJ*. I had thought then that each article was just superb and now they are all neatly packed together in this ABC, I am of the opinion that this is an excellent book which achieves its aim completely. On the cover, it says "it is an ideal reference for doctors, nurses, students and all those not involved in the area of sexual health," and Professor Adler adds in the foreword that this book will put the profession in touch with the real world, real people, with real problems, and fill a large gap in our knowledge.

John Tomlinson, the editor, has pulled together an excellent group of experts who have practical experience in the field and have managed to condense that experience into a series of short articles, all of which make informative, yet entertaining reading. In my opinion, no specific background is required to gain information from these articles and I have recommended specific sections of this book for individual patients who need to read about their problem.

Those of us who work in sexual medicine were amused that the *BMJ* had to carry a warning about the sexually explicit material inside and, indeed, John Tomlinson refers to this in the preface and admits that a very small number of readers were offended. However, given the general reticence in society about sexual matters, this is not surprising.

Sexual health is an essential part of having a happy and fulfilling life, and everyone who works in a caring profession should be

comfortable when the conversation drifts into areas of sexuality. Patients, who often broach the topic with trepidation, need to be assured of a sensitive hearing. In my opinion, this excellent book will give anyone in the caring profession a good grounding in sexual matters, so that they can explore these areas with patients when appropriate, without embarrassment and have some idea of likely strategies of management.

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NOTICES

International Herpes Alliance and International Herpes Management Forum

The International Herpes Alliance has introduced a website (www.herpesalliance.org) from which can be downloaded patient information leaflets. Its sister organisation the International Herpes Management Forum (website: www.IHMF.org) has launched new guidelines on the management of herpesvirus infections in pregnancy at the 9th International Congress on Infectious Disease (ICID) in Buenos Aires.

Pan-American Health Organization, regional office of the World Health Organization

A catalogue of publications is available online (www.paho.org). The monthly journal of PAHO, the Pan American Journal of Public Health, is also available (subscriptions: pubsvic@tsp.sheridan.com).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics, and Gynaecology, Advanced Course for Obstetricians and Gynaecologists, 19-23 June 2000

Further details: Symposium Office, Imperial College School of Medicine, Queen Charlotte's and Chelsea Hospital, Goldhawk Road, London W6 0XG (tel: 020 8383 3904; fax: 020 8383 8555; email: sympreg@ic.ac.uk).

Australasian Sexual Health Conference, Ven Troppo, Carlton Hotel, Darwin, Northern Territory, 21-24 June 2000

Further details: Shirley Corley, Conference manager, Dart Associates, PO Box 781, Lane Cove, 2066 NSW, Australia (tel: 02 9418 9396/97; fax: 02 09418 9398; email: dartconv@mpx.com.au).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics, and Gynaecology, Caring for Sexuality in Health and Illness (for healthcare professionals and nurses), jointly with Association of Psychosexual Nursing 27 June 2000

Further details: Symposium Office, Imperial College School of Medicine, Queen Charlotte's and Chelsea Hospital, Goldhawk Road, London W6 0XG (tel: 020 8383 3904; fax: 020 8383 8555; email: sympreg@ic.ac.uk).

Sexual Health and HIV Conference: Facing the Millennium, Portsmouth Marriott Hotel, Portsmouth, 28 June 2000

Further details: Rebecca Mitchell (tel: 023 9286 6796; fax: 023 9286 6769).

6th ESC Congress on Contraception in the Third Millennium: a (R)Evolution in Reproductive and Sexual Health, Ljubljana, Slovenia, 28 June–1 July 2000

Further details: Orga-Med Congress Office, Mr Peter Erard, Essenestraat 77, B-1740 Ternat, Belgium (tel: +32 2 582 08 52; fax: +32 2 582 55 15; email: orgamed@village.uunet.be).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics, and Gynaecology, New Horizons in Recurrent Pregnancy Loss, 29 June–1 July 2000

Further details: Symposium Office, Imperial College School of Medicine, Queen Charlotte's and Chelsea Hospital, Goldhawk Road, London W6 0XG (tel: 020 8383 3904; fax: 020 8383 8555; email: sympreg@ic.ac.uk).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics, and Gynaecology, Bereavement, 5 July 2000

Further details: Symposium Office, Imperial College School of Medicine, Queen Charlotte's and Chelsea Hospital, Goldhawk Road, London W6 0XG (tel: 020 8383 3904; fax: 020 8383 8555; email: sympreg@ic.ac.uk).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics, and Gynaecology, Advances in Obstetric Medicine: International Meeting of Obstetric Medicine Societies (satellite to ISSHP), Paris, 6–7 July 2000

Further details: Symposium Office, Imperial College School of Medicine, Queen Charlotte's and Chelsea Hospital, Goldhawk Road, London W6 0XG (tel: 020 8383 3904; fax: 020 8383 8555; email: sympreg@ic.ac.uk).

XIII International AIDS Conference, 9–14 July 2000, Durban, South Africa

Further details: Congrex Sweden AB, PO Box 5619, Linnegatan 89A, 114 86 Stockholm, Sweden (tel: +46 8 459 6600; fax: +46 8 661 91 25; email: aids2000@congrex.se).

The Management of Genito-urinary Infections in Women, Royal Society of Medicine, London, 13–14 July 2000

Ethical Issues in International Health Research, Durban, South Africa, 16–21 July 2000 (immediately following XIII International AIDS Conference)

Further details: Marie-Christine Ryckaert, Program director, Ethical Issues in International Health Research, Harvard University, John F Kennedy School of Government, Cambridge, MA 02138, USA (tel: (617) 496-0484 ex 7474; fax: (617) 495-3090; email: Marie-Christine_Ryckaert@harvard.edu).

12th Annual Meeting of the Spanish Association of Cervical Pathology and Colposcopy (AEPCC) and HPV Clinical Workshop, 21–23 July 2000 and 18th International Papillomavirus Conference, 23–28 July 2000, Palau de Congressos, Barcelona, Spain

Further details: PACIFICO, SA, E Granados, 44, 08008 Barcelona, Spain (tel: +34.93.454.54.00; fax: +34.93.451.74.38; email: gp@pacifico-meetings.com).

MSSVD Clinical Developments Fund

The MSSVD Clinical Developments Fund is asking for applications for funding to support projects that advance the understanding and practice of genitourinary medicine. An amount of £10 000 is available to one or more successful applicant(s). Closing date for application is 25 August 2000. Further details: Dr Keith Radcliffe, Honorary Assistant Secretary MSSVD, Whitall Street Clinic, Whitall Street, Birmingham B4 6DH (tel: 0121 237 5719; fax: 0121 237 5729; email: keith.radcliffe@bscht.wmids.nhs.uk).

3rd Congress of the Baltic Association of Dermatovenereology, 7–9 September 2000, Riga, Latvia

Further details: Professor Andris Y Rubins, Department of Dermatovenereology, Medical Academy of Latvia, K Valdemara Street, 76–75, Riga, LV-1013, Latvia (tel: +(371) 7370395; fax: +(371) 7361615; email: arubins@apollo.lv).

National NCCG Update Meeting, Bromsgrove Stakis Hotel, 23–24 September 2000

Further details: Kathy Taylor (tel: 01384 235207; email: palmtraining@tesco.net).

11th Regional Meeting of International Union against Sexually Transmitted Infections, South East Asian and Western Pacific Branch and 24th National Conference of Indian Association for the Study of Sexually Transmitted Diseases and AIDS, 13–15 October 2000, Chandigarh, India

Further details: Dr Bhushan Kumar, Organising Secretary, 11th Regional Meeting of IUSTI-Asia Pacific (SE Asia and W Pacific Branch), Department of Dermatology, Venereology and Leprosy, PGIMER, Chandigarh - 160 012, India (tel: +91 (0172) 745330; fax: +91 (0172) 744401/745078; email: kumarbhushan@hotmail.com).

Consortium of Thai Training Institutes for STDs and AIDS—10th STDs/AIDS diploma course, Bangkok Hospital, Bangkok (30 Oct–12 Nov) and Prince of Songkla University, Hat Yai, Thailand (13–23 Nov) 30 October–23 November 2000

Further details: Hat Yai Secretariat, Dr Verapol Chandeyng, Dept of OB-GYN, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkla 90110, Thailand (fax: (66-74) 446 361; email: cverapol@ratree.psu.ac.th or Bangkok Secretariat, Dr Thanit Palanuvej, Bangkok Hospital, 189 Sathorn Road, Bangkok 10120, Thailand (fax: (66-2) 286 3013; email: pthanit@email.ksc.net).

Consortium of Thai Training Institutes for STDs and AIDS—International Reunion and Refresher Course on Sexual Health, Lee Garden Plaza Hotel, Hat Yai, Thailand 24–26 November 2000

Further details: Hat Yai Secretariat, Dr Verapol Chandeyng, Dept of OB-GYN, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkla 90110, Thailand (fax: (66-74) 446 361; email: cverapol@ratree.psu.ac.th or Bangkok Secretariat, Dr Thanit Palanuvej, Bangkok Hospital, 189 Sathorn Road, Bangkok 10120, Thailand (fax: (66-2) 286 3013; email: pthanit@email.ksc.net).

CORRECTION

An error occurred in the February issue of *STI*. The author of the editorial "The COPE Report 1999" (2000;76:68) is Alexander (not Alexandra) McMillan.

CURRENT PUBLICATIONS

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

Gonorrhoea
Chlamydia
Candidiasis
Bacterial vaginosis
Trichomoniasis
Pelvic inflammatory disease
Syphilis and other treponematoses
Hepatitis
Herpes
Human papillomavirus infection
Cervical cytology and colposcopy
Other sexually transmitted infections
Public health and social aspects
Microbiology and immunology
Dermatology
Miscellaneous

Gonorrhoea

Susceptibility to gonococcal infection during the menstrual cycle.

S NOWICKI, A HARTVANTASSELL, B NOWICKI. *JAMA* 2000;283:1291

'Broken windows' and the risk of gonorrhoea.

D CIHEN, S SPEAR, R SCRIBNER *et al.* *Am J Pub Health* 2000;90:230-6

High HIV seroprevalence associated with gonorrhoea: New York City Department of Health, sexually transmitted disease clinics, 1990-1997.

LV TORIAN, HA MAKKI, IB MENZIES *et al.* *AIDS* 2000;14:189-96

Rise in gonorrhoea in London, UK.

IMC MARTIN, CA ISON. *Lancet* 2000;355:623

Urine screening for gonococcal and chlamydial infections at community-based organizations in a high-morbidity area.

CA JONES, RC KNAUP, M HAYES, BP STONER. *Sex Transm Dis* 2000;27:146-51

Evaluation of four commercial transport media for the survival of *Neisseria gonorrhoeae*.

JC ARBIQUE, KR FORWARD, J LEBLANC. *Diag Microbiol Infect Dis* 2000;36:163-8

Antimicrobial resistance of *Neisseria gonorrhoeae* and high prevalence of ciprofloxacin-resistant isolates in Japan, 1993 to 1998.

M TANAKA, H NAKAYAMA, M HARAOKA *et al.* *J Clin Microbiol* 2000;38:521-5

Prevalence and tetM subtype of tetracycline-resistant *Neisseria gonorrhoeae* in Ohio, 1994.

DL TREES, Y FAKILE, SW NEAL, JS KNAPP. *Sex Transm Dis* 2000;27:46-8

Resistance of *Neisseria gonorrhoeae* epidemic strains to antibiotics—report of resistant isolates and surveillance in Zhanjiang, China: 1998 to 1999.

GM LI, Q CHEN, SC WANG. *Sex Transm Dis* 2000;27:115-8

Effects of the immunoglobulin A1 protease on *Neisseria gonorrhoeae* trafficking across polarized T84 epithelial monolayers.

S HOPPER, B VASQUEZ, A MERZ *et al.* *Infect Immun* 2000;68:906-24

Charged tmRNA but not tmRNA-mediated proteolysis is essential for *Neisseria gonorrhoeae* viability.

CH HUANG, WC WOLFGANG, J WITHEY *et al.* *EMBO J* 2000;19:1098-1107

Chlamydia

Acute primary *Chlamydia trachomatis* infection in male adolescents after their first sexual contact.

I SRUGO, R GERSHTEIN, S MADJAR *et al.* *Arch Pediat Adolesc Med* 2000;154:169-72

Evaluation of patient-administered tampon specimens for *Chlamydia trachomatis* and *Neisseria gonorrhoeae*.

SN TABRIZI, CK FAIRLEY, SJ CHEN *et al.* *Sex Transm Dis* 2000;27:133-7

Evaluation of chlamydia and gonorrhoea screening criteria—San Francisco sexually transmitted disease clinic: 1997 to 1998.

EL CIEMINS, CK KENT, J FLOOD, JD KLAUSNER. *Sex Transm Dis* 2000;27:165-7

Impact of switching laboratory tests on reported trends in *Chlamydia trachomatis* infections.

LW DICKER, DJ MOSURE, WC LEVINE, CM BLACK, SM BERMAN. *Am J Epidemiol* 2000;151:430-5

Detection of *Chlamydia trachomatis* in pregnant women by the Papanicolaou technique, enzyme immunoassay and polymerase chain reaction.

CAB PANUCO, ID RODRIGUEZ, JTH MENDEZ *et al.* *Acta Cytol* 2000;44:114-23

Multicenter evaluation of the AMPLICOR and automated COBAS AMPLICOR CT/NG tests for detection of *Chlamydia trachomatis*.

B VANDERPOL, TC QUINN, CA GAYDOS *et al.* *J Clin Microbiol* 2000;38:1105-12

Chlamydial development is adversely affected by minor changes in amino acid supply, blood plasma amino acid levels and glucose deprivation.

A HARPER, CI POGSON, ML JONES, JH PEARCE. *Infect Immun* 2000;68:1457-54

Differential regulation of CD4 lymphocyte recruitment between the upper and lower regions of the genital tract during *Chlamydia trachomatis* infection.

KA KELLY, JC WALKER, SH JAMEEL *et al.* *Infect Immun* 2000;68:1519-28

T-cell epitopes in variable segments of *Chlamydia trachomatis* major outer membrane protein elicit serovar-specific immune responses in infected humans.

L ORTIZ, M ANGEVINE, SK KIM *et al.* *Infect Immun* 2000;68:1719-23

Candidiasis

Vaginal colonization by *Candida* in asymptomatic women with and without a history of recurrent vulvovaginal candidiasis.

P GIRALDO, A VONNOWASKONSKI, FAM GOMES *et al.* *Obstet Gynecol* 2000;95:413-6

Effects of reproductive hormones on experimental vaginal candidiasis.

OL FIDEL, J CUTRIGHT, C STEELE. *Infect Immun* 2000;68:651-63

Evaluation of the Oricult-N dipslide for laboratory diagnosis of vaginal candidiasis.

P CARLSON, M RICHATDSON, J PAAVONEN. *J Clin Microbiol* 2000;38:1063-76

Clonal and spontaneous origins of fluconazole resistance in *Candida albicans*.

JP XU, AR RAMOS, R VILGALYS, TG MITCHELL. *J Clin Microbiol* 2000;38:1214-20

Mechanisms of the proinflammatory response of endothelial cells to *Candida albicans* infection.

AS OROZCO, X ZHOU, SG FILLER. *Infect Immun* 2000;68:1134-49

Bacterial vaginosis

Bacterial vaginosis.

B NIEVES. *Anaerobe* 1999;5:343-6

Metronidazole to prevent preterm delivery in pregnant women with asymptomatic bacterial vaginosis.

JC CAREY, MA KLEBANOFF, JC HAUTH *et al.* *N Engl J Med* 2000;342:534-40

Pre-term labor associated with bacterial vaginosis.

H CALDERAS, B NIEVES, A QUINTANA. *Anaerobe* 1999;5:403-4

Trichomoniasis

Resistance of *Trichomonas vaginalis* to metronidazole: report of the first three cases from Finland and optimization of in vitro susceptibility testing under various oxygen concentrations.

T MERI, TS JOKIRANTA, L SUHONEN, S MERI. *J Clin Microbiol* 2000;**38**:763–7

Antigenicity of *Trichomonas vaginalis* heat-shock proteins in human infections.

SR DAVIS-HAYMAN, PH SHAH, RW FINLEY *et al.* *Parasitol Res* 2000;**86**:115–20

Pelvic inflammatory disease

Pelvic inflammatory disease—an evidence-based approach to diagnosis.

PR MUNDAY. *J Infect* 2000;**40**:31–41

Influence of human immunodeficiency virus infection on pelvic inflammatory disease.

KL IRWIN, AC MOORMAN, MJ OSULLIVAN *et al.* *Obstet Gynecol* 2000;**95**:525–34

Direct medical cost of pelvic inflammatory disease and its sequelae: decreasing but still substantial.

DB REIN, WJ KASSLER, KL IRWIN, L RABIEE. *Obstet Gynecol* 2000;**95**:397–402

Syphilis and other treponematoses

Unraveling the Tuskegee Study for untreated syphilis.

RM WHITE. *Arch Intern Med* 2000;**160**:585–601

Nodular tertiary syphilis mimicking granuloma annulare.

SJ WU, EQ NGUYEN, TA NIELSON, AE PELLEGRINI. *J Am Acad Dermatol* 2000;**42**:378–80

Social network method for endemic foci of syphilis: a pilot project.

R ROTHENBERG, L KINBROUGH, R LEWISHARDY *et al.* *Sex Transm Dis* 2000;**27**:12–8

Geographic variation of HIV infection in childbearing women with syphilis in the United States.

EH KOUMANS, M STERNBERG, M GWINN *et al.* *AIDS* 2000;**14**:279–88

HIV prevalence in patients with syphilis, United States.

ME BLOCKER, WC LEVINE, ME STLOUIS. *Sex Transm Dis* 2000;**27**:53–9

From the CDC—syphilis elimination: history in the making—opening remarks.

J KOPLAN. *Sex Transm Dis* 2000;**27**:63–5

From the CDC—syphilis elimination: history in the making—closing remarks.

D SATCHER. *Sex Transm Dis* 2000;**27**:68–73

Primary and secondary syphilis in the metropolitan area of Nashville and Davidson County, Tennessee—1996 to 1998 epidemic described.

JS HUANG, WB ROGERS, SBC BAILEY. *Sex Transm Dis* 2000;**27**:168–74

Virulent *Treponema pallidum* lipoprotein and synthetic lipopeptides induce CCR5 on human monocytes and enhance their susceptibility to infection by human immunodeficiency virus type 1.

TJ SELLATI, DA WILKINSON, JS SHEFFIELD *et al.* *J Infect Dis* 2000;**181**:283–92

Hepatitis

International congress on viral hepatitis A and B: experience in education and prevention.

Vaccine 2000;**18**:Suppl 1 (whole issue)

The seroprevalence of hepatitis A and B in people testing positive for hepatitis C.

LA KIEFER, A HONISH, G PREDY, JA TALBOT. *Can Med Assoc J* 2000;**162**:207–8

‘Silent killer’ or benign disease? The dilemma of hepatitis C virus outcomes.

KR HIRSCH, TL WRIGHT. *Hepatology* 2000;**31**:536–7

Hepatitis C epidemiology: injecting new tools in the field.

DL THOMAS. *Hepatology* 2000;**31**:790–806

45-Year follow-up of hepatitis C virus infection in healthy young adults.

LB SEEFF, RN MILLER, CS RABKIN *et al.* *Ann Intern Med* 2000;**132**:105–11

Prevalence of hepatitis G virus in patients with hemophilia and their steady female sexual partners.

AET YEO, A MATSUMOTO, JW SHIH *et al.* *Sex Transm Dis* 2000;**27**:178–82

Are booster immunizations needed for lifelong hepatitis B immunity?

J BANATVALA, M KANE, G DAVILLA *et al.* *Lancet* 2000;**355**:561–5

Cellular and humoral immune responses induced by intradermal or intramuscular vaccination with the major hepatitis B surface antigen.

F RAHMAN, A DAHMEN, S HERZOGHAUFF *et al.* *Hepatology* 2000;**31**:521–7

Herpes

Herpes simplex type 2 infection in the developing world: is it time to address this disease?

L COREY. *Sex Transm Dis* 2000;**27**:30–1

Genital herpes and public health: addressing a global problem.

L COREY, HH HANDSFIELD. *JAMA* 2000;**283**:791–4

Reactivation of genital herpes simplex virus type 2 infection in asymptomatic seropositive persons.

A WALD, J ZEH, S SELKE *et al.* *N Engl J Med* 2000;**342**:844–50

Herpes simplex virus type 2 shedding in human immunodeficiency virus-negative men who have sex with men: frequency, patterns and risk factors.

MR KRONE, A WALD, SR TABET *et al.* *Clin Infect Dis* 2000;**30**:261–7

Editorial response: Asymptomatic herpes simplex virus shedding and Russian roulette.

LA STANBERRY. *Clin Infect Dis* 2000;**30**:268–9

Human immunodeficiency virus infection and genital ulcer disease in South Africa: the herpetic connection.

CY CHEN, RC BALLARD, CM BECKSAGUE *et al.* *Sex Transm Dis* 2000;**27**:21–9

Medical care expenditures for genital herpes in the United States.

GY TAO, WJ KASSLER, DB REIN. *Sex Transm Dis* 2000;**27**:32–8

Herpes simplex virus DNA in amniotic fluid without neonatal infection.

A ALANEN, V HUKKANEN. *Clin Infect Dis* 2000;**30**:363–7

Herpes simplex virus infection of the uterine cervix—relationship with a cervical factor?

W EGGERTKRUSE, B MILDENBERGERSANDBRINK, P SCHNITZLER *et al.* *Fert Steril* 2000;**73**:248–57

The herpesvirus proteases as targets for antiviral chemotherapy.

L WAXMAN, PL DARKE. *Antivir Chem Chemother* 2000;**11**:1–22

Monoclonal antibodies suitable for type-specific identification of herpes simplex viruses by a rapid culture assay.

M BYSTRICKA, M ZATOVICOVA, M PETRIKOVA *et al.* *Acta Virol* 1999;**43**:399–402

Establishment of latent herpes simplex virus type 1 infection in resistant, sensitive and immunodeficient mouse strains.

AR ELLISON, L YANG, C VOYTEK, TP MARGOLIS. *Virology* 2000;**268**:17–28

Herpes simplex virus infection blocks events in the G1 phase of the cell cycle.

B SONG, JJ LIU, KC YEH, DM KNIPE. *Virology* 2000;**267**:326–34

A role for MHC class 1 down-regulation in NK cell lysis virus-infected cells.

B HUARD, K FRUH. *Eur J Immunol* 2000; **30**:509–15

Virus-induced neuronal apoptosis blocked by the herpes simplex virus latency-associated transcript.

GC PERNG, C JONES, J CIACCIZANELLA *et al. Science* 2000; **287**:1500–2

Herpes simplex virus type-1 and -2 pathogenesis is restricted by the epidermal basement membrane.

BS WEEKS, RS RAMCHANDRAN, JJ HOPKINS, HM FRIEDMAN. *Arch Virol* 2000; **145**:385–96

Mitochondrial distribution and function in herpes simplex virus-infected cells.

T MURATA, F GOSHIMA, T DAIKOKU *et al. J Gen Virol* 2000; **81**:401–6

Antegrade transport of herpes simplex virus type 1 in cultured, dissociated human and rat dorsal root ganglion neurons.

M MIRANDASAKSENA, P ARMATI, RA BOADLE *et al. J Virol* 2000; **74**:1827–39

The latency-associated transcript gene enhances establishment of herpes simplex virus type 1 latency in rabbits.

GC PERNG, SM SLANINA, A YUKHT *et al. J Virol* 2000; **74**:1885–99

Limited antibody-dependent cellular cytotoxicity antibody response induced by a herpes simplex virus type 2 subunit vaccine.

S JOHL, ED CHARLEBOIS, M SIGOURAUDINIA *et al. J Infect Dis* 2000; **181**:335–9

Effect of route of vaccination with vaccinia virus expressing HSV-2 glycoprotein D on protection from genital HSV-2 infection.

DI BERNSTEIN. *Vaccine* 2000; **18**:1351–8

DNA immunization utilizing a herpes simplex virus type 2 myogenic DNA vaccine protects mice from mortality and prevents genital herpes.

JR GEBHARD, JL ZHU, X CAO *et al. Vaccine* 2000; **18**:1837–46

Evidence for a bidirectional element located downstream from the herpes simplex virus type 1 latency-associated promoter that increases its activity during latency.

H BERTHOMME, J LOKENSGARD, L YANG *et al. J Virol* 2000; **74**:3613–22

Human papillomavirus infection

Smoking, diet, pregnancy and oral contraceptive use as risk factors for cervical intra-epithelial neoplasia in relation to human papillomavirus infection.

L KJELLBERG, G HALLMANS, AM AHREN *et al. Br J Cancer* 2000; **82**:1332–8

Gynecological infections as risk determinants of subsequent cervical neoplasia.

M VIKKI, E PUKKALA, P NIEMINEN, M HAKAMA. *Acta Oncol* 2000; **39**:71–6

Papillomavirus detection: demographic and behavioral characteristics influencing the identification of cervical disease.

E ADAM, Z BERKOVA, Z DAXNEROVA *et al. Am J Obstet Gynecol* 2000; **182**:257–64

Evaluation of a human papillomavirus assay in cervical screening in Zimbabwe.

SD WOMACK, ZM CHIRENJE, PD BLUMENTHAL *et al. Br J Obstet Gynaecol* 2000; **107**:33–8

Determinants of low-risk and high-risk cervical human papillomavirus infections in Montreal university students.

H RICHARDSON, E FRANCO, J PINTOS *et al. Sex Transm Dis* 2000; **27**:79–86

Population-based study of human papillomavirus infection and cervical neoplasia in rural Costa Rica.

R HERRERO, A HILDESHEIM, C BRATTI *et al. J Nat Cancer Inst* 2000; **92**:464–74

Epidemiological aspects of human papillomavirus infection and cervical cancer in Brazil.

SMB CAVALCANTI, LG ZARDO, MRL PASSOS, LHS OLIVEIRA. *J Infect* 2000; **40**:80–7

Human papillomavirus-associated carcinomas in Hawaii and the mainland US.

M FRISCH, MT GOODMAN. *Cancer* 2000; **88**:1464–9

Prevalence of and risks for cervical human papillomavirus infection and squamous intraepithelial lesions in adolescent girls: impact of infection with human immunodeficiency virus.

AB MOSCICKI, JH ELLENBERG, SH VERMUND *et al. Arch Pediat Adolesc Med* 2000; **154**:127–34

A novel and rapid PCR-based method for genotyping human papillomaviruses in clinical samples.

JH NELSON, GA HAWKINS, K EDLUND *et al. J Clin Microbiol* 2000; **38**:688–95

Seroresponses to human papillomavirus types 16, 18, 31, 33 and 45 virus-like particles in South African women with cervical cancer and cervical intraepithelial neoplasia.

DJ MARAIS, RC ROSE, C LANE *et al. J Med Virol* 2000; **60**:403–10

Seroresponses to virus-like particles of human papillomavirus types 16, 18, 31, 33 and 45 in San people of southern Africa.

D MARAIS, RC ROSE, C LANE *et al. J Med Virol* 2000; **60**:331–6

Type specificity and significance of different isotypes of serum antibodies to human papillomavirus capsids.

ZH WANG, L KJELLBERG, H ABDALLA *et al. J Infect Dis* 2000; **181**:456–62

Specific serum IgG, IgM and IgA antibodies to human papillomavirus types 6,11,16,18 and 31 virus-like particles in human immunodeficiency virus-seropositive women.

A PETTER, K HEIM, M GUGUER *et al. J Gen Virol* 2000; **81**:701–8

HPV16 E6 oncogene variants in women with cervical intraepithelial neoplasia.

J LUXTON, C MANT, B GREENWOOD *et al. J Med Virol* 2000; **60**:337–41

Human papillomavirus types 16 E6 and E7 contribute differently to carcinogenesis.

S SONG, A LIEM, JA MILLER, PF LAMBERT. *Virology* 2000; **267**:141–50

The effects of interferon on the expression of human papillomavirus oncogenes.

KY KIM, L BLATT, MW TAYLOR. *J Gen Virol* 2000; **81**:695–700

Human papillomaviruses and DNA ploidy in anal condylomata acuminata.

S RIHET, P BELLAICH, M LOWENZATO *et al. Histopathol* 2000; **15**:79–84

HPV11 mutant virus-like particles elicit immune responses that neutralize virus and delineate a novel neutralizing domain.

SW LUDMERER, WL MCCLEMENTS, XM WANG *et al. Virology* 2000; **266**:237–56

The p53 Arg72Pro polymorphism, human papillomavirus and invasive squamous cell cervical cancer.

MM MADELEINE, K SHERA, SM SCHWARTZ *et al. Cancer Epidem Biomarker Prev* 2000; **9**:225–8

Telomerase, p53 and human papillomavirus infection in the uterine cervix.

P NAIR, PG JAYAPRAKASH, MK NAIR, MR PILLAI. *Acta Oncol* 2000; **39**:65–70

Analysis of human papillomavirus type 16 E6 variants in relation to p53 codon 72 polymorphism genotypes in cervical carcinogenesis.

M VANDUIN, PJF SNIJDERS, MTM VOSSEN *et al. J Gen Virol* 2000; **81**:317–26

The human papillomavirus type 16 E5 protein modulates ERK1/2 and p38 MAP kinase activation by an EGFR-independent process in stressed human keratinocytes.

K CRUSIUS, I RODRIGUEZ, A ALONSO. *Virus Genes* 2000; **20**:65–70

Nuclear matrix attachment regions of human papillomavirus type 16 repress or activate the E6 promoter, depending on the physical state of the viral DNA.

W STUNKEL, ZH HUANG, SH TAN, MJ OCONNOR, HU BERNARD. *J Virol* 2000;74:2489–2509

Repression of the integrated papillomavirus E6/E7 promoter is required for growth suppression of cervical cancer cells.

DA FRANCIS, SI SCHMID, PM HOWLEY. *J Virol* 2000;74:2679–93

Recombinant adeno-associated virus expressing human papillomavirus type 16 E7 peptide DNA fused with heat shock protein DNA as a potential vaccine for cervical cancer.

DW LIU, YP TSAO, JT KUNG *et al.* *J Virol* 2000;74:2888–99

Adeno-associated virus major Rep78 protein disrupts binding of TATA-binding protein to the P97 promoter of human papillomavirus type 16.

PF SU, SY CHIANG, CW WU, FYH WU. *J Virol* 2000;74:2459–76

Correlation of TGβ1 overexpression with down-regulation of proliferation-inducing molecules in HPV-11 transformed human tissue xenografts.

MK SHIER, EB NEELY, MG WARD *et al.* *Anticancer Res* 1999;19:4969–76

Human papillomavirus E7 proteins stimulate proliferation independently of their ability to associate with retinoblastoma protein.

S CALDEIRA, EM DEVILLIERS, M TOMMASINO. *Oncogene* 2000;19:821–6

Mechanisms of human papillomavirus E2-mediated repression of viral oncogene expression and cervical cancer cell growth inhibition.

A NISHIMURA, R ONO, A ISHIMOTO *et al.* *J Virol* 2000;74:3752–60

The hinge of the human papillomavirus type 11 E2 protein contains major determinants for nuclear localization and nuclear matrix association.

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