LETTERS TO THE EDITOR

Carbamazepine in Reiter’s syndrome

EDITOR,—A psoriatic spectrum with Reiter’s syndrome in an HIV infected patient. This is the first report of carbamazepine associated with Reiter’s syndrome in an HIV infected patient. The therapeutic response seen in our patient conforms to that described in the HIV-1 positive patient of Smith et al.1

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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Carbamazepine 200 mg daily in two divided doses was continued in the same dose for 6 months. New lesions stopped appearing. Paedoneisolone was then tapered off rapidly and anogenital lesions were controlled. Carbamazepine was continued in the same dose for 6 months. On follow up at 1 year, the patient showed no recurrence of 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.2 Stimulated mast cells secrete a number of proinflammatory cytokines and proteases that act similarly.3,4 Carbamazepine significantly inhibits the uptake of Na+, Ca2+ ions and blocks a cyclic AMP mediated calcium influx that is associated with neuropeptide release and control of a slow potassium current.5

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 reinstitution of carbamazepine respectively proves its efficacy in our patient. Also, the clinical remission maintained for 1 year after stopping carbamazepine therapy was developed as an HIV risk measure. The therapeutic efficacy of carbamazepine in Reiter’s syndrome is well described in the literature.7 The therapeutic response seen in our patient conforms to that seen in the HIV-1 positive patient of Smith et al.1

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.8

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Accepted for publication 20 March 2000

Condoms and warts

EDITOR,—Wen et al1 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 clarification, 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.

Overall, the reference grid 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 these 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

Figure 1 Close view of erythematous annular papules and plaques on chest before carbamazepine therapy.
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) in June 1998 with 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 in the sun exposed areas (arms, back of neck, face, and ears). The rash was signifi-
cantly worse over his elbows where there was obvious blistering and oedema. His medi-
cation was stopped and 3 weeks later the rash had completely resolved. Hepatitis C anti-
body 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 × 10^3 cells/l, he was commenced on dual antiretroviral therapy with stavudine and lamivudine (450 mg bid) and his CD 4 count was 512 × 10^3 cells/l. However, 9 months following this combination his viral load began to rebound (3192 copies/ml) and a change in antiretroviral therapy was initiated to abacavir and nevirapine which he reported in the normal way (dose escalation at 2 weeks of nevirapine). He was started on this combi-
nation 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 constitu-
tional symptoms it was decided to stop this present combination. One month later, the rash had settled, he then commenced combi-
vir 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 2 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 infection; however, screening for both these conditions was nega-
tive. 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.1 Photosensitivity in the context of HIV infection may not only be a presenting condition but also secondary to concomitant treatment.

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1 Pappert A, Grossman M, DeLeo V. Photosensi-
tivity as the presenting sign in HIV infected patients with human immunodeficiency viral infection. Arch Dermatol 1994;130:618–23.
4 O’Connor WJ, Murphy BM, Darby C, et al. Porphyria abnormalities in acquired immuno-
5 DePaul Pharmaceuticals Company Research Laboratories. Wilmington, DE. In-house data 1980s.
Accepted for publication 20 March 2000

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 hos-
pitals had the same protocol for the treatment of CMV retinitis and employed 3 monthly ophthalmological screening of all HIV in-
fected patients with CD4 counts of less than 50 ×10^3.

The study outcomes were visual loss and death. Moderate visual loss was defined as 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 1–426) 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 1.4–2.2). 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 associ-
ated 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 opportu-
infectious infections. Prolonged survival times with CMV retinitis have been demonstrated in patients who achieve immunological recov-
er with HAART.3 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 HAART 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 pro-
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Letters, Book reviews, Notices, Correction, Current publications

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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 urethritis is becoming more widespread as patient acceptability and improved compliance outweigh cost considerations. However, in men, treatment is often initiated on the basis of negative 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), 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.1

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.

NSU was defined as the presence of at least five polymorphonuclear leucocytes (PMNL) in five or more fields on microscopy of a urethral smear, not in patients on whom no nongonococcal urethritis syndrome in men.2 J Am Med Assoc 1995;274:545–9.

Treatment failure was defined as persisting or confirmed symptoms or clearing of previously positive symptoms or of finding of urethritis before the chlamydial result is available. 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.3

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 chlamydial 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.6

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Table 1 Diagnoses of older and younger clinic attendees

<table>
<thead>
<tr>
<th>STIs</th>
<th>Older clinic</th>
<th>Younger clinic</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Genital herpes</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Genital warts</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Gonorrhoea</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Trichomonas vaginalis</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Other conditions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erectile dysfunction</td>
<td>15</td>
<td>1</td>
</tr>
<tr>
<td>Balanitis</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>Lichen sclerosis</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Zoster balanitis</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Genital priapism</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Genital eczematous sebaceous glands</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Genital skin tag</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Inguinal hernia</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Genital sebaceous cyst</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Miscellaneous (hepatitis B vaccination)</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Many elderly people maintain heterosexual and homosexual activity. Therefore this age group is at a risk of all sexually transmitted infections.7 In our study, a smaller percentage of older attendees had STIs compared with previous studies.7 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 50.8 This finding was seen in our study as well.

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Accepted for publication 30 April 2000

Editorial

EDITOR,—I read Dr Reed’s letter on tertiary syphilis9 with interest.

The regimen he describes for the treatment of early syphilis—arsenic, bismuth, and rhus—has been used in the clock aqueous penicillin, was used in our hospital from 1946–8 although daily penicillin in beeswax was also used. It was unclear how much inactive penicillin K was in the commercial product used. The penicillin was used here was higher than in Lincoln (40 000–75 000 units 3–4 hourly).

There were 10 treatment failures (reinfections) out of 275 patients described.9 Treponema pallidum remains viable in the CSF even after adequate clinical treatment:10

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

<table>
<thead>
<tr>
<th>1997</th>
<th>1998</th>
<th>oyoxytetracyline</th>
<th>azithromycin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number treated</td>
<td>76</td>
<td>52</td>
<td></td>
</tr>
<tr>
<td>Median age (range)</td>
<td>28 (16–83)</td>
<td>25 (16–54)</td>
<td></td>
</tr>
<tr>
<td>No with symptoms (%)</td>
<td>35 (46)</td>
<td>25 (48)</td>
<td></td>
</tr>
<tr>
<td>No cured (%)</td>
<td>29 (38)</td>
<td>27 (52)</td>
<td></td>
</tr>
<tr>
<td>No treatment failures (%)</td>
<td>6 (8)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Outcome uncertain*</td>
<td>41 (54)</td>
<td>25 (48)</td>
<td></td>
</tr>
<tr>
<td>Symptomatic DNA</td>
<td>8/35 (23)</td>
<td>4/25 (16)</td>
<td></td>
</tr>
<tr>
<td>Asymptomatic DNA</td>
<td>13/41 (32)</td>
<td>7/27 (26)</td>
<td></td>
</tr>
</tbody>
</table>

*Originally asymptomatic with clear two glass urine, did not reattend (dna), possibly reinfected.
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 treponemal antibodies for other intercurrent infections, so that any neurosyphilis that developed would either be modified with few physical signs or would be completely treated and clinically cured. However, others disagree with this. Dr Reed's question, we haven't seen anyone treated since the second world war who has developed neurosyphilis in subsequent years.

DAVID GOLDMEIER

1 Reed TAG. Tertiary syphilis. Sex Transm Inf 1999;75:75.

BOOK REVIEWS


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 anchors and could be a short textbook in itself.

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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: Ora-Med Congress Office, Mr Peter Erazd, Essenestraat 77, B-1740 Ter nat, 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 Recurr
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).

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 (AEPPCC) and HPV Clinical Workshop, 21–23 July 2000 and 18th International Papillomavirus Conference, 23–28 July 2000, Palau de Congressos, Barcelona, Spain
Further details: PACIFICOF, 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 Dermatovenerology, 7–9 September 2000, Riga, Latvia
Further details: Professor Andris Y Rubins, Department of Dermatovenerology, 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) 745530; fax: +91 (0172) 744001/745078; 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 Chandeying, Dept of OB-GYN, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkla 90110, Thailand (fax: (66-74) 446 361; email: cverapol@zatreep.psu.ac.th or Bangkok Secretariat, Dr Thanit Palanuvej, Bangkok Hospital, 189 Sathorn Road, Bangkok 10120, Thailand (fax: (66-2) 286 3013; email: ptbanit@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 Chandeying, Dept of OB-GYN, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkla 90110, Thailand (fax: (66-74) 446 361; email: cverapol@zatreep.psu.ac.th or Bangkok Secretariat, Dr Thanit Palanuvej, Bangkok Hospital, 189 Sathorn Road, Bangkok 10120, Thailand (fax: (66-2) 286 3013; email: ptbanit@email.ksc.net).

CORRECTION

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
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Candidiasis
Gonorrhoea

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or to Neisseria gonorrhoeae trafficking

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Impact of switching laboratory tests on

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infections.

Detection of Chlamydia trachomatis in

pregnant women by the Papanicolaou

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Mechanisms of the proinflammatory

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Candida albicans infection.

Bacterial vaginosis.

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and lower regions of the genital tract during Chlamydia trachomatis infec-

tion.

T-cell epitopes in variable segments of Chlamydia trachomatis major outer

membrane protein elicit serovar-
specific immune responses in infected

humans.

Vaginal colonization by Candida in

asymptomatic women with and without a

history of recurrent vulvovaginal candidiasis.

Effects of reproductive hormones on

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Metronidazole to prevent preterm deliv-

ery in pregnant women with asympto-
matic bacterial vaginosis.

Pre-term labor associated with bacterial vaginosis.

Gonorrhoea

Susceptibility to gonococcal infection
during the menstrual cycle.

‘Broken windows’ and the risk of gonorrhea.


Rise in gonorrhoea in London, UK.

Urine screening for gonococcal and

chlamydial infections at community-
based organizations in a high-morbidity

treatment area.

Evaluation of four commercial transport

media for the survival of Neisseria gonorrhoeae.


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

Resistance of Neisseria gonorrhoeae
epidermic strains to antibiotics—report

or resistant isolates and surveillance in


Effects of the immunoglobulin A1 pro-
tease on Neisseria gonorrhoeae trafficking

across polarized T84 epithelial monolayers.

Charged tmRNA but not tmRNA-

mediated proteolysis is essential for

Neisseria gonorrhoeae viability.

Chlamydia

Acute primary Chlamydia trachomatis

infection in male adolescents after their

first sexual contact.

Evaluation of patient-administered tam-

pon specimens for Chlamydia trachoma-

sis and Neisseria gonorrhoeae.

Evaluation of chlamydia and gonorrhoea

screening criteria—San Francisco sexu-

ally transmitted disease clinic: 1997 to

1998.

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Detection of Chlamydia trachomatis in

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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.

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

Pelvic inflammatory disease

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

Influence of human immunodeficiency virus infection on pelvic inflammatory disease.

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

Syphilis and other treponematoses

Unraveling the Tuskegee Study for untreated syphilis.

Nodular tertiary syphilis mimicking granuloma annulare.

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

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

HIV prevalence in patients with syphilis, United States.

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

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

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

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

Hepatitis

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

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

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

Hepatitis C epidemiology: injecting new tools in the field.

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

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

Are booster immunizations needed for lifelong hepatitis B immunity?

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Genital herpes and public health: addressing a global problem.

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Medical care expenditures for genital herpes in the United States.

Herpes simplex virus DNA in amniotic fluid without neonatal infection.

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The herpesvirus provestes as targets for antiviral chemotherapy.

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Microbiology and immunology

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