LETTERS TO THE EDITOR

Prevalence of HPV cervical infections among imprisoned women in Barcelona, Spain

EDITOR,—The penitentiary centres in Spain harbour inmates in whom the combination of HIV infection, history of injecting drug use, and prostitution is common.[1] Extensive protocols to detect sexually transmitted diseases and tuberculosis are implemented in these centres; however, human papillomavirus (HPV) infections and related lesions are not routinely searched for. Although Spain is characterised by a very low incidence of cervical cancer,[2] a high rate of cervical cancer has been reported recently among the AIDS female population in Catalonia.[3] We carried out a study aiming to characterise HPV cervical infection and related cervical lesions among women with many potential risk factors for cervical neoplasia. The study was done in the only institution in Barcelona where women are imprisoned. The population consisted of 157 women attending the medical office of the prison between February and December 1996 and represented 90% of all women staying in prison for more than 3 days. Women who agreed to participate underwent a gynaecological examination, collection of cervical cells, a structured interview by a trained nurse, determination of HIV, hepatitis B and C serostatus, and detection of HPV DNA in the cervical cells by means of PCR. L1 consensus primers MY09/MY11 were used with modifications as described by Hidelsheim *et al*.[4]

HPV DNA was detected in 48% of the women. The prevalence of cervical abnormalities was 29.9%; 19 women had a atypical squamous cells of undetermined significance (ASCUS) and 28 women were diagnosed with squamous intraepithelial lesion (SIL), five of whom had a high grade lesion. All women with a SIL and 42% of those with a ASCUS were HPV positive. Prostitution was reported by 38.2% and injecting drug use by 64.3% women. HIV infection was detected in 56.1%. HPV detection was significantly related to HIV, to injecting drug use, to prostitution practices, and to hepatitis C positive serology. After adjusting for these variables, HPV detection remained significantly associated with HIV and with length of time injecting drugs (table 1). No association between HPV detection was found with other reproductive and sexual characteristics. In addition, HIV positive women had an increased risk to develop SIL compared with HIV negative women (POR=5.02, 95% CI=1.69–14.89). As previously reported, the risk for SIL increased with low CD4 T cell counts, although POR did not reach statistical significance.[5]

Data from an ongoing study in a nearby area indicate that the prevalence of cervical abnormalities in the general population is around 4% (manuscript in preparation). This is the first time that we have documented in Spain a group of women with a very high rate of HPV infection linked to injecting drug use and with a rate of pre-neoplastic cervical lesions about seven times higher than that observed in the general population.

While in prison these women were appropriately treated for HIV infection and for SIL. When out of prison or in jail, a gynaecological screening every 6–12 months should be organised and recommended.

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Table 1 Age adjusted prevalence odds ratios for human papillomavirus infection (HPV DNA) in the cervical cells by different characteristics

	HPV DNA Negative		*HPV DNA positive*				
	No	*%*	*No*	*%*	*PORc*	*PORa*	*95% CI*
HIV							
Negative	54	63.5	15	20.8	1	1	
Positive	31	36.5	57	79.2	7.3	4.7	1.96–11.4
Prostitution							
No	59	69.4	38	52.8	1	1	
Yes	26	30.6	34	47.2	2.0	1.2	0.5–2.5
Injecting drug use							
No	44	51.8	12	16.7	1	1	
Yes	41	48.2	60	83.3	5.4	2.4	0.98–6.1
Length of use:							
0–9 years	22	26.5	26	36.6	4.2	2.2	0.8–6.0
⩾10 years	17	20.5	33	46.5	7.0	2.9	1.0–8.2
Hepatitis C							
Negative	49	59.8	21	31.3	1	1	
Positive	33	40.2	46	68.7	3.1	1.2	0.5–2.9

PORc = age adjusted.
PORa = adjusted for age and the other variables in the table.

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Detection of penicillinase producing *Neisseria gonorrhoeae* strains in Cuba, 1995–8

EDITOR,— Since the 1940s, penicillin has been recommended for the treatment of gonorrhoea. In the 1950s the first strains of *Neisseria gonorrhoeae* with reduced susceptibility to this antibiotic, as a result of chromosomal mutations, were isolated, and in 1976 the first penicillinase producing *Neisseria gonorrhoeae* (PPNG) strains emerged in South East Asia and Africa, causing high level resistance to penicillin (MIC 16 µg/ml).[1] In Cuba, the first report of a PPNG strain was made in 1986 (C Almanza, personal communication). We report here on the proportion of PPNG strains received at the Neisseria Reference Laboratory, Tropical Medicine Institute "Pedro Kouri" (IPK), Cuba between January 1995 and December 1998.

In all, 110 strains of *N gonorrhoeae* isolated from 10 of the 14 Cuban provinces were examined for their β lactamase activity by the chromogenic method (Nitrocefin, Oxoid). These strains were transported to the IPK using a novel transport and conservation medium for gonococci developed at our laboratory.[2] *N gonorrhoeae* WHO E and WHO A were used as positive and negative control strains, respectively. All strains were identified as gonococci by standard procedures.[3]

Table 1 shows the distribution of Cuban PPNG and non-PPNG strains detected in our laboratory during 1995–8. The PPNG strains predominated totally (61/110, 55.5%). The percentage of PPNG strains was high in all years analysed.[4] To our knowledge it is the first study developed in Cuba, analysing the β lactamase activity of *N gonorrhoeae* isolated from different provinces in which a high percentage of PPNG strains was found. Previous studies developed in specific Cuban hospitals in Havana City have revealed a lower percentage of PPNG strains (M Berroa *et al*, 1988; C Almanza *et al*, 1988, personal communications).

Penicillin has been the drug of choice for treatment of gonococcal infections in Cuba since 1972.[5] The results of this study indicate that any policy to treat such infections should not include penicillin or other similar drugs. Other antimicrobials recommended by the World Health Organisation for treatment gonorrhoea—for example, spectinomycin, cephalosporins, quinolones, and azithromycin

Table 1 Distribution of Cuban PPNG and non-PPNG strains from 1995 to 1998

Year	No of gonococci examined	PPNG strains		Non-PPNG strains	
		No	%	No	%
1995	63	33	52.4	30	47.6
1996	21	14	66.6	7	33.4
1997	21	13	61.9	8	38.1
1998	5	1	20	4	80
Total	110	61	55.5	49	44.5

PPNG = penicillinase producing *N gonorrhoeae*.

have been recently evaluated in Cuba with good results (R Llanes, *et al*, unpublished data, 1999).

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Rising HIV prevalence in STD clinic attenders at Chandigarh (north India)—a relatively low prevalence area

EDITOR,—The patients attending the STD clinics are at risk of having concurrent HIV infection. The trends of HIV infection in these patients may reflect the trends of HIV epidemic in the community. We have analysed the HIV status of 981 patients (824 males, 157 females) who attended our STD clinic from January 1993 to July 1999 (about 6½ years). The screening for HIV was done by ELISA. Those who were found positive were tested by repeat ELISA utilising another blood sample and considered HIV seropositive only, if both samples were found positive. The STDs were diagnosed by appropriate laboratory tests. The majority of the attenders had STDs; however, a small but significant proportion of patients had psychosexual disorders and other non-sexually transmitted genital diseases. Four per cent of the 981 patients—that is, 40 patients (26 males, 14 females) were found to be seropositive for HIV. The annual prevalence showed a rising trend (1993, 0.56%; 1994, 4.4%; 1995, 2.4%; 1996, 4%; 1997, 4.4%; 1998, 5.7%; and January to July 1999, 8.7%). The prevalence of HIV seropositivity in different STDs is shown in table 1. Large proportions of seropositive patients were truckers (15/40, 37.5%) and housewives (12/40, 30%). Among 12 housewives, four were wives of truckers. All of the 26 seropositive male patients confessed to at least one sexual contact with commercial sex workers (CSWs). Twenty eight (70%) seropositive patients had one STD, while the remaining 12 (30%) patients had more than one STD; 18 (45%) seropositive patients had STDs with either atypical morphologies or unusual severity, the remaining 22 (55%) presented with usual morphologies.

India is a country with a wide variation in geographical, cultural, and behavioural patterns. This is also reflected in the trends of current HIV epidemic in the various regions of the country. We believe that no other country has such a high intranation variation in HIV epidemic status. Comparison of our data on HIV prevalence with STD clinics of different regions of the country highlights this difference. The high HIV prevalence zones of the country include western and southern zones, where HIV prevalence among STD clinic attenders varies from 15% to 33%.[1–3] On the other hand, in eastern and northern zones, it is still low and varies from 0.2 to 4%.[1 3–5]

In our study we found that a high proportion of HIV positive patients were truckers, who generally acquired infection from CSWs from the highways to Bombay or Chennai, two metropolitan cities of the western and southern zones respectively. These long distance truckers have a high risk sexual behaviour and contribute in the spread of HIV infection throughout the country in a short time.[2 6]

Even though the present figures for HIV seropositivity in STD clinic attenders are not very high, the HIV epidemic in this region is now progressing at an alarming rate. In our study, the prevalence in our STD clinic increased from 0.56% in 1993 to 8.7% in 1999 (to July). This indicates that northern India is entering from a low level epidemic (HIV prevalence less than 5% in STD patients) to a concentrated epidemic.[1] This calls for an immediate vigorous intervention programme to be introduced in this region.

Table 1 Frequency of HIV seropositivity in different sexually transmitted diseases

STDs	No screened	HIV seropositive	Seropositivity rate (%)
Ulcerative STDs			
Genital herpes	188	19	10.1
Syphilis	107	6	5.6
Chancroid	21	1	4.76
Donovanosis	5	0	0
Lymphogranuloma venereum	5	0	0
All ulcerative STDs	322	25	7.6
Non-ulcerative STDs			
Condyloma accuminata	184	13	7
Balanoposthitis	75	2	2.66
Gonorrhea	35	1	2.85
Molluscum contageosum	27	3	11.1
Non-gonococcal urethritis	27	0	0
Vaginosis	23	1	4.3
All non-ulcerative STDs	368	18	4.9
All STD clinic attendees*	981	40	4

*The discrepancy in total is due to the presence of more than one STD in some patients.

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HIV seropositivity in women with syphilis in Delhi, India

EDITOR,—There has been a progressive rise in the prevalence of human immunodeficiency virus (HIV) infection in India, which currently has the largest number of HIV infected people in the world.[1] The spread of HIV is predominantly by heterosexual transmission in India.[2] Sexually transmitted disease (STD), particularly genital ulcer disease (herpes, syphilis, and chancroid), has an important role in the transmission of HIV, and the two have been observed to be interrelated.[3 4] We conducted a pilot study to assess the relation between syphilis and HIV infection among non-pregnant women attending gynaecology and STD clinics of our hospital.

From June 1998 to July 1999, sera from 281 non-pregnant women were tested for syphilis by VDRL (Serologist, India) and confirmed by TPHA (Immunotrep, Omega Diagnostic Ltd, UK). Sera that tested positive for syphilis were tested for HIV without identifying the patient. Individual informed consent for HIV was not obtained as results were not aimed to be linked to the identity of those tested. Serum was tested first with one ELISA/rapid/simple (ERS) assay, utilising either of the these different enzyme linked immunosorbent assay (UBI, HIV-1/2, United Medical Inc, USA, Recombigens HIV-1/HIV-2, EIA, Cambridge Biotech Galway, Ireland, and HIV spot Genelabs Diagnostic, Singapore). Any reactive sample was retested using a different assay. Samples that were reactive in all the three tests were considered HIV antibody positive. A sample that was non-reactive on the first test was considered HIV negative, as was a sample that was reactive in the first and non-reactive in the next test.[5]

Of 281 sera tested, 48 (17%) were seropositive for syphilis. HIV antibody was detected in sera of six (12.5%) patients who were seropositive for syphilis (table 1). None of the 233 patients with negative syphilis serology tested

Table 1 Details of patients undergoing serological test for syphilis

Clinical diagnosis	*No of samples (%)*	*Positive for syphilis serology*	*Positive for HIV*
Previous pregnancy loss*	89/281 (31.6)	16/89 (17.9%)	0/16 (0%)
Vaginal discharge	101/281 (55.8)	9/101 (8.9%)	1/9 (11.1%)
Genital growth	49/281 (17.4)	6/49 (12.2%)	1/6 (16.6%)
Genital ulcer	42/281 (14.9)	17/42 (40.47%)	4/17 (23.5%)

*Intrauterine death, still birth, repeated abortions.

positive for HIV antibody. This was highly significant ($p<0.001$, Fisher's exact test). Presence of HIV antibody was associated with genital ulcer in 23.5% women, followed by genital growth and vaginal discharge in 16.6% and 11.1% respectively.

There is a higher prevalence of STD and HIV infection among men compared with women. HIV seropositivity has been associated with a reactive serological test for syphilis among males. This could be probably due to higher percentage of male attendance in STD clinics.[6] We therefore undertook this study to evaluate if some association exists between syphilis and HIV among non-pregnant women attending the gynaecology clinic, as well as the STD clinic. Untreated STDs, especially those with ulcerative disease, can enhance both susceptibility of a person to HIV infection as well as infectivity of HIV positive individual. Breach in the epithelial surface of a genital ulcer may be an important factor in the transmissibility of HIV. This is evident from our results where incidence of positive serology for HIV was highest among women with genital ulcer (23.5%). Our study demonstrates a significant association between positive serology for syphilis and presence of HIV infection. We feel that the diagnosis of syphilis in non-pregnant women may act as a marker to detect the presence of HIV infection.

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Immune reconstitution CMV pneumonitis

EDITOR,—A 41 year old white homosexual man presented in late July 1999 with a 5 day history of exertional dyspnoea, non-productive cough, fever with sweats, and anorexia. An empirical course of broad spectrum antibiotics did not improve his symptoms and Sao_2 remained ≤95% on air at rest. The chest radiograph showed non-specific abnormalities. He had been found to be HIV-1 antibody positive in August 1991; cutaneous Kaposi's sarcoma defined AIDS in June 1992. In May 1995 biopsy confirmed cytomegalovirus (CMV) oesophagitis and colitis were treated with intravenous ganciclovir for 2 weeks; no maintenance therapy was given. At this time the CD4 count was 130 cells $\times 10^6$/l. In October 1996 the patient had *Pseudomonas aeruginosa* pneumonia. He had a complex antiretroviral history, having taken combinations of reverse transcriptase inhibitors and protease inhibitors. He had discontinued all antiretroviral therapy in January 1999 as therapy had failed to maintain CD4 counts and HIV viral load had risen: co-trimoxazole primary *Pneumocystis carinii* pneumonia prophylaxis had been continued. In early June 1999 HIV viral load had risen to 223 000 copies/ml and CD4 count had fallen to 70 cells $\times 10^6$/l. Two weeks before the onset of respiratory symptoms the patient had recommenced antiretroviral therapy with d4T, 3TC, and amprenivir/saquinavir. Four weeks after starting antiretroviral therapy viral load had fallen to 1500 copies/ml and CD4 had risen to 170 cells $\times 10^6$/µl. A computed tomography (CT) scan of the thorax 4 weeks after the onset of respiratory symptoms and 6 weeks after starting antiretroviral therapy showed focal areas of ground glass shadowing, largely in the left upper lobe but also involving other lobes; in addition, chronic changes resulting from the previous episode of pneumonia were noted, including multifocal fibrotic change with thickened interlobular septae, cystic air spaces, and minor bronchiectasis involving all lobes. Repeat viral load at this time = 200 copies/ml and CD4 = 160 cells $\times 10^6$/l. At bronchoscopy, performed after 8 weeks of antiretroviral therapy, the endobronchial appearances were normal. Bronchoalveolar lavage (BAL) was performed from the left upper lobe. Analysis of BAL fluid revealed a lymphocytic reaction; many cells had intranuclear/cytoplasmic inclusions typical of CMV infection. In situ hybridisation for CMV was positive. Staining and culture for bacteria, mycobacteria, *P carinii* and other fungi were negative. Intravenous ganciclovir 10 mg/kg per day was given for 21 days, in addition, antiretroviral therapy and co-trimoxazole were continued. With this there was a rapid defervescence of fever, a reduction in exertional dyspnoea and improvement in Sao_2 to ≥98% on air. Repeat CT of the thorax after 3 weeks of intravenous ganciclovir showed an improvement in ground glass shadowing and persistence of the chronic changes. The patient was subsequently maintained on oral ganciclovir.

The diagnosis of CMV pneumonitis was made by identifying CMV as the sole pathogen in BAL fluid and the improvement in symptoms, Sao_2, and CT appearances with ganciclovir as monotherapy. This diagnosis was made in the context of a rapidly falling viral load and an increase in CD4 count indicating partial immune reconstitution.

Partial restoration of cell mediated immunity induced by antiretroviral therapy, as shown by recovery of part of CD4 T cell reactivity to memory antigens,[1 2] may cause development of sufficient inflammatory responses to produce symptoms and signs in patients latently infected with opportunistic infections. Reactivation mycobacterial lymphadenitis,[3] cryptococcal meningitis,[4] and CMV retinitis[5 6] have been described. The case described here suggests CMV pneumonitis should be added to the list of immune reconstitution phenomena.

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BOOK REVIEWS

Common Gynaecological Problems. Ed by Patricia Wilson. Pp 312; Price £24.95. Oxford: Blackwell Science, 1999. ISBN 0-632-05174-4.

A book with a title such as this one makes it difficult for the author to decide what to exclude. This book certainly fulfils its major objective of providing an easy reference manual for the diagnosis and management of common gynaecological conditions. It deals with almost all the gynaecological conditions that could be encountered in the community and the common gynaecological problems in hospital medicine. Overall, the topics covered are well presented with special points highlighted.

The use of pictures relating to almost all the conditions dealt with by the book breaks up what would otherwise be a book of lists. The use of two different views of the same woman exercising on a treadmill certainly made me smile. The first picture tells us she is an intensively training sportswoman who may develop amenorrhoea and osteoporosis with stress fractures while the second picture, on a page dealing with advice to women who do not want HRT, reveals she is a grandmother taking regular exercise.

From a genitourinary medicine trainee point of view, I would have liked to see a more comprehensive chapter on pelvic infections and sexually transmitted diseases (this is the second smallest chapter in the book), and would have preferred this chapter to follow the one on vaginal and vulval problems. I am, however, glad to see that the role of the genitourinary clinic in the management of pelvic infections is emphasised.

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Sex, Disease and Society. A comparative history of sexually transmitted diseases and HIV/AIDS in Asia and the Pacific. Ed by Milton Lewis, Scott Bamber and Michael Waugh. Pp 296; £55.95. London: Greenwood Press, 1997. ISBN 0-313-29442-9.

Histories of Sexually Transmitted Diseases and HIV/AIDS in Sub-Saharan Africa. Ed by Philip W Setel, Milton Lewis and Maryinez Lyons. Pp 267; £59.95. London: Greenwood Publishing Group, 1999. ISBN 0-313-29715-0.

These two books provide histories of STDs and HIV in nine sub-Saharan African countries and another 11 countries in the Asia-Pacific region. The contributors are mostly historians or social scientists and the historical accounts take the reader up to 1995. Each volume is divided up into well referenced scholarly monographs on individual countries and individual chapters will be of considerable interest to anyone with an interest in sexual health in the countries studied. The number of readers of this journal who will want to read both books throughout is likely to be much less, given that these books are fairly specialist medical historical studies written mainly by historians for historians. The decision of the editors to treat each country separately has led inevitably to much repetition of certain themes. Many chapters rehearse the familiar story of how governments have responded to public pressure to regulate prostitution and the difficulty of demonstrating whether such efforts have had any real impact on STD transmission. The most interesting example in this context is the account of the attempts to eradicate prostitution and STDs in China, a subject where it is peculiarly difficult to separate the facts from the propaganda. Not only were STDs allegedly expunged from the population but they were deleted from medical textbooks too! Another theme to which contributors constantly return is the problem of differentiating non-venereal from venereal treponematosis. We are constantly reminded that syphilis reporting may be distorted by this issue but other pertinent issues such the unitarian theory of treponematosis, the lack of specificity of older serological test methods, the impossibility of determining the mode of transmission from serological results or, in many instances, from observed clinical manifestations, receive rather patchy and inconsistent coverage. A third recurring theme is the unreliability of passive reporting systems. While this is often acknowledged, contributors still feel obliged to cite whatever data they can unearth and to discuss observed trends that are unlikely to bear much relation to any true epidemiological situation.

What is there in these books for the clinician or epidemiologist with an interest in STDs? There is no shortage of entertaining anecdote such as the expatriate doctor in Uganda who had himself publicly injected with mercury to demonstrate his faith in this treatment. The account of regular penicillin injections for prostitutes in Indonesia will interest those who are following studies of targeted periodic presumptive treatment in Africa such as the Lesedi Project. Having worked in Papua New Guinea, I was interested to see what was written about spectacular epidemic of donovanosis that affected the Marind-anim tribe in the 1920s. I felt that the account given failed to bring alive the unique nature of this epidemic and the campaign to control it. The main problem for more clinically oriented readers is the wealth of innovative approaches to STD and HIV control that have been explored in these countries since 1995 and which are too recent for inclusion in these volumes. The accounts of HIV go little further than the difficulties experienced in galvanising governments out of denial and into action. For detailed accounts of the Mwanza and Rakai trials and their impact on policy and for the discussion of more topical controversies such as the possible role of polio vaccine development in the Congo in triggering the HIV pandemic we will have to look to future historians.

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Handbook of Genitourinary Medicine. Ed by S Barton, P Hay. Pp 496; £45. London: Edward Arnold, 1999. ISBN 0-340-740841.

This book is a terrific read and should be read cover to cover by all practising genitourinary medicine physicians and trainees. Generally the quality of the writing is excellent. Genitourinary medicine is a rapidly advancing field so read the book now before it becomes out of date. Already the incubation period of the text shows in places. Some statistics relate to 1992 where 1997 figures are available. Some statements are also slightly out of date.

In a book of this size the referencing presents a challenge. If one references every statement (and considers all the conflicting evidence) the handbook turns into a weighty and unmanageable tome. Mostly, the authors have managed a sensible compromise. Statements that are uncontroversial or old hat are not referenced. Occasionally more controversial statements remain unreferenced. This may present a problem for the trainee. There are also some surprising omissions. I could find no descriptions of desquamative vaginitis or focal vulvitis. However, I believe that this handbook could serve as an excellent basis for discussions between trainer and trainee and stimulate further reading around these topics.

Get this book. You will enjoy it. A number of chapters are absolute gems.

CHRIS CARNE
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NOTICES

1st Annual Teesside Sexual Health Conference, 11 March 2000
Further details: Mandy Bruce (tel: 01642 854809).

9th International Congress on Infectious Diseases, 9–12 April 2000, Buenos Aires, Argentina
Further details: International Society for Infectious Diseases, 181 Longwood Avenue, Boston, MA 02115, USA (tel: (617) 277-0551; fax: (617) 731-1541; email: isidbos@aol.com).

Sexually Transmitted Diseases in a Changing Europe, 14–15 April 2000, Rotterdam, The Netherlands
Further details: Mediscon, Organisation for Medical Congresses, PO Box 113, 5660 AC Geldrop, Netherlands (tel: +31-(0)40-2852212; fax: +31-(0)40-2851966; email: MEDISCON@IAEhv.nl).

20th Scientific Conference of Venereological Section of the Polish Society of Dermatologists, Bialystok, 28–30 April 2000
The conference will be on epidemiological and clinical aspects of sexually transmitted infections. Further details: Dept Dermatology and Venereology, Sw Rocha 3, 15-879 Bialystok, Poland (tel/fax: (085) 7422778; email: bozchod@amb.ac.bialystok.pl).

Joint meeting of the MSSVD and the ASTDA, 3–7 May 2000, Baltimore Marriott Inner Harbor Hotel, Baltimore, Maryland, USA
Further details: Dr Keith Radcliffe, honorary assistant secretary, MSSVD (fax: +44(0) 121-237 5729; email: k.w.radcliffe@bham.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).

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).

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).

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@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 Chandeying, 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).

CURRENT PUBLICATIONS

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

Gonorrhoea
Chlamydia
Candidiasis
Bacterial vaginosis
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

Predicting *Neisseria gonorrhoeae* and *Chlamydia trachomatis* infection using risk scores, physical examination, microscopy and leukocyte esterase urine dipsticks among asymptomatic women attending a family planning clinic in Kenya.
MW TYNDALL, N KIDULA, J SANDE *et al. Sex Transm Dis* 1999;**26**:476–82

Increase in oral sex and pharyngeal gonorrhoea: an unintended effect of a successful condom promotion programme for vaginal sex.
ML WONG, RKW CHAN, D KOH, S WEE. *AIDS* 1999;**13**:1981

Cervical wet mount as a negative predictor for gonococci- and *Chlamydia trachomatis*-induced cervicitis in a gravid population.
JT BOHMER, G SCHEMMER, FNH HARRISON *et al. Am J Obstet Gynecol* 1999;**181**:283–5

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S NOWICKI, R SELVARANGAN, G ANDERSON. *Infect Immun* 1999;**67**:4974–6

Comparison of direct inoculation and copan transport systems for isolation of *Neisseria gonorrhoeae* from endocervical specimens.
CC OLSEN, JR SCHWEBKE, WH BENHAMIN *et al. J Clin Microbiol* 1999;**37**:3583–9

T lymphocyte response to *Neisseria gonorrhoeae* porin in individuals with mucosal gonococcal infections.
SD SIMPSON, Y HO, PA RICE, LM WETZLER. *J Infect Dis* 1999;**180**:762–73 38

Decreased azithromycin susceptibility of *Neisseria gonorrhoeae* due to mtrR mutations.
L ZARANTONELLI, G BORTHAGARAY, EH LEE, WM SHAFER. *Antimicrob Agents Chemother* 1999;**43**:2468–78 44

The farAB-encoded efflux pump mediates resistance of gonococci to long-chained antibacterial fatty acids.
EH LEE, WM SHAFER. *Mol Microbiol* 1999;**33**:839–45 40

Chlamydia

Partner notification for chlamydial infections among private sector clinicians in Seattle-King County: a clinician and patient survey.
MR GOLDEN, WLH WHITTINGOTN, PM GORBACH *et al. Sex Transm Dis* 1999;**26**:543–7

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LH BACHMANN, CM RICHEY, K WAITES *et al. Sex Transm Dis* 1999;**26**:496–9

Completeness of and duration of time before treatment after screening women for *Chlamydia trachomatis* infections.
G FOGLIA, P RHODES, M GOLDBERG, ME STLOUIS. *Sex Transm Dis* 1999;**26**:421–5

Control of *Chlamydia trachomatis* infections in female army recruits: cost-effectiveneness screening and treatment in training cohorts to prevent pelvic inflammatory disease.
MR HOWELL, JC GAYDOS, KT MCKEE *et al. Sex Transm Dis* 1999;**26**:519–26

Lack of association between serum antibodies to *Chlamydia trachomatis* and a history of recurrent pregnancy loss.
M PAUKKU, M TULPPALA, M PUOLAKKAINEN *et al. Fert Steril* 1999;**72**:427–30

How adequate is adequate for the collection of endocervical specimens for *Chlamydia trachomatis* testing?
JL BEEBE, KA GERSHMAN, JK KELLEY *et al. Sex Transm Dis* 1999;**26**:579–83

The impact on accuracy and cost of ligase chain reaction testing by pooling urine specimens for the diagnosis of *Chlamydia trachomatis* infections.
J KREPEL, J PATEL, A SPROSTON *et al. Sex Transm Dis* 1999;**26**:504–7

Ability of the Digene Hybrid Capture II test to identify *Chlamydia trachomatis* and *Neisseria gonorrhoeae* in cervical specimens.
J SCHACHTER, EW HOOK WM MCCORMACK *et al. J Clin Microbiol* 1999;**37**:3668–75

Impact of reference standard sensitivity on accuracy of rapid antigen detection assays and a leukocyte esterase dipstick for diagnosis of *Chlamydia trachomatis* infection in first-void urine specimens from men.
M CHERNESLU, D JANG, J KREPEL. *J Clin Microbiol* 1999;**37**:2777–80

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KB WAITES, KR SMITH, MA CRUM *et al. J Clin Microbiol* 1999;**37**:3072–5

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Lower prevalence of *Chlamydia pneumoniae* DNA compared with *Chlamydia trachomatis* DNA in synovial tissue of arthritis patients.
HR SCHUMACHER, HC GERARD, RK ARAYSSI *et al. Arthritis Rheum* 1999;**42**:1889–93

Lack of cell wall peptidoglycan versus penicillin sensitivity: new insights into the chlamydial anomaly.
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The effect of doxycycline treatment and the development of protective immunity in a murine model of chlamydial genital infection.
H SU, R MORRISON, R MESSER, W WHITMIRE *et al. J Infect Dis* 1999;**180**:1252–8

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The use of fluconazole and itraconazole in the treatment of *Candida albicans* infections: a review.
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Local production of chemokines during experimental vaginal candidiasis.
M SAAVEDRA, B TAYLOR, N LUKACS, PL FIDEL. *Infect Immun* 1999;**67**:5820–33

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P MARICHAL, J GORRENS, L LAURIJSSENS *et al. Antimicrob Agents Chemother* 1999;**43**:2663–70

Control of filament formation in *Candida albicans* by polyamine levels.
AB HERRERO, MC LOPEZ, S GARCIA *et al. Infect Immun* 1999;**67**:4870–8

Bacterial vaginosis

Is the lack of concurrence of bacterial vaginosis and vaginal candidosis explained by the presence of bacterial amines?
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Association of indicators of bacterial vaginosis with a female geintal tract factor that induces expression of HIV-1.
GG OLINGER, FB HASHEMI, BE SHA, GT SPEAR. *AIDS* 1999;**13**:1905–12

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Incident syphilis among women with multiple admissions to jail in New York City.
S BLANK, M STERNBERG, LL NEYLANS *et al. J Infect Dis* 1999;**180**:1159–63

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The use of Western blotting as the confirmatory test for syphilis in patients with rheumatic disease.
FT MURPHY, R GEORGE, K KUBOTA *et al. J Rheumatol* 1999;**26**:2448–53

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DR BLANCO, CI CHAMPION, MA LEWINSKI *et al. J Immunol* 1999;**163**:2741–6

Hepatitis

Cost-effectiveness analysis of hepatitis A vaccination strategies for adults.
JB OCONNOR, TF IMPERIALE, ME SINGER. *Hepatology* 1999;**30**:1077–81

The Denver school-based adolescent hepatitis B vaccination program: a cost analysis with risk simulation.
RR DEUSON, EJ GOEKTRA, R SEDJO *et al. Am J Public Health* 1999;**89**:1722–7

Pathogenesis of chronic hepatitis C: immunological features of hepatic injury and viral persistence.
A CERNY, FV CHISARI. *Hepatology* 1999;**30**:595–601

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A prospective study of new infections with herpes simplex virus type 1 and type 2.
AGM LANGENBERG, L COREY, RL ASHLEY *et al. N Engl J Med* 1999;**341**:1432–8

Is sexual transmission an important pattern for herpes simplex type 2 virus seroconversion in the Spanish general population?
P GARCIACORBEIRA, R DALRE, L AGUILAR *et al. J Med Virol* 1999;**59**:194–7

Quality of life and use of health care among people with genital herpes in France.
R TABOULET, B HALIOUA, JE MALKIN. *Acta Derm Venereol* 1999;**79**:380–4

The differential impact of training stress and final examination stress on herpesvirus latency at the United States Military Academy of West Point.
R GLASER, SB FRIEDMAN, J SMYTH *et al. Brain Behav Immun* 1999;**13**:240–51

College students' attitudes regarding vaccination to prevent genital herpes.
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Ecthyma secondary to herpes simplex virus infection.
A KINYAIASADI, FA TAUSK, HC NOUSARI. *Clin Infect Dis* 1999;**29**:454

Acquired lymphedema of the hand due to herpes simplex virus type 2.
DF BUTLER, PJ AMLOUF, RC BATZ, CL STETSON. *Arch Dermatol* 1999;**135**:1125

Whole cell lysate enzyme immunoassays vs recombinant glycoprotein G2-based immunoassays for HSV-2 seroprevalence studies.
P GARCIACORBEIRA, W HOGREFE, L AGUILAR *et al. J Med Virol* 1999;**59**:502–6

A double-blind, randomized study assessing the equivalence of valacyclovir 1000 mg once daily versus 500 mg twice daily in the episodic treatment of recurrent genital herpes.
P SAIAG, D PRAINDHUI, C CHASTANG. *J Antimicrob Chemother* 1999;**44**:525–32

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A ALVAREZMCLEOD, J HAVLIK, KE DREW. *Clin Infect Dis* 1999;**29**:937

The comparative effects of famciclovir and valacyclovir on herpes simplex virus type 1 infection, latency and reactivation in mice.
RA LEBLANC, L PESNICAK, M GODLESKI, SE STRAUS. *J Infect Dis* 1999;**180**:594–9

Antiviral properties of isoborneol, a potent inhibitor of herpes simplex virus type 1.
M ARNAKA, E PAPANIDOLAOU, A SIVROPOULOU, M ARSENAKIS. *Antivir Res* 1999;**43**:79–92

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N BOURNE, DI BERNSTEIN, LR STANBERRY. *Antimicrob Agents Chemother* 1999;**43**:2685–8

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LAT expression during an acute HSV infection in the mouse.
RG JARMAN, EK WAGNER, DC BLOOM. *Virology* 1999;**262**:384–97

Inhibition of dendritic cell maturation by herpes simplex virus.
M SALLO, M CELLA, M SUTER, A LANZAVECCHIA. *Eur J Immunol* 1999;**29**:3245–53

Human papillomavirus infection

Assessing gains in diagnostic utility when human papillomavirus testing is used as an adjunct to Papanicolaou smear in the triage of women with cervical cytologic abnormalities.
EL FRANCO, A FERENCZY. *Am J Obstet Gynecol* 1999;**181**:382–6

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Do HPV-negative cervical carcinomas exist?—revisited.
CS HERRINGTON. *J Pathol* 1999;**189**:1–3

Human papillomavirus is a necessary cause of invasive cervical cancer worldwide.
JMM WALBOOMERS, MV JACOBS, MM MANOS *et al. J Pathol* 1999;**189**:12–9

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M JIMENEZPEREZ, DB THOMAS. *Int J Cancer* 1999;**82**:804–9

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A FERRERA, JP VELEMA, M FIGUEROA *et al. Int J Cancer* 1999;**82**:799–803

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Intralesional or topical cidofovir for the treatment of recurrent genital warts in HIV-1-infected patients.
G ORLANDO, MM FASOLO, R BERETTA *et al. AIDS* 1999;**13**:1978–80

Histologic and immunologic associations of an HPV16 variant in low-grade smears.
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ME HAGENSEE, J SLAVINSKY, CM GAFFGA *et al. Obstet Gynecol* 1999;**94**:653–8

Serum antibodies to human papillomavirus 16 proteins in women from Brazil with invasive cervical carcinoma.
YP SUN, J ELUFNETO, FX BOSCH *et al. Cancer Epidem Biomarker Prev* 1999;**8**:935–40

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MJ SCANLON, JD GORDAN, B WILLIANSON *et al. Int J Cancer* 1999;**83**:449–55

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A quantative polymerase chain reaction-enzyme immunoassay for accurate measurements of human papillomavirus type 16 DNA levels in cervical scrapings.
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SY LI, S LABRECQUE, MC GAUZZI *et al. Oncogene* 1999;**18**:5727–37

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Organised vs spontaneous pap-smear screening for cervical cancer: a case-control study.
P NIEMINEN, M KALLIO, A ANTTILA, M HAKAMA. *Int J Cancer* 1999;**83**:55–8

Effect of organised screening on cervical cancer incidence and mortality in Finland, 1963-1995: recent increase in cervical cancer incidence.
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Angiogenesis in cervical intraepithelial neoplasia and the risk of recurrence.
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Hormone receptor status in cervical intraepithelial neoplasia: correlation with the stage of disease.
EL SALAZAR, AM ROMAN, JL GOZALEZSANCHEZ. *Med Sci Res* 1999;**27**:681–4

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Serologic evidence of human herpesvirus 8 transmission by homosexual but not heterosexual sex.
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LR SANMATEO, KL TOFFER, PE ORNDORFF, TH KAWULA. *Infect Immun* 1999;**67**:4963–73

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FR ZARETZKY, TH KAWULA. *Infect Immun* 1999;**67**:5352–60

Public health and social aspects

STD testing policies and practices in US city and county jails.
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