Investigation of the increased incidence of gonorrhoea diagnosed in genitourinary medicine clinics in England, 1994–6

Gwenda Hughes, Nick Andrews, Mike Catchpole, Matthew Goldman, Dorothy Forsyth-Benson, Marion Bond, Amanda Myers

**Objectives:** To determine important risk factors associated with cases of gonorrhoea in England, and whether any particular risk groups were associated with the substantial rise in numbers of cases seen between 1994 and 1996.

**Design:** Two retrospective cross sectional surveys.

**Setting:** 70 randomly selected genitourinary medicine (GUM) clinics in England.

**Subjects:** 10% of all gonorrhoea patients attending GUM clinics in England in 1994 (847 patients) and 1996 (1146 patients).

**Main outcome measures:** For risk factors in 1996 (study 1), unadjusted rates per 100 000 population aged 14–70 and relative rates (RR) with 95% confidence intervals (CIs). For the change in risk factors between 1994 and 1996 (study 2), adjusted odds ratios (ORs) with 95% CIs, derived from logistic regression analyses of data on patients in 1996, with patients in 1994 as the comparison group.

**Results:** The incidence of gonorrhoea in 1996 was higher in homosexual males (812 per 100 000; RR=30.2, CI= 25.2 to 36.0) compared with heterosexual males (27 per 100 000); in black Caribbeans (467 per 100 000; 21.4, 17.9 to 25.5) and black Africans (235 per 100 000; 10.8, 7.5 to 15. 5) compared with white people (22 per 100 000); and in previous GUM clinic attenders (433 per 100 000; 37.93, 35.46 to 40.56) compared with those who had not attended previously (11 per 100 000). However, most patients were either white or heterosexual. Homosexual patients in 1996 were significantly more likely to have reduced sensitivity to penicillin (2.55, 1.20 to 5.41) than those in 1994. Male homo/bisexual patients in 1996 were significantly more likely to be from the north west (3.77, 1.45 to 9.80) and to have either reduced sensitivity (2.63, 1.03 to 6.73) or complete resistance (1.98, 1.03 to 3.78) to penicillin, compared with those in 1994.

**Conclusions:** Homo/bisexual men and the black Caribbean population in England experience a disproportionate burden of gonococcal infections, however, the bulk of diagnoses are in white heterosexuals. No single risk group was associated with the rise in numbers of cases between 1994 and 1996. Resistance to penicillin is widespread and has increased in homo/bisexual men, and it is possible that a rise in treatment failures has, to some extent, enhanced transmission of gonorrhoea and contributed to the rise in numbers of diagnoses in this group.

*(Sex Transm Inf 2000;76:18–24)*

Keywords: gonorrhoea; genitourinary medicine clinics; England

**Introduction**

Between 1995 and 1996, there was a 20% rise in the number of cases of gonorrhoea seen in genitourinary medicine (GUM) clinics in England.1 This followed a smaller increase between 1994 and 19951 and the trend has continued into 1997. The rise between 1995 and 1996 was the largest proportional annual increase in diagnoses since 1945 (Communicable Disease Surveillance Centre (CDSC), unpublished data). It took place in almost every health region, occurred in heterosexuals as well as in homo/bisexual men, and was greatest in men and women aged 16–19 years.1

Gonorrhoea rates are thought to be a reasonable indicator of changes in sexual behaviour.1 The dramatic and substantial decline in numbers of people attending medicine GUM with gonorrhoea during the 1980s1 may have resulted from changes in sexual behaviour in homosexual men1 5 6 as well as heterosexuals5 associated with the increased media coverage of AIDS. Gonorrhoea is the only sexually transmitted infection for which a target was set in the Health of the Nation1 as it was thought to be a convenient marker of behaviour likely to influence HIV transmission.1 Consequently, the resurgence of gonorrhoea diagnoses during the 1990s is of considerable public health concern.

In looking for possible factors behind the recent rise it is noteworthy that higher rates of gonorrhoea among black groups than among the general white population have recently been highlighted in two urban areas.5 7 However, as routine statistics from GUM clinics give limited risk factor information,11 it was not possible to determine whether black groups are at higher risk throughout England or if they contributed disproportionately to the latest increase.

This investigation had two aims. Firstly, we wished to determine important risk factors for
gonorrhea at the national level. Secondly, we examined whether the recent rise in numbers of gonorrhea diagnoses was associated with changes in risk behaviours or with any particular risk group.

Subjects and methods

STUDY DESIGN AND SAMPLE SIZE

The investigation was in two parts: (1) an analysis of risk factors for gonorrhoea patients in 1996 (study 1), and (2) a comparison of risk factors for gonorrhoea in 1996 with those in 1994 to determine whether there had been a significant change in risk factors between these years (study 2). Two cross sectional surveys of gonorrhoea patients attending GUM clinics in 1994 and 1996 were carried out. A total sample size of about 2000—that is, approximately 10% of all patients with gonorrhoea seen in GUM clinics in England in 1994 and 1996, was required in order to detect an odds ratio (OR) of 1.5 or more when 10% or more of patients in 1994 belonged to a risk group (5% significance, 80% power). A necessity to analyse by sexual orientation meant that detectable ORs were 2.4 or more for homo/bisexual men and 1.7 or more for heterosexuals.

SAMPLING STRATEGY

Clinics

Two thirds of genitourinary medicine (GUM) clinics in inner London and a third of GUM clinics in the rest of England were randomly selected from each of nine size categories, determined by the number of gonorrhoea cases selected from each of nine size categories, from 1996 were compared directly between 1994 and 1996 using logistic regression with being a case of gonorrhoea seen in GUM clinics, study cases from 1996 were compared by sex, age group, ethnic group, and regional distribution to all gonorrhoea cases seen in GUM clinics in England in 1996 (KC60 data)

Data analysis

UNLESS otherwise specified, the data and analyses presented here include adjustments for the oversampling of patients from inner London clinics. Data from inner London clinics were downweighted by 50%. As there were no more than six repeat attenders included in the data set, both within and between the 2 years, it was not deemed necessary to account for duplicates in the analyses. To determine whether the study patients were representative of all cases of gonorrhoea seen in GUM clinics, study cases from 1996 were compared by sex, age group, sexual orientation, and regional distribution to all gonorrhoea cases seen in GUM clinics in England in 1996 (KC60 data)

Study 1: Risk factors for cases in 1996

Numbers of patients from inner London clinics were halved to account for oversampling. Incidence rates per 100 000 population aged 14–70 and relative rates were calculated for selected patient characteristics in 1996 with denominators derived from 1996 mid-year population estimates and the 1991 census adjusted for undercoverage by age, sex, and ethnic group (ethnic group and area of birth estimates) and the National Study of Sexual Attitudes and Lifestyles (sexual orientation and previous GUM clinic attendance estimates). To estimate incidence rates, the number of patients in 1996 was first multiplied by 12 (as after the London clinics were downweighted by 50% one twelfth of patients had been sampled). Relative rates (RRs) in 1996 were calculated from \[ \frac{C(e)}{C(b)} \] where \( C(e) \) denotes the proportion of patients exposed, \( C(b) \) the proportion of patients with the baseline characteristic, \( P(e) \) the proportion of the population exposed, and \( P(b) \) the proportion of the population with the baseline characteristic.

Confidence intervals were calculated as for relative risks (equivalent in this case). Cases for which ethnicity was not recorded were assumed to be white and those for which country of birth was not recorded were assumed to have been born in the United Kingdom.

Study 2: Change in risk factors between 1994 and 1996

The odds ratios among those with the disease were compared directly between 1994 and 1996 using logistic regression with being a case in 1996 (as opposed to 1994) as the outcome variable. If a factor is not associated with the outcome variable then the increase in cases observed between 1994 and 1996 is similar in the groups defined by the factor. It was not possible to calculate odds ratios for many sexual behaviour and clinical characteristics for 1994 and 1996 directly, as data on the frequency of these characteristics in the general population are not available. For this method it was assumed that the proportion of the population belonging to a given risk group changed little between 1994 and 1996.

Univariable and multivariable analyses were carried out using STATA software.

Data for patients from inner London clinics were given
a sampling weighting of 6, and those from remaining clinics a weighting of 12, since the probabilities of patients from these areas being included in the sample were 1/6 and 1/12, respectively (using the [pweight=] command in stata35). All patient and infection characteristics collected on the proforma were compared between cases in 1994 and 1996 using univariable analyses. Only those factors which were significant at p<0.2 in the univariable analyses (sex, sex abroad, previous GUM clinic attendance, concurrent acute sexually transmitted infection (STI), site, sensitivity to penicillin, treatment), and those which were of particular interest (region, age, ethnic group, number of partners, previous acute STI), were included as explanatory variables in the multivariable models. Separate models were run for heterosexuals and for homo/bisexual men because the distribution of many demographic and behavioural characteristics varied considerably between these groups. In the heterosexual model, interactions between sex and each of the explanatory variables were investigated. As there were strong regional differences in the recording of ethnic group in patients’ case notes, interactions between region and the presence or absence of ethnic group data were investigated in both models.

Results

Data were collected on 847 patients in 1994 and 1146 patients in 1996, giving an unadjusted total of 1993 patients overall. Just over half of these patients were seen in inner London clinics.

The study patients in 1996 were not significantly different from all cases reported in the 1996 KC60 data set by sex ($\chi^2 = 1.2; df = 1; p = 0.27$) or age ($\chi^2 = 4.1; df=3; p = 0.25$). However, 30% of males in the study in 1996 were homosexual or bisexual, significantly more than the 21% reported nationally ($\chi^2 = 24.4; df = 1; p < 0.001$). The regional distribution also differed significantly between the two data sets (STUDY 1: RISK FACTORS FOR GONORRHOEA PATIENTS IN 1996

<table>
<thead>
<tr>
<th>Factor</th>
<th>Number of cases* (%)</th>
<th>Rate per 100 000 population†</th>
<th>Relative rate (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Region</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>North Thames</td>
<td>211 (25)</td>
<td>56</td>
<td>1.00</td>
</tr>
<tr>
<td>South Thames</td>
<td>89 (11)</td>
<td>42</td>
<td>0.76 (0.63,0.93)</td>
</tr>
<tr>
<td>North West</td>
<td>148 (17)</td>
<td>38</td>
<td>0.68 (0.55,0.84)</td>
</tr>
<tr>
<td>Northern and Yorkshire</td>
<td>37 (4)</td>
<td>10</td>
<td>0.18 (0.12,0.25)</td>
</tr>
<tr>
<td>Trent</td>
<td>108 (13)</td>
<td>35</td>
<td>0.64 (0.51,0.80)</td>
</tr>
<tr>
<td>Anglia and Oxford</td>
<td>48 (5)</td>
<td>14</td>
<td>0.26 (0.19,0.35)</td>
</tr>
<tr>
<td>South and West</td>
<td>55 (6)</td>
<td>14</td>
<td>0.25 (0.19,0.34)</td>
</tr>
<tr>
<td>West Midlands</td>
<td>49 (6)</td>
<td>15</td>
<td>0.28 (0.21,0.38)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>268 (32)</td>
<td>18</td>
<td>1.00</td>
</tr>
<tr>
<td>Male</td>
<td>579 (68)</td>
<td>39</td>
<td>2.14 (1.90,2.40)</td>
</tr>
<tr>
<td>Age group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13–15</td>
<td>11 (1)</td>
<td>12</td>
<td>0.13 (0.07,0.24)</td>
</tr>
<tr>
<td>16–19</td>
<td>159 (19)</td>
<td>83</td>
<td>1.00</td>
</tr>
<tr>
<td>20–24</td>
<td>224 (27)</td>
<td>85</td>
<td>1.02 (0.84,1.25)</td>
</tr>
<tr>
<td>25–29</td>
<td>181 (22)</td>
<td>57</td>
<td>0.68 (0.55,0.83)</td>
</tr>
<tr>
<td>30–34</td>
<td>124 (15)</td>
<td>36</td>
<td>0.44 (0.35,0.55)</td>
</tr>
<tr>
<td>35–44</td>
<td>103 (12)</td>
<td>18</td>
<td>0.21 (0.17,0.27)</td>
</tr>
<tr>
<td>45–70</td>
<td>23 (3)</td>
<td>2</td>
<td>0.03 (0.02,0.04)</td>
</tr>
<tr>
<td>Not recorded</td>
<td>24 (3)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Male sexual orientation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heterosexual</td>
<td>385 (66)</td>
<td>27</td>
<td>1.00</td>
</tr>
<tr>
<td>Homo/bisexual</td>
<td>166 (29)</td>
<td>812</td>
<td>30.15 (25.23,36.04)</td>
</tr>
<tr>
<td>Not known</td>
<td>29 (5)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Female sexual orientation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heterosexual</td>
<td>260 (97)</td>
<td>18</td>
<td>1.00</td>
</tr>
<tr>
<td>Homo/bisexual</td>
<td>2 (1)</td>
<td>23</td>
<td>1.28 (0.32,1.5)</td>
</tr>
<tr>
<td>Not recorded</td>
<td>7 (2)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Ethnic group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>362 (43)</td>
<td>222</td>
<td>1.00‡</td>
</tr>
<tr>
<td>Black Caribbean</td>
<td>155 (18)</td>
<td>467</td>
<td>21.36 (17.91,25.47)</td>
</tr>
<tr>
<td>Black African</td>
<td>31 (4)</td>
<td>35</td>
<td>10.77 (7.51,15.45)</td>
</tr>
<tr>
<td>Indian/Pakistani/Bangladesi</td>
<td>18 (2)</td>
<td>22</td>
<td>1.02 (0.64,1.63)</td>
</tr>
<tr>
<td>Other/mixed</td>
<td>32 (4)</td>
<td>72</td>
<td>3.29 (2.30,4.69)</td>
</tr>
<tr>
<td>Not recorded</td>
<td>251 (30)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Area of birth</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UK</td>
<td>614 (72)</td>
<td>215</td>
<td>1.00§</td>
</tr>
<tr>
<td>Rest of Europe</td>
<td>31 (4)</td>
<td>31</td>
<td>1.50 (1.05,2.16)</td>
</tr>
<tr>
<td>Latin America and Caribbean</td>
<td>28 (3)</td>
<td>112</td>
<td>5.43 (3.72,7.91)</td>
</tr>
<tr>
<td>Africa</td>
<td>17 (2)</td>
<td>64</td>
<td>3.08 (1.91,4.99)</td>
</tr>
<tr>
<td>Other</td>
<td>23 (3)</td>
<td>16</td>
<td>0.51 (0.11,1.77)</td>
</tr>
<tr>
<td>Not recorded</td>
<td>135 (16)</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

§Includes patients for whom country of birth was not recorded.
‡Includes patients for whom ethnic group was not recorded.
†Population sizes were estimated using data from mid-year population estimates for 1996/1 (region, sex, and age estimates); the 1991 census* adjusted for undercoverage by age, sex, and ethnic group16 (ethnic group and area of birth estimates), and the National Study of Sexual Attitudes and Lifestyles17 (sexual orientation and previous GUM clinic attendance estimates).

STUDY 1: RISK FACTORS FOR GONORRHOEA PATIENTS IN 1996

The distributions of patients in 1996 (adjusted for London oversampling) are presented by region, sex, age group, sexual orientation, ethnic group, area of birth, and previous GUM clinic attendance in table 1.

White people were the largest ethnic group nationally and in all regions except South Thames. Northern and Yorkshire was the only region with no black Caribbean patients recorded and, in the remaining regions, the proportion of patients who were black Caribbean ranged from 4% in the North West to 62% in South Thames. Overall, ethnic group was not recorded for 30% of patients although this ranged from 6% in Trent to 67% in the North West. Ethnic group was not recorded for 10% of patients in South Thames, for 13% in North Thames, and for 20% in the West Midlands. Seventy four per cent of white people and 96% of black Caribbeans were heterosexual.

Twenty nine per cent of patients in 1996 had a concurrent acute sexually transmitted infection (STI) (see box) and 22% had previously had an acute STI. Eight per cent had a penicillin resistant gonococcal strain (that is, all penicillinase producing Neisseria gonorrhoeae (PPNG) and chromosomally mediated penicillin resistant N gonorrhoeae (CMRNG)) whereas less than 1% were resistant to quinolones or to tetracycline. Penicillin resistance was more common in homo/bisexual men, with 18% of patients resistant, compared with women and heterosexual men, with 4% and 7% resistant, respectively.

Site of infection was recorded for 98% of patients in 1996. Ninety per cent of females, over 99% of heterosexual males, and 67% of homo/bisexual men had genital gonorrhoea. Rectal gonorrhoea was found in 24% of homo/bisexual men and 10% of females, but not in heterosexual males. Throat infections were...
Definition of an acute sexually transmitted infection

Infectious syphilis
Uncomplicated gonorrhoea
Complicated gonorrhoea
Chancroid/lymphogranuloma venereum/donovanosis
Uncomplicated chlamydial infection
Complicated chlamydial infection
Uncomplicated non-gonococcal/non-specific urethritis in males
Complicated non-gonococcal/non-specific infection
Herpes simplex (first attack)
Wart virus infection (first attack)
Molluscum contagiosum
Trichomoniasis
Scabies/ediculosis

found in 9% of homo/bisexual men but in less than 1% of heterosexual men and women.

Number of partners in the past 3 months, and sex abroad in the past 3 months, was recorded for 98% and 92%, respectively, of patients in 1996. Twenty nine per cent of heterosexual females, 54% of heterosexual males, and 59% of homo/bisexual males reported two or more partners during this period. Five per cent of patients reported sex abroad.

Incidence and relative rates for selected characteristics of patients in 1996 are shown in table 1. Incidence was particularly high for homo/bisexual males compared with heterosexual males, black Caribbean and black African cases relative to white cases, and for those who had previously attended a GUM clinic.

STUDY 2: CHANGE IN RISK FACTORS FOR GONORRHOEA BETWEEN 1994 AND 1996

Descriptive analyses

Between 1994 and 1996, diagnoses in females rose by 25% (215 to 268), in heterosexual males by 46% (263 to 385), and in homo/bisexual males by 31% (126 to 166). In females, the largest increase was in 16–19 year olds and percentage increases diminished with age (fig 1A). In heterosexual males large rises occurred across most age groups (fig 1B) while in homo/bisexual males, the largest proportional increases were in the over 35s (fig 1C).

Multivariable analyses

Adjusted risk factors for being a gonorrhoea patient in 1996 compared to being a patient in 1994 are presented in table 2.

For heterosexuals, patients in 1996 were significantly more likely to be males, to have had no or one sexual partners in the past 3 months compared with two, and to have reduced sensitivity to penicillin than cases in 1994. There were no interactions between sex and any of the explanatory variables in the model.

For homo/bisexual men, patients in 1996 were significantly more likely to be from the North West and to have either reduced sensitivity or complete resistance to penicillin than cases in 1994.

Discussion

We report on the first national investigation of risk factors associated with cases of gonorrhoea, and of possible causes of the recent rise in numbers of diagnoses, in a study which sampled 10% of all gonorrhoea cases seen in GUM clinics in England in 1994 and 1996. Overall, gonorrhoea patients selected for this study were found to be fairly representative of all gonorrhoea cases seen in GUM clinics in England. The oversampling of cases from the North West and of homo/bisexual men probably arose from the random sampling of particularly large clinics in the North West, and of clinics attended predominantly by homo/bisexual men. Consequently, the incidence and relative rate of gonorrhoea in homo/bisexual men and in the North West region in 1996 are likely to be overestimated.

STUDY 1: RISK FACTORS FOR GONORRHOEA PATIENTS IN 1996

The high rates of gonorrhoea in homo/bisexual men are of continuing concern. This study
Hughes, Andrews, Catchpole, et al

indicated that about a quarter of infections in homo/bisexual men were rectal, whereas infections of the throat only, indicative of safer homosexual behaviour in terms of HIV transmission, made up fewer than 10% of cases. Variations in policy on anatomical sites routinely screened for gonorrhoea may have led to an underestimate of the proportion of rectal and throat infections. Clearly, however, many homo/bisexual men attending GUM clinics with gonorrhoea have placed themselves at risk of HIV infection through unsafe sexual practice. In 1997, an estimated 9% of homo/bisexual men attending GUM clinics in London, and 4% attending clinics elsewhere in England and Wales, were infected with HIV.

At the national level, incidence of gonorrhoea was far higher for black Caribbeans, who constituted at least 18% of all patients, than for white people. This is in accordance with results from localised studies. None the less, the bulk of gonorrhoea patients were white heterosexuals.

Recording of ethnicity tended to be better in the Thames regions, which along with the West Midlands have much higher black ethnic minority populations. To prevent overestimation of relative rates for gonorrhoea in ethnic minorities, cases for which ethnicity and/or country of birth data were not recorded were assumed to be white and/or born in the United Kingdom. This was reasonable since areas with the most missing data have relatively small black ethnic minority populations. The likely effect of misclassification due to this assumption will have been to underestimate the size of relative rates associated with non-white ethnic groups. Variations in the methods of recording ethnicity (such as whether ethnicity is assigned by the patient or clinic reception staff) also place limitations on the robustness of ethnic groupings.

Despite these limitations, this study confirms that the black Caribbean population experiences a disproportionate burden of gonococcal infections. The reasons for the unequal distribution of gonorrhoea across ethnic groups are likely to be complex. Sexual behaviours and mixing patterns are determined by cultural background to a large extent resulting in variations in the transmission patterns of STIs by ethnic group. As people more often have sexual partners within their own ethnic group, existing high levels of infection are likely to be maintained. However, there are known racial inequalities in socioeconomic status and in the access to, and use of, healthcare services with ethnic minorities being less likely to have access to good healthcare services, made up fewer than 10% of cases. Variations in policy on anatomical sites routinely screened for gonorrhoea may have led to an underestimate of the proportion of rectal and throat infections. Clearly, however, many homo/bisexual men attending GUM clinics with gonorrhoea have placed themselves at risk of HIV infection through unsafe sexual practice. In 1997, an estimated 9% of homo/bisexual men attending GUM clinics in London, and 4% attending clinics elsewhere in England and Wales, were infected with HIV.

At the national level, incidence of gonorrhoea was far higher for black Caribbeans, who constituted at least 18% of all patients, than for white people. This is in accordance with results from localised studies. None the less, the bulk of gonorrhoea patients were white heterosexuals.

Recording of ethnicity tended to be better in the Thames regions, which along with the West Midlands have much higher black ethnic minority populations. To prevent overestimation of relative rates for gonorrhoea in ethnic minorities, cases for which ethnicity and/or country of birth data were not recorded were assumed to be white and/or born in the United Kingdom. This was reasonable since areas with the most missing data have relatively small black ethnic minority populations. The likely effect of misclassification due to this assumption will have been to underestimate the size of relative rates associated with non-white ethnic groups. Variations in the methods of recording ethnicity (such as whether ethnicity is assigned by the patient or clinic reception staff) also place limitations on the robustness of ethnic groupings.

Despite these limitations, this study confirms that the black Caribbean population experiences a disproportionate burden of gonococcal infections. The reasons for the unequal distribution of gonorrhoea across ethnic groups are likely to be complex. Sexual behaviours and mixing patterns are determined by cultural background to a large extent resulting in variations in the transmission patterns of STIs by ethnic group. As people more often have sexual partners within their own ethnic group, existing high levels of infection are likely to be maintained. However, there are known racial inequalities in socioeconomic status and in the access to, and use of, healthcare services with ethnic minorities being less likely to have access to good

### Table 2: Adjusted risk factors for being a case of gonorrhoea in 1996 compared with being a case in 1994

<table>
<thead>
<tr>
<th>Variable</th>
<th>Heterosexual men and women (n=1173)</th>
<th>Homo/bisexual men (n=388)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Odds ratio (adjusted)</td>
<td>95% CI</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>0.72</td>
<td>0.53–0.97</td>
</tr>
<tr>
<td>Region</td>
<td></td>
<td></td>
</tr>
<tr>
<td>North Thames</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>South Thames</td>
<td>0.97</td>
<td>0.67–1.39</td>
</tr>
<tr>
<td>North West</td>
<td>1.54</td>
<td>0.88–2.70</td>
</tr>
<tr>
<td>Northern and Yorkshire</td>
<td>2.03</td>
<td>0.86–4.81</td>
</tr>
<tr>
<td>Trent</td>
<td>0.82</td>
<td>0.51–1.31</td>
</tr>
<tr>
<td>Anglia and Oxford</td>
<td>0.81</td>
<td>0.46–1.44</td>
</tr>
<tr>
<td>South and West</td>
<td>0.80</td>
<td>0.44–1.47</td>
</tr>
<tr>
<td>West Midlands</td>
<td>1.21</td>
<td>0.65–2.27</td>
</tr>
<tr>
<td>Age</td>
<td>0.99</td>
<td>0.98–1.02</td>
</tr>
<tr>
<td>Ethnic group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Black Caribbean</td>
<td>0.72</td>
<td>0.59–1.33</td>
</tr>
<tr>
<td>Black African</td>
<td>0.63</td>
<td>0.35–1.13</td>
</tr>
<tr>
<td>Indian/Pakistani/Bangladeshi</td>
<td>0.76</td>
<td>0.28–1.38</td>
</tr>
<tr>
<td>Other/mixed</td>
<td>0.72</td>
<td>0.59–1.33</td>
</tr>
<tr>
<td>Partners in last 3 months</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0.70</td>
<td>0.52–0.93</td>
</tr>
<tr>
<td>3 or more</td>
<td>1.07</td>
<td>0.64–1.79</td>
</tr>
<tr>
<td>Sex abroad in past 3 months</td>
<td>0.71</td>
<td>0.39–1.28</td>
</tr>
<tr>
<td>Previous GUM clinic attendance</td>
<td>0.75</td>
<td>0.52–1.08</td>
</tr>
<tr>
<td>Concurrent acute STI</td>
<td>1.24</td>
<td>0.93–1.65</td>
</tr>
<tr>
<td>Previous acute STI</td>
<td>1.19</td>
<td>0.79–1.80</td>
</tr>
<tr>
<td>Site of infection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genital</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Rectal</td>
<td>1.74</td>
<td>0.81–3.73</td>
</tr>
<tr>
<td>Throat</td>
<td>1.78</td>
<td>0.34–9.35</td>
</tr>
<tr>
<td>Sensitivity to penicillin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensitive</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Reduced sensitivity</td>
<td>0.71</td>
<td>0.52–0.93</td>
</tr>
<tr>
<td>Resistant</td>
<td>0.76</td>
<td>0.47–1.27</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Penicillin</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Quinolone</td>
<td>0.92</td>
<td>0.66–1.30</td>
</tr>
<tr>
<td>Other drug type</td>
<td>0.71</td>
<td>0.42–1.20</td>
</tr>
</tbody>
</table>

*Not estimable owing to small numbers.
†Multiple site infections were categorised as follows: “rectal” includes concurrent genital, throat, and “other” infections, “genital” includes concurrent throat and “other” infections but not rectal infections, and “throat” includes infections of the throat only.
‡Relates solely to drugs given to treat gonococcal infection. For multiple treatments “quinolone” includes penicillin treatment and “other drug type” includes penicillin treatment and antimicrobial agents.

Within their own ethnic group, existing high transmission patterns of STIs by ethnic group. However, there are known racial inequalities in use of, healthcare services, with ethnic minorities being less likely to have access to good healthcare services.
medical care. Such inequalities could facilitate infection transmission by limiting access to treatment and the effectiveness of partner notification. None the less, certain studies have suggested that disparities in gonorrhoea prevalence by ethnic group may exist even when controlling for socioeconomic status.

The study suggests that almost one in five homo/bisexual men and about one in 16 heterosexuals presenting with gonorrhoea at GUM clinics in England in 1996 had a penicillin resistant strain. In homo/bisexual men, resistance of gonococcal strains to penicillin had also increased significantly since 1994. Unfortunately, in this study we did not collect information on type of resistance and minimum inhibitory concentration ranges. As there is likely to be considerable variability in the methodology used for measuring resistance, this somewhat limits the significance of this finding.

None the less, samples submitted to the genitourinary infection reference laboratory in Bristol indicate that chromosomally mediated resistance to penicillin has been increasing since 1994, whereas penicillinase producing (plasmid) gonococcal strains have declined since 1989. Penicillin is still the preferred treatment for gonorrhoea in many clinics in the United Kingdom and the finding from this study warrant a review of recommended treatment of gonorrhoea at the local, if not the national, level, especially in homo/bisexual men.

Almost half the cases in 1996 had previous contact with GUM clinic services and had presumably received advice on safer sexual behaviour. It is also noteworthy that older homo/bisexual men experienced a greater (though non-significant) rise than younger homo/bisexual men, despite the fact that these men are most likely to have been exposed to the HIV safer sex campaigns in the 1980s. There appears to be scope for improvements in sexual health promotion in GUM clinics as a priority area for the control and prevention of gonorrhoea.

STUDY 2: CHANGE IN RISK FACTORS FOR GONORRHOEA BETWEEN 1994 AND 1996

There is no evidence to suggest that the rise in cases of gonorrhoea seen at GUM clinics in England between 1994 and 1996 was driven by any single risk group. The rise was greatest in heterosexual men but there were also large rises in women and in homo/bisexual men. It is particularly important to recognise that although black Caribbeans are at higher risk than whites for the acquisition of gonorrhoea, the rate of increase of new cases in whites and black Caribbeans was similar. Recording of ethnicity improved in the Thames regions between 1994 and 1996, but not elsewhere, and this may explain the slightly higher (though non-significant) odds ratios associated with black Caribbeans compared with white people. Otherwise, the odds ratios calculated for study 2 are likely to be valid, as a major shift in the size of the various population subgroups between 1994 and 1996 is unlikely.

It is possible that the rise reflects an increase in incidence following sexual behaviour changes generally. A smaller increase in numbers of diagnoses among homosexual men in 1989 and 1990 was associated with changes in risk behaviour which resulted in increased HIV transmission in this group. Furthermore, the significant increases in penicillin resistant gonorrhoea observed in homo/bisexual men in this study may also have increased the likelihood of treatment failure in this group. If so, it is possible that this enhanced gonorrhoea transmission in homo/bisexual men to some extent contributed to the rise in numbers of diagnoses.

It is also possible that the rise in numbers of diagnoses could be contributed to by changes in the use of genitourinary medicine services, such as changes in health seeking behaviour resulting from sexual health promotion initiatives, or increased referrals from other healthcare settings. A change in attendance resulting from successful health promotion initiatives is perhaps unlikely in this case. Heterosexual males were the largest group in this study, and they experienced the greatest rise.

Almost all infections in heterosexual males were genital, and as genital infections in males are usually asymptomatic, it is reasonable to conjecture that attendance at the GUM clinic in this group was stimulated by the presence of symptoms. Also, a study of 18 general practices in 1994 found very few were treated for gonorrhoea in this setting (unlike those who presented with genital herpes and warts, half of whom were treated in general practice without being referred to GUM clinics), such that a major shift from general practice to GUM clinics for the treatment of gonorrhoea seems implausible (CDSC, unpublished data).

In heterosexual patients, the rise among teenage females was not significantly greater than in older females but may represent a general sexual health problem in that group. Almost one in five (though not significantly greater than in older females) reporting one sexual partner may be due to increased flow of infections from high risk core groups to non-core groups, resulting in a greater proportion of cases in 1996 reporting only one sexual partner. Caution with this interpretation is required, however, given the likelihood of variations in accuracy and recording of information on numbers of partners.

Homosexually acquired gonorrhoea increased particularly in the North West. Given the substantial caseload of HIV infections in the Manchester area, this demonstrates the value of gonorrhoea surveillance data in indicating where there is a potential or real need for local public health action.

The study protocol was submitted to the Public Health Laboratory Service ethics committee who, after consultation with the chairman of the South Thames multicentre research ethics committee (MBREC), felt that this study was essentially an outbreak investigation designed to inform immediate public health policy and, as such, did not require ethical approval.

The study would not have been possible without the commitment and hard work of medical, nursing, and administrative staff at all the participating GUM clinics. They are gratefully acknowledged. The authors also wish to thank Dr A Swan and Mrs P Rogers for additional statistical input, Mr A Brady, Dr K Fenton, Dr G Luzzi, Mr N Macdonald, Dr Macdonald-Burns, Dr A Nicoll, Mr I Simms, Dr M Tennant-Flowers, and Dr N Thin for helpful comments on an earlier draft of this paper, and Mrs P Deeks and Ms S Wellstead for administrative support.

Contributors: GH, NA, and MC designed the study; GH analysed the results with statistical guidance from NA; GH, MC,
Hughes, Andrews, Catchpole, et al.

The following clinicians and clinics participated in the study.

Anglia and Oxford
Dr Shannagaranjan, Bedford Hospital; Dr Fawcett, Peterborough District Hospital (Fenland Wing); Dr Greenhouse, Ipswich Hospital; Dr Carne, Addenbrooke’s Hospital, Cambridge; Dr Luzzi, Wycombe General Hospital; Dr Sherrard, Orchard Health Centre, Banbury.

North Thames
Dr Simmons, St Bartholomew’s Hospital; Dr Forster, Royal London Hospital; Dr Macdonald-Burts, Royal Free Hospital; Dr Murphy, Central Middlesex Hospital (Patrick Clements Clinic); Dr Matti, Hertford County Hospital; Dr Wear, Barnet General Hospital; Dr Jebakumar, Halstead Hospital; Dr Coelho, St John’s Hospital, Chelmsford; Dr Daniel, West Midlands University Hospital; Dr Kell, Whittington Hospital (Archway Sexual Health Clinic); Dr Mercer, Mortimer Market Centre; Dr Wisdom, Newham General Hospital; Dr John, Hemel Hempstead General Hospital; Dr McLean, Charing Cross Hospital (Chelsea and Westminster); Dr Lawrance, Westminister Hospital (Victoria Clinic); Dr Barton, St Stephens Centre (John Hunter Clinic).

South and West
Dr Chatterjee and Dr Mandal, Manchester Royal Infirmary; Dr Lach, Walsall Street Health Centre, Rochdale; Dr O’Mahoney, Countess of Chester Hospital; Dr Ferrer, Royal Albert Edward Infirmary, Wigan; Dr Woolley, Trafford General Hospital, Manchester; Dr Curless, Bolton General Hospital; Dr Huggins, North Manchester Hospital; Dr Thambur, Bury General Hospital; Dr Ahmed, Tameside General Hospital, Ashton under Lyne.

Northern and Yorkshire
Dr Battidge, Harrogate District Hospital; Dr Rajah, North Tees General Hospital, Stockton on Tees; Dr Opaneye, Middlesbrough General Hospital; Dr Stanley, Workington Infirmary, Cumbria; Dr Jebakumar, Halstead Hospital; Dr Saravanmuttu, Sunhope Parade Health Centre, South Shields; Dr Watson, Tynemouth Victoria Jubilee Infirmary, North Shields; Dr Al-Egaily, Princess Royal Community Health Centre, Rochdale; Dr O’Molloy, Salford General Hospital, Ashton under Lyne.

South Thames
Dr Sulaiman, Cheltenham General Hospital (Benhall Clinic); Dr Wilcox, Torbay Hospital, Torquay; Dr George, North Devon District General Hospital, Barnstaple; Dr Basu-Ray, Panel Suite, Royal Bournemouth Hospital; Dr Keane, Royal Cornwall Hospital, Truro; Dr Sulaiman, Gloucester Royal Hospital; Dr Baku, St Mary’s Hospital, Isle of Wight; Dr Tobin, St Mary’s Hospital, Portsmouth; Dr Morrison, Freedom Fields Hospital, Plymouth; Dr Woodcock, Royal Hampshire County Hospital; Dr Rabindran, Chippingham Community Hospital; Dr Kazi, Princess Margaret Hospital, Swindon; Dr Jackson, North Hampshire Hospital (Basingstoke District Hospital).

Trent
Dr Kinghorn, Royal Hallamshire Hospital, Sheffield; Dr Gupta, Scunthorpe General Hospital; Dr Fisk, Leicester Royal Infirmary; Dr Fraser, Chesterfield and North Derbyshire Hospital, Chesterfield; Dr Clay, Pilgrim Hospital, Boston.

North East
Dr Kinghorn, Royal Hallamshire Hospital, Sheffield; Dr Carne, Addenbrooke’s Hospital, Cambridge; Dr Luzzi, Wycombe General Hospital; Dr Sherrard, Orchard Health Centre, Banbury.

West Midlands
Dr Kinghorn, Royal Hallamshire Hospital, Sheffield; Dr Carne, Addenbrooke’s Hospital, Cambridge; Dr Luzzi, Wycombe General Hospital; Dr Sherrard, Orchard Health Centre, Banbury.

South and West
Dr Chatterjee and Dr Mandal, Manchester Royal Infirmary; Dr Lach, Walsall Street Health Centre, Rochdale; Dr O’Mahoney, Countess of Chester Hospital; Dr Ferrer, Royal Albert Edward Infirmary, Wigan; Dr Woolley, Trafford General Hospital, Manchester; Dr Curless, Bolton General Hospital; Dr Huggins, North Manchester Hospital; Dr Thambur, Bury General Hospital; Dr Ahmed, Tameside General Hospital, Ashton under Lyne.

Northern and Yorkshire
Dr Battidge, Harrogate District Hospital; Dr Rajah, North Tees General Hospital, Stockton on Tees; Dr Opaneye, Middlesbrough General Hospital; Dr Stanley, Workington Infirmary, Cumbria; Dr Jebakumar, Halstead Hospital; Dr Saravanmuttu, Sunhope Parade Health Centre, South Shields; Dr Watson, Tynemouth Victoria Jubilee Infirmary, North Shields; Dr Al-Egaily, Princess Royal Community Health Centre, Rochdale; Dr O’Molloy, Salford General Hospital, Ashton under Lyne.

South Thames
Dr Sulaiman, Cheltenham General Hospital (Benhall Clinic); Dr Wilcox, Torbay Hospital, Torquay; Dr George, North Devon District General Hospital, Barnstaple; Dr Basu-Ray, Panel Suite, Royal Bournemouth Hospital; Dr Keane, Royal Cornwall Hospital, Truro; Dr Sulaiman, Gloucester Royal Hospital; Dr Baku, St Mary’s Hospital, Isle of Wight; Dr Tobin, St Mary’s Hospital, Portsmouth; Dr Morrison, Freedom Fields Hospital, Plymouth; Dr Woodcock, Royal Hampshire County Hospital; Dr Rabindran, Chippingham Community Hospital; Dr Kazi, Princess Margaret Hospital, Swindon; Dr Jackson, North Hampshire Hospital (Basingstoke District Hospital).

Trent
Dr Kinghorn, Royal Hallamshire Hospital, Sheffield; Dr Gupta, Scunthorpe General Hospital; Dr Fisk, Leicester Royal Infirmary; Dr Fraser, Chesterfield and North Derbyshire Hospital, Chesterfield; Dr Clay, Pilgrim Hospital, Boston.
**LETTERS TO THE EDITOR**

**Successful treatment of recalcitrant condyloma with topical cidofovir**

**Editor,**—Despite the high prevalence of condylomata acuminata, their treatment remains unsatisfactory for both patients and physicians. Epidemiological studies estimated the prevalence of genital warts between 1–31% with a peak occurring in young adults. As a consequence, the economic burden of human papillomavirus (HPV) infection in the United States is estimated to exceed $8.5 billion per year.1 Current treatments rely on the ablation of warts (cryotherapy, laser vaporisation, electrodissection, or trichloroacetic acid) or the interruption of cell division (podophyllotoxin, intralesional or systemic interferon, and 5-fluorouracil). Recently, imiquimod has been successfully used as a topical immune response modifier for the treatment of external anogenital warts.2 However, there remains a substantial number of patients who fail to respond to traditional and newer drugs. We report on such a patient with recalcitrant condylomata acuminata on the glans and shaft of the penis who was successfully treated using the novel virustatic cidofovir as a 1.5% gel.

A 48 year old man with a 2 1⁄2 year history of condylomata acuminata presented to the Oxford genitourinary medicine clinics with small erosions surrounded by a marked erythema on all treated sites (fig 1). The lesions were painful. Condylomata were still present in the corona sulcus. At this point treatment was stopped and antiseptic treatment was given with betadine solution once daily. Seven weeks later (week 13) all lesions had completely healed (fig 1). Neither scarring nor dysaesthesia were noted. No recurrence has occurred since. Cidofovir, 1-[(S)-3-hydroxy-2-(phosphono-methoxy)-propyl]cytosine, is a member of a new class of antiviral agents (phosphonylethyl ether nucleotide analogues).3 It shows potent in vitro activity against a broad spectrum of herpesviruses, including human cytomegalovirus (CMV), HSV-1 and HSV-2, and adenoviruses.4 Recent in vitro and in vivo studies have demonstrated activity against papillomavirus and poxvirus.

Cidofovir is a nucleotide analogue of deoxyxytidine monophosphate (dCMP). Analogous to the metabolism of dCMP to dCTP, cidofovir is converted to the active cidofovir diphosphate that inhibits viral DNA polymerases. Once cidofovir enters cells is slow, but the intracellular half life of the various metabolites is between 6 and 87 hours, thus allowing infrequent dosing.5 Compared with the general mechanism of activation of ganciclovir, which requires phosphorylation by the virus encoded UL97 gene, cidofovir does not depend on viral infection for its phosphorylation and can therefore prime cells to an antiviral state (prophylaxia).

The metabolism of cidofovir is negligible, since the majority (>80%) is recovered unchanged in the urine. The principal systemic toxicity (nephrotoxicity) can be avoided by topical application.

This initial case report suggests that topical cidofovir may represent a valuable addition to the armamentarium of hard to treat condyloma. However, a careful evaluation of the dose and frequency of cidofovir application is warranted.

U R HENGGE
Department of Dermatology and Venerology, University of Essen, Hufelandstrasse 55, 45122 Essen, Germany

G TIETZE
Hospital Pharmacy, University of Essen, Hufelandstrasse 55, 45122 Essen, Germany

Correspondence to: U R Hengge
dermatology@uni-essen.de


**Bladder carcinoma presenting to genitourinary medicine departments**

**Editor,**—Large numbers of patients are seen in departments of genitourinary medicine with symptoms suggesting lower urinary tract inflammation of the genitourinary tract. Although bladder neoplasms typically cause painless haematuria, in a subgroup of patients they cause other urinary symptoms that may produce diagnostic confusion. We identified five patients who were referred to the genitourinary medicine service, and who were found to have bladder carcinoma (see table 1). Four of the patients presented to the genitourinary medicine department at High Wycombe (5500 new attendances per annum) between 1991 and 1998; the fifth patient presented to the Oxford genitourinary medicine department (9000 new attendances per annum) in 1997. None of the patients had an occupational history that placed them at higher risk for bladder cancer.

Men with bladder carcinoma typically present in later life (median age 70 years), but the condition may occur at younger ages. A subgroup of patients develops frequency, urgency, and dysuria—symptoms usually associated with bladder infection. Rarely, penile and perineal pain mimicking prostatitis may be a presenting feature, as in patients 3 and 4, who have been described in more detail elsewhere.

Non-specific urethritis (NSU) is diagnosed commonly in genitourinary medicine clinics in men of all ages. In this series, patient 2 was referred with presumed NSU, and patient 4 had attended previously with a diagnosis of NSU. 2 years before the bladder cancer was diagnosed (at that time there were 5–10 white cells/high power field (×1000) on a urethral smear, and a chlamydia ELISA test and cultures for Neisseria gonorrhoeae were negative; no haematuria was detected). Both patients were subsequently noted to have neoplastic infiltration in the bladder neck area and prostatic urethra.

In all five cases a persistent microscopic haematuria was noted at presentation; in patient 4 this was never greater than a trace on dipstick testing. Patient 1 reported intermittent painless macroscopic haematuria at presentation; he was referred by his general practitioner with suspected sepsis, without haematuria; he was referred with presumed NSU, and patient 4 had attended previously with a diagnosis of NSU. 2 years before the bladder cancer was diagnosed (at that time there were 5–10 white cells/high power field (×1000) on a urethral smear, and a chlamydia ELISA test and cultures for Neisseria gonorrhoeae were negative; no haematuria was detected). Both patients were subsequently noted to have neoplastic infiltration in the bladder neck area and prostatic urethra.
Table 1 Patient details

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age (years)</th>
<th>Smoker</th>
<th>Referal source</th>
<th>Referal diagnosis</th>
<th>Presenting features</th>
<th>Urine dipstick</th>
<th>Urine cytology</th>
<th>Diagnosis and treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26</td>
<td>NR</td>
<td>GP</td>
<td>?Infection</td>
<td>3 months intermittent painless haematuria; 6 weeks frequency, dysuria</td>
<td>Blood +ve (trace)</td>
<td>ND</td>
<td>Well differentiated bladder papillary TCC; non-invasive; resected</td>
</tr>
<tr>
<td>2</td>
<td>34</td>
<td>Yes</td>
<td>GP</td>
<td>?Urethritis</td>
<td>6 weeks frequency, dysuria; suprapubic pain</td>
<td>Blood +ve</td>
<td>ND</td>
<td>Poorly differentiated adenocarcinoma; bladder calculus also present; tumour resection, chemotherapy, and radiotherapy</td>
</tr>
<tr>
<td>3</td>
<td>53</td>
<td>No</td>
<td>GP</td>
<td>Recurrent prostatitis</td>
<td>1 year penile and perineal pain; frequency, dysuria</td>
<td>Blood +ve</td>
<td>Malignant</td>
<td>Extensive transitional cell carcinoma in situ, involving prostatic urethra; cystoprostatectomy</td>
</tr>
<tr>
<td>4</td>
<td>42</td>
<td>No</td>
<td>GP</td>
<td>Sterile pyuria</td>
<td>1 year penile and perineal pain</td>
<td>Blood +ve (trace)</td>
<td>Malignant</td>
<td>Extensive TCC plus carcinoma in situ, involving prostatic urethra; cystoprostatectomy</td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>Yes</td>
<td>GP</td>
<td>?Infection</td>
<td>6 weeks frequency, urgency, dysuria</td>
<td>Blood +ve</td>
<td>Suspicious</td>
<td>Poorly differentiated TCC at bladder neck; muscle invasion; cystoprostatectomy, and chemotherapy</td>
</tr>
</tbody>
</table>

NR = not recorded; ND = not done; TCC = transitional cell carcinoma.

Atrial myxoma and HIV infection

The patient was diagnosed with asymptomatic HIV infection in February 1987 when she was aged 50 years. Her CD4 count was 690 ×10^3/μl at this time. HIV infection was acquired from a bisexual male partner. In December 1990 the CD4 lymphocyte count had fallen to 190 ×10^3/μl and zidovudine monotherapy was started. The patient died in 1996 when she was prescribed a combination regimen. These cases highlight the importance of careful follow up of patients presenting with persistent irritative-type bladder symptoms, especially in an older age group, when specific tests for genitourinary infection are negative, and where microscopic haematuria is a feature. Bladder carcinoma should be considered in this subgroup; urine cytology and referral for cystourethroscopy may be indicated. Although rare in younger adult males, bladder cancer should not be ruled out in men under the age of 45 years, and our experience strengthens the case for continuing with routine urine testing in genitourinary medicine clinics. The myxo-
endogenous healthy vaginal lactobacillus? In an interesting hypothesis, Blackwell described the possible effect of biochemical and microbial abnormalities in the vagina on BV recurrence.7 She also quoted Berger’s description of concordant vaginal floras in lesbian couples, suggestive of a mechanical transfer of an infectious agent.8 Is it not possible for mouth organisms or hostile salivary enzymes to induce biological and microbial abnormalities in the vagina? Furthermore, mechanical transfer of infectious agents in lesbian couples is most likely to occur via cunnilingus, a not uncommon thing an index case is told when his/her cooperation is called for in the contact tracing process. Only when it becomes widely known in a clinic that such confidentiality is thoroughly pursued will counterproductive fears be eliminated. With understanding and cooperation it can be done.

Table 1 BV prevalence results

<table>
<thead>
<tr>
<th>Lesbians</th>
<th>BV diagnosed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>No of women</td>
</tr>
<tr>
<td>Practised receptive cunnilingus in previous 4 weeks</td>
<td>17</td>
</tr>
<tr>
<td>Did not practise receptive cunnilingus</td>
<td>9</td>
</tr>
<tr>
<td>Heterosexual women</td>
<td>No of women</td>
</tr>
<tr>
<td>Total</td>
<td>256</td>
</tr>
<tr>
<td>Practised receptive cunnilingus in previous 4 weeks</td>
<td>111</td>
</tr>
<tr>
<td>Did not practise receptive cunnilingus in past 4 weeks</td>
<td>145</td>
</tr>
</tbody>
</table>

Is partner notification in the public interest?

EDITOR,—We recently conducted a national urban random sample survey of 1400 men of sexually active in the Dominican Republic to measure possible change in sexual behaviour. This sexual behaviour change (SBC) survey was prompted by results from the 1996 demographic and health survey, which found that 84.8% of a national random sample of Dominican men claimed that they had changed their behaviour in some way because of their fear of, or concern about, AIDS. The proportion of respondents reporting behavioural change such as becoming monogamous or reducing their number of sexual partners was about triple the proportion reporting condom adoption. In our SBC survey, 79% of respondents claimed to have changed behaviour because of concern about AIDS. A majority (52.2%) said they had become monogamous or reduced their number of sexual partners. This was followed by condom adoption (14.6%), only having sexual relations with a person they know (13.9%), avoiding relations with “prostitutes” (9.0%), or becoming abstinent (1.6%). A small proportion (2.8%) had not yet begun to have sexual relations. As with the Dominican DHS findings, we see that most answers are classifiable as behaviour change, as distinct from condom adoption. This follows a pattern found in recent studies in countries such as Uganda and Zambia. A recent review of findings from behavioural change surveys in 16 countries in Africa, Latin America, and the Caribbean shows that partner reduction is more often reported than condom adoption.9 If sizeable numbers of men reduce their number of sexual partners, can this have significant impact on HIV infection rates? Urban HIV seroprevalence among the general or low risk Dominican population seems to have stabilised at the 1.9–2.0% level since 1995, according to the US Census Bureau. Recent studies that have modelled the impact of different interventions on HIV infection rates in east Africa suggest that reduction in number of partners can have a great impact on averting HIV infections, in fact greater than either condom use or treatment of STDs.10 Of course, impact of partner reduction on HIV infection rates would be especially strong where there is relatively high HIV seroprevalence among potential partners. In view of these modelling studies as well as population based surveys such as the two cited from the Dominican Republic, perhaps there ought to be greater equity in resource allocation between HIV/AIDS prevention programmes promoting behaviour change—such as monogamy/fidelity or at least reduction of number and frequency of change of sex partners—and far more familiar programmes that promote and provide condoms.

EDWARD C GREEN
ALDO CONDE
2807 38th Street, NW Washington, DC 20007, USA
Correspondence to: Dr Green


Accepted for publication 25 February 2000

Features of AIDS and AIDS defining diseases during the highly active antiretroviral therapy (HAART) era, compared with the pre-HAART period: a case-control study

EDITOR,—To assess the features of AIDS defining illnesses during the HAART era versus those observed before the introduction of HAART, the characteristics of 72 consecutive patients diagnosed in 1995 were compared with those of 144 subjects randomly selected from the 436 patients diagnosed from 1985 to 1995, in a case-control study.

An impressive drop in AIDS diagnosis was seen shortly after the introduction of HAART, with only 38, 21, and 13 cases per 1000 patient years observed in 1997, 1998, and 1999 respectively, versus a mean frequency >60 cases per 1000 patient years, demonstrated during the pre-HAART period. A tendency toward an increased incidence of female sex was shown in 1997–9 compared with 1985–95 (33.3% versus 27.1%), together with a rise of mean CD4 lymphocyte count (86.8 (SD 99.4) versus 72.1 (93.7) cells x10³/µl), while an increase in the mean patient age was highly significant (39.8 (8.3) versus 34.6 (7.7) years; p<0.0001). When considering the exposure to HIV infection, drug abuse became significantly less important in the HAART era (p<0.05), while heterosexual transmission was notably increased (34.7% versus 13.2% of cases; p<0.0003). The distribution of AIDS defining disorders during the HAART era showed a tendency to a reduction in cytomegalovirus, cryptocoecosis, mycobacteriosis, cryptococcosis, and HIV encephalopathy, while a relative increase in pneumocystis, non-Hodgkin’s lymphoma, and Kaposi’s sarcoma were stable (table 1). However, while pneumocystis, candida, and non-Hodgkin’s lymphoma were found; neurotoxoplasmosis and Kaposi’s sarcoma were stable (table 1). However, while pneumocystis, candida, and cryptocoecosis, mycobacteriosis, and HIV encephalopathy, mycobacteriosis (which ranked fifth to eighth in


Accepted for publication 25 February 2000

frequency during the pre-HAART era, virtually disappeared after the introduction of HAART (28 versus four overall cases; p<0.007), together with cryptosporidiosis. Neoplasms and HIV related disorders (encephalopathy) and wasting syndrome showed a slightly increased frequency during the HAART era (16.8% and 9.2% during 1997–9, versus 13.2% and 7.9% respectively, during the pre-HAART period). A considerable trend to increased mean CD4+ count during the pre-HAART period (a drop of cytomegalovirosis, mycobacteriosis, cryptococcosis, and wasting syndrome), and prophylactic strategies. The European Network for HIV/AIDS research and prevention in prostitution, Department of Clinical and Experimental Medicine, University of Bologna, was treated with HAART for more than 3 months (70.1%).

AIDS defining events and mean CD4+ lymphocyte count at disease occurrence, in the two considered time periods

<table>
<thead>
<tr>
<th>AIDS defining diseases</th>
<th>No of diseases (%)</th>
<th>Mean CD4+ count (cells x10^6/L (SD))</th>
<th>No of cases</th>
<th>Mean CD4+ count (cells x10^6/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumocystis carinii pneumonia</td>
<td>40 (26.5)</td>
<td>58.6 (49.0)</td>
<td>22 (28.9)</td>
<td>62.4 (72.1)</td>
</tr>
<tr>
<td>Oropharyngeal candidiasis</td>
<td>21 (13.3)</td>
<td>71.3 (62.2)</td>
<td>16 (21.0)</td>
<td>129.9 (98.1)</td>
</tr>
<tr>
<td>Neurotoxoplasmosis</td>
<td>17 (11.3)</td>
<td>79.9 (62.1)</td>
<td>9 (11.8)</td>
<td>75.6 (39.2)</td>
</tr>
<tr>
<td>Kaposi's sarcoma</td>
<td>15 (9.9)</td>
<td>98.1 (101.3)</td>
<td>7 (9.2)</td>
<td>133.3 (68.3)</td>
</tr>
<tr>
<td>Cryptococcal meningitis or disseminated disease</td>
<td>21 (13.9)</td>
<td>77.3 (100.2)</td>
<td>11 (1.3)</td>
<td>148 (10.6)</td>
</tr>
<tr>
<td>HIV encephalopathy (AIDS-dementia complex)</td>
<td>7 (4.6)</td>
<td>81.1 (45.9)</td>
<td>2 (2.6)</td>
<td>102.0 (29.7)</td>
</tr>
<tr>
<td>Extrapulmonary pneumocystosis</td>
<td>6 (4.0)</td>
<td>25.2 (19.4)</td>
<td>0 (5.0)</td>
<td>6 (0.0)</td>
</tr>
<tr>
<td>Disseminated mycobacteriosis</td>
<td>5 (3.3)</td>
<td>62.4 (41.1)</td>
<td>4 (5.3)</td>
<td>96.8 (11.3)</td>
</tr>
<tr>
<td>Wasting syndrome</td>
<td>5 (3.3)</td>
<td>38.4 (41.1)</td>
<td>5 (6.6)</td>
<td>121.2 (54.0)</td>
</tr>
<tr>
<td>Non-Hodgkin’s lymphoma or primary CNS lymphoma</td>
<td>5 (3.3)</td>
<td>116.3 (41.1)</td>
<td>4 (5.3)</td>
<td>125.9 (71.2)</td>
</tr>
<tr>
<td>Cryptosporidiosis</td>
<td>4 (2.7)</td>
<td>38.3 (30.2)</td>
<td>0 (5.0)</td>
<td>6 (0.0)</td>
</tr>
<tr>
<td>Tuberculosis (pulmonary or disseminated disease)</td>
<td>3 (2.0)</td>
<td>148.2 (51.4)</td>
<td>5 (6.6)</td>
<td>289.3 (71.2)</td>
</tr>
<tr>
<td>Other AIDS defining events</td>
<td>13 (8.6)</td>
<td>55.3 (48.9)</td>
<td>4 (5.3)</td>
<td>73.3 (101.1)</td>
</tr>
</tbody>
</table>

How do you begin to address the sexual health needs of commercial sex workers (CSWs)? Here you will find (most of the) answers. This immensely practical book is essential for those setting up an outreach service, or simply wishing to know more about commercial sex work. It is the outcome of a series of projects and workshops, written by people providing services to CSWs throughout Europe, and drawn from the lessons learnt by these pioneering workers and clients. It is written with great clarity and frankness. The A4 layout is bold, imaginative and attractive, with illustrations of promotional literature. Its European inclusiveness means that sadly it cannot be specific regarding, for example, the law as it applies to commercial sex. It does, however, give the broad framework within which providers must acquaint themselves wherever they work. It takes us through the steps; of funding, the scope of the service, useful contacts, where to make contact with CSWs, and so on. Importantly, in the current climate there are sections on evaluation and monitoring of the service, the legal and political context of the work, and dealing with the media. It stresses the heterogeneous nature of commercial sex workers whether male, female, or transsexual, and the spectrum of commercial sex venues. Peer educator programmes are covered in some detail. There are fascinating pieces of practical advice—for example, cooperate with police, but don’t be identified too closely with law enforcement. Advising police of your outreach vehicle’s registration number may prevent you being stopped for kerb crawling! You can set up a flawless screening service and find only a few CSWs attend. The book reminds us middle class, health aware professionals that, for many, sexual health is not a priority. We are perennially worried about the “indifference, hostility and self destructive behaviour”; that her next fix, a roof over her head, or the desire to have a baby might be more important to the CSW than the nugilous risk of HIV. Address some of these needs and you have the carrot to attract attention to and confidence in your service. The spin off is that clients can then benefit from STD screening and safer sex advice. Simply providing toilets and somewhere safe to have a cup of tea may be enough for some.

I would have liked to see a further reading list, but this book fulfills its remit excellently.

MARY STEVENSON

Hustling for Health. Developing Services for Sex Workers in Europe. Pp 83; Price 10 euros. The European Network for HIV/STI Prevention in Prostitution (EUROPAP/TAMPEP), 1998. Contact Judith Kelvin/Helen Ward, Coordinating Centre, European Network for HIV/STI Prevention in Prostitution, Department of Epidemiology and Public Health, Imperial College School of Medicine, London W2 1PG (tel: 0207 594 3315; fax: 0207 402 2150; email: europap@ic.ac.uk). Also available in nine other European languages (Danish, Finnish, Flemish, French, German, Greek, Italian, Portuguese, Spanish), and the full text (without illustrations) can be found online on the website (http://www.med.ic.ac.uk/idf/dhm/hivprophylactic/press.htm).

“Venerale diseases are like the fine arts—it is pointless to ask who invented them.” (Voltaire, Dictionary philosophy)

Sexually transmitted diseases (STDs) now rank among the top ten diseases for which adults in developing countries seek health care. The economic burden of STDs on both developed and developing countries is enormous. Infection with conventional STDs is a risk factor for transmission of infection with HIV, and therefore for the development and spread of the AIDS.

It is important that laboratory services are available to guide the clinician to the correct diagnosis and treatment of these conditions, and to give an accurate epidemiological picture of their prevalence in a particular community in order to target appropriate preventative programmes and ensure optimal and economic use of available resources. Yet, the availability of both funds and technology varies widely between different settings.

This manual sets out to give comprehensive guidance on tests available and applicable to the level of expertise and funding available.

Nine chapters cover the major STDs, encompassing bacterial and viral infections, and under the umbrella of genitalia in adults; trichomoniasis, candidiasis, and bacterial vaginosis. Each chapter begins with a brief description of the microbiology of the infective agent and the clinical spectrum of disease. The detail given is not consistent, being comprehensive for chancroid and granuloma inguinale, and surprisingly brief for HIV and chlamydia by way of contrast. Then follows a description of collection and transport requirements, and of techniques for diagnosis. The emphasis is on tests that are possible in a reasonably well equipped laboratory and are not too dependent on specific facilities. Tests that are suitable for use in the field are highlighted. An evaluation of sensitivity and specificity is also given. Other tests available in central or reference laboratories are mentioned in brief, usually with supporting references.

Two annexes cover media, reagents and stains, and details of equipment required to diagnose each condition. A third annex is an interesting table of which tests should be available at “peripheral”, “intermediate”, and “central” laboratories.

Overall, this manual is to be welcomed as an educational and reference source for medical microbiologists, technologists, and clinicians. However, I would recommend that the authors “road test” the manual to discover omissions in technical detail that would prevent the sole use of the manual in the field.

Indifferent colour reproduction detracts from the quality of the text—for example, blue reactions appearing as red in the figure. For the next edition, a chapter on basic microbiological techniques and another on the general principles and interpretation of laboratory tests would provide useful introductions to an otherwise excellent publication.

R S MORTON

Department of Clinical Microbiology, UCH Accident and Emergency Building, London WC1E 6DB
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: isidbox@aol.com).

Sexually Transmitted Diseases in a Changing Europe, 14–15 April 2000, Rotterdam, The Netherlands
Further details: Medicson, Organisation for Medical Congresses, PO Box 113, 5660 AC Geldrop, Netherlands (tel: +31-(0)40-2852212; fax: +31-(0)40-2851966; email: MEDICSON@IAEvil.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 Venerology, Sw Rocha 3, 15-879 Bialystok, Poland (tel/fax: (095) 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@bbam.ac.uk).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics, and Gynaecology, Advanced Course in Fetal Medicine, 22–24 May 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, Advanced Course in Fetal Medicine, 22–24 May 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, Advanced Course in Fetal Medicine, 22–24 May 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, Advanced Course in Fetal Medicine, 22–24 May 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, Advanced Course in Fetal Medicine, 22–24 May 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, Advanced Course in Fetal Medicine, 22–24 May 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, Advanced Course in Fetal Medicine, 22–24 May 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, Advanced Course in Fetal Medicine, 22–24 May 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, Linneagenat 89A, 114 86 Stockholm, Sweden (tel: +46 8 459 6600; fax: +46 8 661 91 25; email: aids2000@congrex.se).

Further details: Shirley Corley, Conference manager, Dart Associates, PO Box 781, Lane Cove, 2066 NSW, Australia (tel: 02 9418 9396/97; fax: 02 9418 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 19; 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).

Consortium of Thai Training Institutes for STDs and AIDS—International Review and Refresher Course on Sexual Health, Lee Garden Plaza Hotel, Hat Yai, Thailand 24–26 November 2000
Further details: Dr Yai Secretariat, Dr Verapol Palanuvej, Bangkok Hospital, 189 Sathorn Road, Bangkok 10120, Thailand (fax: (66-2) 286 3013; email: pthanit@email.ksc.net).

Corrections
An error occurred in an original article by Hughes et al that appeared in the February issue of the journal (2000;76:18–24). In the participants section under West Midlands, “Dr Wade, Coventry and Warwickshire Hospital” should read “Dr Wade and Dr Allan, Coventry and Warwickshire Hospital.”
Chlamydia

Chlamydia trachomatis infection as a risk factor for invasive cervical cancer.

RJ SUCHLAND, DD ROCKEY, JP BANNANTINE, WE BRADY, KS STURMAD, H BRUMBLAY, K DIOP, J IKEHATA, K NUMAZAKI, S CHIBA, S MACMILLAN, A TEMPLETON.

Screening for Chlamydia trachomatis in subfertile women.

S MACMELAN, A TEMPLETON. Hum Reprod 1999;14:3009–12

Analysis of Chlamydia trachomatis sevors in endocervical specimens derived from pregnant Japanese women.


Molecular epidemiology of genital Chlamydial trachomatis infection in high-risk women in Senegal, West Africa.


Evaluation of a rapid assay for diagnosis of Chlamydia trachomatis infections in outpatient clinics in South Kalimantan, Indonesia.


Seroactivity to Chlamydia trachomatis Hsp10 correlates with severity of human genital tract disease.


Immunogenic and protective ability of the two developmental forms of Chlamydia in a mouse model of infertility.

S PAL, J RANGEL, EM PETERSON, LM DELAMAZA. Vaccine 1999;18:752–63

Subclinical chlamydial infection of the female mouse genital tract generates a potent protective immune response: implications for development of live attenuated chlamydial vaccine strains.


Isolates of Chlamydia trachomatis that occupy nonfusogenic inclusions lack IncA, a protein localized to the inclusion membrane.


The intercellular adhesion molecule type-I is required for rapid activation of T helper type 1 lymphocytes that control early acute phase of genital chlamydial infection in mice.


Candidiasis

Species and genotypic diversities and similarities of pathogenic yeasts colonizing women.


Isolated candidal prostatitis.

A ELELT, R VONKNOBLOCH, R NUSSE et al. J Urol 2000;163:244

Multilocus genotypes and DNA fingerprints do not predict variation inazole resistance among clinical isolates of Candida albicans.


Bacterial vaginosis

Prevalence of bacterial vaginoses and correlation of clinical to gram stain diagnostic criteria in low risk pregnant women.


Direct or referral microscopy of vaginal wet smear for bacterial vaginosis: experience from an STD clinic.

CS PETERSEN, AG DANIELSEN, J RENNEBERG. Acta Dermato-Venereol 1999;79:473–4

Trichomoniasis

Improved diagnosis of Trichomonas vaginalis infection by PCR using vaginal swabs and urine specimens compared to diagnosis by wet mount microscopy, culture and fluorescent staining.


Identification of Trichomonas vaginalis α-actin as the most common immunogen recognized by sera of women exposed to the parasite.


Pelvic inflammatory disease


Patterns of diagnosis and referral in women consulting for chronic pelvic pain in UK primary care.


Syphilis and other treponematoses

Response to standard syphillis treatment in patients infected with the human immunodeficiency virus.


Identification of Treponema pallidum subspecies pallidum in a 200-year-old skeleton specimen.


Validation of the INNO-LIA syphillis kit as a confirmatory assay for Treponema pallidum antibodies.


Hepatitis

Low risk of vertical transmission of hepatitis C virus by breast milk.


Urine from chronic hepatitis B virus carriers: implications for infectivity.


Herpes

Prevalence and incidence of herpessimplex virus type 2 infection among male Zimbabwean factory workers.


Relation between herpes simplex viruses and human immunodeficiency virus infections.

JL SEVERSON, SK TYRING. Arch Dermatol 1999;135:1393–7

References

1. STAMM, RHoeae transformation rates in risk factor for invasive cervical cancer.

2. OPA expression correlates with elevated transformation rates in Neisseria gonorrhoae.


Persistent stress as a predictor of genital herpes recurrence.

Rapid detection of HSV from cytologic specimens collected into ThinPrep fixative.

Treatment of primary herpes simplex virus infection in guinea pigs by imiquimod.

Protective immune correlates can segregate by vaccine type in a murine herpes model system.

Cellulose acetate phthalate (CAP): an ‘inactive’ pharmaceutic excipient with antiviral activity in the mouse model of genital herpesvirus infection.

Co-infection of acyclovir-resistant and acyclovir-sensitive herpes simplex type 2 virus strains in BS-C-1 cells.
K KEYWAN, E KATZ. Intervirology 1999;42:247–51

Immune responses and protection against vaginal infection after nasal or vaginal immunization with attenuated herpes simplex virus type-2.
EL PARR, MB PARR. Immunology 1999;98:639–45

Immunity induced by DNA immunization with herpes simplex virus type 2 glycoproteins B and C.
JC MESTER, TA TWOMEY, ET TEPE, DI BERNSTEIN. Vaccine 1999;18:875–83

Persistence of infectious herpes simplex virus type 2 in the nervous system in mice after antiviral chemotherapy.

Repression of viral transcription during herpes simplex virus latency.

The major neutralizing antigenic site on herpes simplex virus glycoprotein D overlaps a receptor-binding domain.

Herpes simplex virus type 2 glycoprotein G-negative clinical isolates are generated by single frame shift mutations.

Potential role for human, the cellular homolog of herpes simplex virus VPA16 (α gene trans-inducing factor) in herpesvirus latency.

Granzyme A, a noncytotoxic component of CD8(+) cell granules, restricts the spread of herpes simplex virus in the peripheral nervous systems of experimentally infected mice.

Intracellular localization of the UL31 protein of herpes simplex virus type 2.

Human papillomavirus infection

Pernicious papillomavirus infection.

Type-specific persistence of human papillomavirus DNA before the development of invasive cervical cancer.

Epidemiology of acquisition and clearance of human papillomavirus infection in women from a high-risk area for cervical cancer.

HPV transmission—still feeling the way.
A MINDEL, R TIDEMAN. Lancet 1999;354:2097

HPV DNA testing of self-collected vaginal samples compared with cytologic screening to detect cervical cancer.

HPV DNA testing in cervical cancer: screening results from women in a high-risk province of Costa Rica.
M SCHIFFMAN, R HERRERO, A HILDESHIEIM et al. JAMA 2000;283:87–93

Human papillomavirus testing for primary cervical cancer screening.

HPV-based cervical cancer screening in a population at high risk for HIV infection.

Screening for cervical neoplasia by self-assessment for human papillomavirus DNA.

Spontaneous evolution of human papillomavirus infection in the uterine cervix—a prospective observational study.

Cervical recurrence in women with human papillomavirus type 16, 18, 31, and 35 virus-like particles in a case-control study of cervical squamous intraepithelial lesions.

Anal intraepithelial neoplasia.

A randomized, controlled, safety study using imiquimod for the topical treatment of anogenital warts in HIV-infected patients.

Human papillomavirus type 16 E6 variant in cervical carcinoma: relationship to host genetic factors and clinical parameters.
CS BRADY, MF DUGGANKEEN, JA DAVIDSON et al. J Gen Virol 1999;80:3253–60

Favorable clinical outcome of cervical cancers infected with human papilloma virus type 58 and related types.
HC LAI, CA SUN, MH YU et al. Int J Cancer 1999;84:533–7


Improved amplification of genital human papillomaviruses.

Additional human papillomavirus types detected by the hybrid capture tube test among samples from women with cytological and colposcopical atypia.

PCR-RFLP-detected human papilloma virus infection in a group of Senegalese women attending an STD clinic and identification of a new HPV-68 subtype.

Detection of human papilloma virus genomes by the primed in situ (PRINS) labelling technique.

DNA vaccination of mice with plasmid expressing human papillomavirus 6 major capsid protein L1 elicits type-specific antibodies neutralizing pseudovirions constructed in vitro.

Capture ELISA and in vitro cell binding assay for the detection of antibodies to human papillomavirus type 6b virus-like particles in patients with anogenital warts.

Detection of high-risk cervical intraepithelial neoplasia and cervical cancer by amplification of transcription derived from integrated papillomavirus oncoproteins.
Antibodies against oncoproteins E6 and E7 of human papillomavirus types 16 and 18 in cervical-carcinoma patients from Russia.

HPV 16 E6 blocks TNF-mediated apoptosis in mouse fibroblasts LM cells.
PJ DUESKENTHUGHES, J YANG, SB SCHWARTZ. Virology 1999;264:55–65

CD4(+) tumor-infiltrating lymphocytes in cervical cancer recognize HLA-DR-restricted peptides provided by human papillomavirus-E7.

The E6 protein of human papillomavirus type 16 binds to and inhibits co-activation by CBP and p300.
D PATEL, SM HUANG, LA BAGLIA, DJ MCCANCE. EMBO J 1999;18:5061–72

The human papillomavirus type 16 E5 protein modulates phospholipase C-γ-1 activity and phosphatidylinositol turnover in mouse fibroblasts.
K CRUSIOS, M KASEGIN, V KINZEL, A ALONSO. Oncogene 1999;18:6714–8

Interaction between the HPV-16 E2 transcriptional activator and p53.
P MASSIMI, D PIM, C BERTOLI et al. Oncogene 1999;18:7748–54

The E8–E2C protein, a negative regulator of viral transcription and replication, is required for extrachromosomal maintenance of human papillomavirus type 31 in keratinocytes.

The differentiation-specific factor GDP/Cut represses transcription and replication of human papillomaviruses through a conserved silencing element.

Cervical cytology and colposcopy

Cervical cytology after 2000: where to go?

Comparative evaluation of seven cell collection devices for cervical smears.

Efficacy of cervical smear collection devices: a systemic review and meta-analysis.
P MARTHNER, R LIFORD, G JARVIS, HC KITCHENER. Lancet 1999;354:1763–70

Detection of false-negative Papanicolaou smears by rapid rescreening in a large routine cervical cytology laboratory.
B G WRIGHT, J A HALFORD, DJ DITCHMAN. Pathology 1999;31:379–81

Determining the cost-effectiveness of mass screening for cervical cancer using common analytic models.

A prototype computer image-based Papanicolaou smear proficiency test.

The diagnostic value of computer-assisted primary cervical smear screening: a longitudinal cohort study.
H DOORNJWAARD, YT VANDERSCHOUW, Y VANDERGRAAF et al. Mod Pathol 1999;12:995–1000

Detection of human herpesvirus 8 in cervical cells of Chinese women with abnormal Papanicolaou smears.

A study of the follow up patterns of women treated for CIN 2 and 3 before and after the introduction of the 1992 guidelines.

Cidofovir, a new approach for the treatment of cervix intraepithelial neoplasia grade III (CIN III).

Effects of chemotherapy and tamoxifen on cervical and vaginal smears in bone marrow transplant recipients.

Serum carotenoids and vitamins and risk of cervical dysplasia from a case-control study in Japan.

Vaginal 5-fluorouracil for high-grade cervical dysplasia in human immunodeficiency virus infection: a randomized trial.

Preclinical feasibility study of NMP179, a nuclear matrix protein marker for cervical dysplasia.

Fhit alterations in cancerous and non-cancerous cervical epithelium.

Other sexually transmitted infections

A randomized, double-blind, placebo-controlled trial of single-dose ciprofloxacin versus erythromycin for the treatment of chancroid in Nairobi, Kenya.
IM MALONZA, MW TYNDALL, JO NYINDIACHOKA et al. J Infect Dis 1999;180:18693

Cytotoxic distending toxin of Haemophilus ducreyi induces apoptotic death of Jurkat T cells.

Public health and social aspects

Encouraging use of coupons to stimulate condom purchase.
DP DAHL, GI GORN, CB WERNBERG. Am J Public Health 1999;89:1866–8

Microbiology and immunology

Human herpesvirus 8 cellular immune responses in homosexual men.

Correlation of behaviours with microbiological changes in vaginal flora.
JR SCHWERKE, CM RICHEY, HL WEISS. J Infect Dis 1999;180:1632–6

The identification of vaginal Lactobacillus species and the demographic and microbiologic characteristics of women colonized by these species.
MA ANTONIO, SE HAYES, ML HILLER. J Infect Dis 1999;180:1950–6

Common mucosal immunity: a novel hypothesis.
FA MOORE. Ann Surg 2000;231:9–10

Immunoglobulin concentrations and antigen-specific antibody levels in cervico-vaginal lavages of rhesus macaques are influenced by the stage of the menstrual cycle.

Evaluation of the bacterial flora of the prostate using a 16s rRNA gene based polymerase chain reaction.

Dermatology

Incidence of preputial lichen sclerosis in adults: histologic study of circumcision specimens.

Penile cancer among patients with genital lichen sclerosus.


Vulvar melanoma, biologically different from other cutaneous melanomas. CJ DUNTON, DB BEED. Lancet 1999;354:2013

Cytomegalovirus balanitis in a renal transplant recipient. A RODRIGUEZ, B HILL, R GOPOLAN, GN SKLAR. J Urol 1999;162:2086

The imidazoxinones, imiquimod and R-485 induce functional but not phenotypic systemic vasculitis. RP BURNS, B FERBEL, M TOMAI A RODRIGUEZ, B HILL, R GOPOLAN, GN SKLAR. CJ DUNTON, D BERD. NH COX, DJ EEDY, CA MORTON. F SCRIMIN, S RUSTJA, R RADLJ. Clin Infect Dis 2000;31:13–23

Miscellaneous

The staying power of sexually transmitted diseases. W CATES, G DALLABETTA. Lancet 1999;354:62

Breaking the silence surrounding rape. S RAMSAY. Lancet 1999;354:2018


Symptoms of reproductive-tract infection—not all that they seem to be. K TROLLEPANKUMAR. Lancet 1999;354:1745


Sexual and reproductive health: what about boys and men: Education and service provision are the keys to increasing involvement. G YAMEY. BMJ 1999;319:1315

Male adolescents and physician sex preference. CJ VANNES, DA LYNCH. Arch Pediat Adolesc Med 2000;154:49–54


Lesbians’ sexual history with men: implications for taking a sexual history. AL DIAMANT, MA SCHUSTER, R MCGUIGAN, J LEVER. Arch Intern Med 1999;159:2730–8

Hysterectomy and sexual function. JC RHODES, KH KERULFF, KW LANGENBERG, GM GUZINSKI. JAMA 1999;282:1934–41


Prophylactic antibiotics for intrauterine device insertion: a metaanalysis of the randomized controlled trials. DA GRIMES, RF SCHULZ. Contraception 1999;60:57–64


Neurochemical characterization of the vestibular nerves in women with vulvar vestibulitis syndrome. N BOHMSTARK, M HILGES, C FALCONER, E RYLANDER. Gynecol Obstet Invest 1999;48:270–5


Pudendal nerve injury associated with avid bicycling. VS RICHIUTI, CA HAAS, AD SEFTEL et al. J Urol 1999;162:2099


Asthma and epididymitis: the calm before the storm. GHM GEORGE, JR AXFORD. Ann Rheum Dis 1999;58:731–6


Is there a place for large vessel disease in the diagnostic criteria of Behçet’s disease? M SCHIRMER, KI CALAMIA, JD ODDY. J Rheumatol 1999;26:2511–2

Secondary inflammation of the appendix via the vagina. SA BUTLERMANUEL, PT TOWNSEND. J Roy Soc Med 1999;92:465

Two forms of reactive arthritis? P TOVANEN, A TOVANEN. Ann Rheum Dis 1999;58:737–41

Reactive or infectious arthritis. JG KULPERS, L KOHLER, H ZEIDLER. Ann Rheum Dis 1999;58:661–4

Beaver fever—a rare cause of reactive arthritis. M TUPCHONG, A SIMOR, C DEWAR. J Rheumatol 1999;26:2701–2