MATTERS ARISING

STD and inflammatory cervical cytology

The results as reported in the abstract of the paper *The association between sexually transmitted disease and inflammatory cervical cytology* by Dimian, *et al* (*Genitourin Med* 68:305-6) are at odds with the data presented in the paper. Assuming the numbers reported for ectropion and wart virus infection in table 3 and in the body of the paper are correct, the associated odds ratios are:

Ectropion 2.00 (95% CI 1.3 to 3.3, *p* = 0.005)
WVI; Mild dyskaryosis: 0.51 (95% CI 0.28 to 0.93, *p* = 0.035).

Note also that the numbers of "moderate, severe dyskaryosis" in table 3 appear to be interchanged and that the *p*-value in the footnote is incorrect. Thus contrary to the results section, there is a significant association between inflammatory cytology and cervical ectropion and dyskaryosis, as well as

with *Trichomonas vaginalis* and *Chlamydia trachomatis*. The results section of the abstract also states that "there was no association with chlamydia alone" and this too is at odds with the numbers presented in Table 2.

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Dr Dimian and Dr Bradbeer reply:
There are several errors which we would like to correct. These result from the omission, at the request of the referee, of some of the data combined with two typographical errors.

Firstly, in order to explain our reference to dual infection with chlamydia and trichomonas, we would like to add the following table (left out at request of the referee) (table 4).

Table 4 Incidence of trichomonas (TV) and Chlamydia trichomatis (CT)

	Inflammatory <i>n</i> = 101 (%)	Non-inflammatory <i>n</i> = 262 (%)
TV alone	11 (10.9)	15 (5.7)
Chlamydia (CT) alone	11 (10.9)	15 (5.7)
Dual infection	7 (7)*	1 (0.4)

**p* < 0.001.

In addition we would like to correct the results section (para 4) so that it reads "Although the prevalence of cervical ectropion was higher in patients with inflammatory smears, when infections with chlamy-

dia and trichomonas were excluded, this difference was not statistically significant". This should be read in conjunction with the corrected table 3 as follows:

Table 3 Incidence of ectropion and dyskaryosis

	Inflammatory <i>n</i> = 101 (%)	Non-inflammatory <i>n</i> = 262 (%)
Ectropion	43 (42.6)	70 (26.7)*
Ectropion in the absence of TV and CT	31 (30.6)	59 (22.5)
WVI; mild dyskaryosis	15 (14.8)	67 (25.5)*
Moderate, severe dyskaryosis	5 (4.9)	13 (4.9)

**p* < 0.05.

Because of these changes the results section of the abstract should read: "Dual infections with chlamydia and trichomonas were significantly associated with inflammatory changes, but the association with chlamydia alone was not statistically significant. Over 90% of trichomonas infections were detected on cytology. Thus cervical cytology showing inflammatory changes

without trichomonas was not significantly associated with sexually transmitted diseases. Dyskaryosis was negatively associated with inflammatory smears."

We do apologise for any confusion these errors may have caused, and thank Dr Peter Sasiени, of the Imperial Cancer Research Fund Laboratories, for bringing them to our attention.

Fournier's gangrene and HIV: wider issues

The curious publication of two articles by your journal at different times, each claiming to be the first to report Fournier's gangrene in a patient with HIV infection,^{1,2} raises a number of points for debate. First, why publish single case reports simply documenting the occurrence of uncommon conditions in the context of HIV? Why shouldn't Fournier's occur in those with HIV? (It might be more noteworthy and fascinating if it didn't.) What is more

important is whether it occurs with greater frequency in HIV, and whether the natural history is modified. A single case report is insufficient to address these questions. Paradoxically, by questioning whether the Nelson *et al*¹ case actually was a true example of Fournier's, Murphy and Mulcahy³ reduce the force of their own earlier observation: a further report of this rare condition hints that the frequency may be increased in HIV, and should alert clinicians to this.

Although now much discussed, there is still too much pressure on doctors in training to publish. Much greater emphasis is

placed on the number of publications on a CV than on their quality. Inevitably, single case reports and letters to journals are churned out.

This was a small oversight, made worse because both papers appeared in the same journal, and similar errors must be inevitable from time to time even in journals of the highest quality. Nonetheless, a further and more important issue which it provokes is that of quality management of medical journals in general. How could the existence of the earlier Murphy *et al* article have been missed by the later report's authors, referees and section editors?

In nearly all areas of medical practice we are now required to monitor quality, and institute change where necessary. Medical publishing, with its great responsibilities in the areas of medical and clinical education, is not excepted. Peer review is used by tradition to ensure the quality and suitability of articles for publication. Yet in general reviewers are anonymous, unpaid and unaccountable. By using more than one referee, a substandard response from a single reviewer may be identified, but this might be impracticable for small items including letters and single case reports.

Medical journals may need to consider whether managing the quality of their articles now requires a different approach. Has the time come for the names of reviewers to be identified or available on request? Should they be paid, and how should they be accountable?

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- 1 Nelson MR, Cartledge J, Barton SE, Gazzard BG. Fournier's gangrene following hyfreca-tion in a male infected with the human immunodeficiency virus. *Genitourin Med* 1993;68:401-2.
- 2 Murphy M, Buckley M, Corr J, Vinayagamoorthy S, Grainger R, Mulcahy FM. Fournier's gangrene of the scrotum in a patient with AIDS. *Genitourin Med* 1991;67:339-41.
- 3 Murphy M, Mulcahy F. Fournier's gangrene and HIV disease. *Genitourin Med* 1993;69:326-7.

Editors note:
We are grateful to Dr Luzzi for pointing out the flaws in our current peer review system. We are constantly trying to improve the peer review process and have now instituted a process for searching for papers of similar title and content prior to publication.

The quality of referees and their response is continuously monitored and we ask each of our referees to follow a set format when reviewing manuscripts. Unfortunately, from time to time, errors still occur. However, we trust this does not detract from the overall quality of the journal.

Mycoplasmas and non-gonococcal urethritis

Although *Mycoplasma genitalium* was discovered more than 10 years ago,¹ and was noted to have considerable pathogenic potential,² there has been little information about its role in disease because of the great difficulty in culturing it. However, the advent of the polymerase chain reaction (PCR) technique has made detection of *M genitalium* possible and we^{3,4} and Jensen *et al*⁵ have reported recently on its significant association with non-gonococcal urethritis (NGU). It is clear that the strength of the

association is influenced by the way in which the control group is selected and in this regard we question the nature of controls studied by Jensen and colleagues. It appears that none of the asymptomatic control subjects was examined microscopically to determine the existence of urethral polymorphonuclear (PMN) leucocytes. Indeed, men with "asymptomatic" urethritis may have been included in the control group. Few investigators have compared the prevalence of mycoplasmas in men with microscopic urethritis who have no signs or symptoms with that in men without urethritis. However, Swartz *et al*⁶ found that *Chlamydia trachomatis* was isolated more frequently from men with asymptomatic NGU than from those without objective urethritis, suggesting that urogenital pathogens may be involved in the aetiology of this condition. In addition, it is unclear whether Jensen and colleagues examined the asymptomatic subjects clinically at enrolment. Clearly, asymptomatic men with a discharge on examination and objective urethritis (≥ 5 PMN leucocytes/high-power microscopic field) have "clinical urethritis" and should be excluded from the control group and included in the study group. Inclusion of asymptomatic men who have objective urethritis, with or without an observable discharge, in the control group would prevent proper evaluation of negative associations. This may have influenced to some extent the significance given to *M genitalium* by Jensen and colleagues and biased their results against detecting an association of *U urealyticum* with NGU.

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- 1 Tully JG, Taylor-Robinson D, Cole RM, Rose DL. A newly discovered mycoplasma in the human urogenital tract. *Lancet* 1981;i: 1288-91.
- 2 Taylor-Robinson D, Furr PM, Tully JG, Barile MF, Möller BR. Animal models of *Mycoplasma genitalium* urogenital infection. *Isr J Med Sci* 1987;23:561-4.
- 3 Taylor-Robinson D, Gilroy CB, Hay PE. Occurrence of *Mycoplasma genitalium* in different populations and its clinical significance. *Clin Infect Dis* 1993;17(Suppl 1): 66-8.
- 4 Horner PJ, Gilroy CB, Thomas BJ, Naidoo ROM, Taylor-Robinson D. Association of *Mycoplasma genitalium* with acute nongonococcal urethritis. *Lancet* 1993;342: 582-5.
- 5 Jensen JS, Ørsum R, Dohn B, Uldum S, Worm A-M, Lind K. *Mycoplasma genitalium*: a cause of male urethritis? *Genitourin Med* 1993;69:265-9.
- 6 Swartz SL, Kraus SJ, Herrmann KL, Stargel MD, Brown WJ, Allen SD. Diagnosis and etiology of nongonococcal urethritis. *J Infect Dis* 1978;138:445-54.

***Neisseria gonorrhoeae* isolates at St Mary's Hospital, London 1980-91**

Renton *et al*¹ report an increase in isolates of *gonorrhoeae* at St Mary's in 1990—and a similar pattern has been reported nationally—accompanied by speculation that this may be an indicator of varying success in educational programmes in safe sex. We feel that, before elaborate behavioural explanations are pursued, it must be clarified that demographic factors would in any case suggest a peaking of multiple-partner sexual activity around 1989 and 1990 for England & Wales as a whole.

We attach the latest single year popula-

tion estimates for 1991 between the ages 15 and 35 years. The peak in the mid-20s is very marked—and will be still more acute in the revised estimates due shortly. Johnson *et al*² identified clear age differences in reported heterosexual multiple-partner sexual activity—although even their sample size (18 876) did not permit calculation of rates for single ages. Nevertheless, an equivalent peaking in the mid-20s may be inferred. As might be expected, attendance at STD clinics was strongly associated with reported numbers of sexual partners—both homosexual and heterosexual. A coincidence of the two peaks in 1990 might well generate the national pattern observed.

From this perspective, it is the dramatic

fall in isolates throughout the 1980s that requires special explanation in terms of changing sexual practices—while the 1990 peak could be partly the reassertion of a demographic secular pattern, as observed in the 50 years up to 1975.

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- 1 Renton AM, Ison CA, Whitaker L, Kirtland K, Kúpak E, Harné JRW. *Neisseria gonorrhoeae* isolated at St Mary's Hospital, London 1990-91. *Genitourin Med* 1991;69:286-9.
- 2 Johnson AM, Wadsworth J, Wallings K, Bradshaw S, Field J. Sexual lifestyles and HIV risk. *Nature* 1992;360:410-2.

BOOK REVIEWS

The Human Herpes Viruses. Edited by Bernard Roizman, Richard J Whitely and Carlos Lopez. New York, Raven Press. (US \$113.50, pp 447.) 1993. ISBN 0-7817-0024-8.

Over the past 25 years there have been several major advances in human herpes viruses research. Firstly, there has been an appreciation of the diversity of clinical diseases caused by this group of viruses, ranging from the trivial to the life-threatening; and the list is getting longer all the time. Second was the recognition that this group of viruses have evolved a variety of complex mechanisms (many of which are still poorly understood) for persisting in the host, probably for life. Third, there has been an appreciation of the vast and complex diversity of this group of viruses. And finally, there has been the recent introduction of safe and effective drugs to treat some of these infections.

The names Roizman, Whitely and Lopez are inextricably linked with human herpes

viruses and the publication of this volume reflects the editors' research commitments, clinical expertise and above all, enthusiasm for the subject. The volume comprises 16 chapters dealing with basic virology, epidemiology, clinical syndromes, anti-viral therapy, immunity and vaccine development. All 16 chapters are well written and readable, although several chapters, notably the chapter on anti-viral therapy, have a paucity of references (including several key publications) from European journals.

I was a little disappointed with the chapter *The Epidemiology and Clinical Manifestations of Herpes Simplex Virus Infection* which did not provide any information concerning the risk of acquisition of either orolabial or genital herpes from symptomatic and asymptomatic sources. In addition, the overview of HSV seroepidemiology was uncritical and referred only to the type specific assays developed in Atlanta, not even mentioning the Western blot type specific assays developed in Seattle.

The most readable chapters, which bring together a good deal of useful and diverse information, are chapter 2 *Herpes Simplex Viruses and their Replication*, chapter 9 *Human Herpes Viruses 6 and 7*; *Molecular Biology and Clinical Aspects*, chapter 10 *The*