NOTICE OF REDUNDANCY

The hanging committee of Sexually Transmitted Infections wishes to announce that two published papers by van Valkengoed et al. exhibit a degree of overlap. Specifically, the female patients are the same in both papers. They are indistinguishable from the point of population size (5714), age (15–40), setting, participation rate (51%), chlamydia prevalence rate (2.8%; CI: 2.1–3.4%), and the number of women excluded because of never having been sexually active (125). There is also a certain degree of overlap between the two papers in the introduction, methods, results, and discussion sections.


Reply

We strongly disagree with your conclusion that we are guilty of duplicate publication. The objectives, analyses, and results presented in the two papers in Sexually Transmitted Infections are indistinguishable from the point of view of the hanging committee. They are indistinguishable from the point of view of the hanging committee.

Prevalence of Chlamydia trachomatis IgG antibodies in antenatal patients from Trinidad

Editor,—A recent study in Jamaica by Dowe et al using cell culture and a direct fluorescence antibody assay (DFA) showed a prevalence of Chlamydia trachomatis infection in 47% of gynaecological patients. Unfortunately, there are no comparable data for cell culture and DFA in Trinidad. Moreover, we cannot find any reports on serological studies for C trachomatis IgG antibody in the West Indies. In an attempt to shed more light on the prevalence of C trachomatis IgG antibody in pregnant women in Trinidad, we collected 56 serum specimens (mean age of patients 27 years) with ethics committee approval from one clinic at the general hospital, Port of Spain. As well as testing these sera by an in-house ELISA method based on that described by Ossewaarde et al., we also used a commercial ELISA test specific for C trachomatis IgG (Savyon Diagnostics, Israel) and the whole inclusion immunofluorescence (WHIF) test as previously described by Richmond and Cail.

All collected sera were stored at −70°C until analysis. Samples were subsequently coded and tested blind in duplicate in laboratories in Sheffield and Bristol. Details of the in-house ELISA test methodology and interpretation of readings using microimmunofluorescence (MIF) serum positive and negative controls were described in Keay et al. The commercial ELISA was performed according to the manufacturer’s instructions. The WHIF test consisted of chlamydial inclusions of infected mammalian cells with LGV2 mounted on a glass slide or coverslip. The WHIF titre is described as the highest dilution of antibody where the inclusion can be clearly seen by fluorescence staining.

For the ELISA tests, results were recorded as positive, equine, negative, or equivocal. For the WHIF test, titres between 1:64 and 1:256 were recorded as such; a low titre was ≤1:64 and a high titre ≥1:512. Twenty-five (45%) and 29 (52%) samples were positive for the commercial and in-house ELISA tests respectively. Eighteen (32%) samples had a titre of ≥512 in the WHIF test, as shown in table 1.

The latter finding is of note. It is accepted that C trachomatis is an established pelvic pathogen and in a recent study of 34 women positive for C trachomatis IgG (≥1:128) by ELISA, at laparoscopy 31 (91.2%) were diagnosed as having tubal disease. It is likely that significant damage could be occurring in these patients as a previous study looking at high C trachomatis IgG titres showed 46% positive and 8% positive in infertile women with damaged and normal tubes, respectively.

Although these findings are based on relatively small numbers, they are of significant concern if combined with the other recent study. It would appear that the prevalence rate for C trachomatis may well be high and that data presented here suggest possible future PID development and resultant sequelae. It is clear that further studies are warranted and that screening and treatment strategies may be required urgently to curtail considerable morbidity in Trinidad and throughout the West Indies in general.

Table 1 Comparison of ELISA and WHIF tests showing the Chlamydia trachomatis IgG antibody titre distribution

<table>
<thead>
<tr>
<th>WHIF test</th>
<th>Commercial ELISA</th>
<th>In-house ELISA</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥512</td>
<td>15</td>
<td>−</td>
</tr>
<tr>
<td>256</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>128</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>64</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>≤64</td>
<td>1</td>
<td>1</td>
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</tbody>
</table>

Eq = equivocal.

Financial support was provided by the University of Sheffield and Bristol Public Health Laboratory.

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Implications of random measurement error in studies adjusting for sexual behaviours

Editor,—In their recent review of methodological issues in sexual behaviour research, Fenton et al provide a comprehensive overview of the major types of sexual behaviour research, the sources of measurement error which may affect such research, and different approaches to measuring various types of sexual behaviour. In their article, they describe by Fenton et al is widely recognised. Given their impact on inferences of association, great care is taken in most studies to avoid these biases. The effects of random measurement error on non-differential misclassification of a dichotomous covariate may increase the risk of mistaking exposure for outcome, and/or outcome measures will lead to an attenuation of the resulting measure of association. However, the fact that random measurement error in potential confounding variables may increase the risk on the inferences which are made from study results is seldom acknowledged. The non-differential misclassification of a dichotomous confounding variable may lead to inaccurate statistical adjustment (often referred to as residual confounding) and the false appearance of statistical interaction when none is present. When confounders are measured as polytomous or continuous variables, conditional on other confounders, random measurement error can bias the adjusted measure of association less accurately than the crude. These forms of misclassification are generally of greatest concern when the true exposure-disease association is relatively weak compared with the exposure-confounder and outcome-confounder relation, as is the case in most research around STIs. Even small random errors can have major effects on adjusted measures of association, and the uncorrectability of the effects of measurement error in confounders may be compounded in multivariate analyses.

With this in mind, Fenton et al’s review of the difficulties involved in the accurate measurement of sexual behaviour has powerful implications for studies attempting to control for covariates associated with risk for STIs. Studies which attempt to adjust during statistical analysis for numbers and types of sexual partners, frequency of sexual contacts, or condom use practices, are likely to encounter some degree of random measurement error. Although these errors may be differential with respect to exposure or outcome, this mismeasurement may lead to uninterpretable biases and/or mis-specified analyses, and in turn, spurious inferences.

In summary, the random measurement of sexual behaviour requires special consideration in any study attempting to adjust for the confounding role of sexual behaviours in associations involving STIs. We hope that Fenton et al’s review of the challenges posed by the collection of sexual behaviour data helps to draw attention to this frequently overlooked methodological aspect of the epidemiology of STIs.

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Is Mycoplasma hominis a vaginal pathogen?

Editor,—We would like to comment on the study by Arya and colleagues 1 which failed to find evidence for Mycoplasma hominis being pathogenic in the vagina, or otherwise contributing to bacterial vaginosis (BV). They mentioned the 21 year old view of Taylor-Robinson and McCormack2 who surmised that M hominis might act in symbiosis with other organisms or as a sole pathogen in BV. The latter was referred to as non-specific vaginitis or Gardnerella associated vaginitis and the term BV was being used from about 1984. Since then, much has been learned about the vaginal microflora in health and disease, but the question of which bacteria, if indeed any, cause BV remains unanswered. Few M hominis organisms in the healthy vagina appear to behave as commensals. We challenged the suggestion of Mårdh and colleagues that M hominis was associated with a range of genital signs and symptoms and BV had been excluded, our assertion being that M hominis organisms outside the context of BV would be present in small numbers and, therefore, unlikely to cause a problem. In contrast, the few M hominis organisms in the healthy vagina increase in number, perhaps by 10 000-fold or more, in women of women with BV. This increase, however, occurs only late in the development of BV: Indeed, it is rare to find large numbers in the “intermediate” (grade 2) stage between the normal vaginal flora and “full-blown” BV (grade 3). Thus, in the study by Arya and colleagues we have difficulty in understanding why only 35 (48%) of the 73 women with M hominis positive BV had large numbers of organisms (>104). A Gram stain evaluation should have distinguished women with grade 2 flora from those with grade 3. Be this as it may, the authors contend that because the additional presence of M hominis with G vaginalis and strict anaerobes did not seem to increase the likelihood of the women developing BV, M hominis is not involved. It is clear that M hominis organisms are not essential for the development of BV and unlikely that their initial presence in the vagina increases the likelihood of BV developing. However, if they are present in the vagina initially, then they will multiply as indicated and large numbers will ensue. The data of Arya and colleagues do not resolve the issue of whether large numbers contribute to the disease process or are involved in its persistence. Against this, as they point out, is a study 3 in which metronidazole, inactive in vitro against M hominis, cleared vaginal infections. However, if they are present in large numbers to volunteers, indicating the pathogenic potential of the organisms. Furthermore, the M hominis species is heterogeneous, some strains having greater epithelial cell adherence properties than others. We do not see any data that point to M hominis being a sole pathogen or co-pathogen in the vagina but, equally, we are not convinced by data that purport to show that it is not.

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Is Mycoplasma hominis a vaginal pathogen?


predominant renal lesion in black patients, whereas immune complex and membranous nephropathy occur more commonly in white patients. Improvements in renal function have been described with highly active antiretroviral therapy (HAART) when the underlying renal lesion is HIVAN or membranous nephropathy. We report here an HIV infected patient in whom renal disease caused by hepatitis B induced membranoproliferative glomerulonephritis improved with HAART.

A 34 year old white homosexual man was found to be HIV-1 antibody positive in August 2000 after he presented with biopsy proven Kaposis sarcoma. At this time he also reported 2 months of fatigue and frothy urine. In the past he had been found to be hepatitis BeAg positive in 1996. Examination revealed multiple cutaneous Kaposis sarcoma, BP = 170/100, no peripheral oedema, and scanty retinal haemorrhages on funduscopy. Investigations showed blood urea = 9.2 (normal = 2.8–7.6) mmol/l, serum creatinine = 178 (normal = 80–133) µmol/l, normal serum, potassium, and sodium. Liver function tests were normal apart from a serum albumin of 29 (normal = 35–50) g/l. The haemoglobin was 9.3 g/dl and white blood cell and platelet counts were normal. The C4 count was 110 cells × 10^9/l and HIV viral load was 47 500 copies/ml. Complement C3 = 0.56 (normal = 0.9–1.8) g/l, C4 was 0.07 (normal = 0.1—0.4) g/l. Immunoglobulin quantification showed normal IgA, IgG = 23.2 (normal = 7.0–16.0) mg/l and IgM = 4.4 (normal = 0.4—2.3) g/l. Hepatitis B serology showed HBeAg and HbsAg* (titre 1:3200). Urinalysis showed blood ++++ and ++++ protein. Urine protein was 5.8 g/24 hours and creatinine clearance = 66 ml/min. Ultrasound examination showed normal sized kidneys. Histology of a renal biopsy showed membranoproliferative glomerulonephritis. Staining showed marked deposition of hepatitis B core and surface antigens (fig 1).

The patient was managed conservatively. HAART was commenced with efavirenz, didanosine, stavudine and hypertension was treated with ramipril. After 8 weeks of HAART the CD4 count was 140 cells × 10^9/l and viral load was 100 copies/ml. The serum creatinine returned to normal and there was no persistent proteinuria.

Cervical cytology smears in sexually transmitted infection clinics in the United Kingdom

Editor,—I found the article by Janet Wilson and Wendy Parsons on behalf of the BCCG, concerning cervical cytology practice in UK genitourinary medicine clinics, comprehensively and reasonably within the limits of our practice.

The final statement “there is therefore no evidence from this study to support the practice of additional smears in the presence of an effective national cytology screening programme” is both justified and a case well made.

The paper calls additional smears “opportunistic” and recognises them as being performed in women less than the age of 20, women with genital warts, and in some who have had a normal smear within the previous 3 years.

The OED definition of opportunity is “favourable, appropriate or advantageous combination of circumstances.”

There is no evidence to suggest that offering smears in these circumstances fulfils this description. If this is so, then we are depriving women such as teenagers of a valuable health screen and patently this is not the case.

I would like to propose, therefore, that we no longer continue to call these smears “opportunistic” but use the term “unnecessary.”

The recognition of this could be an advance for evidence based practice, help to reduce unnecessary anxiety, and release much needed resources.

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BOOK REVIEW


This is a profound work describing the impact of venereal diseases and conventional morality in the build up to AIDS. It is written by an American, who has been personally affected by the impact of AIDS. He has written this book on topics in a history of sex, sickness, morality, and infectious diseases, which have had an impact on the public response to AIDS. Throughout, one senses the author’s very real loss in what to him and many others have been tragic times.

It is interesting to see how different the general public moral climate is in different societies in the developed world. Thankfully, some forms of evangelism do not have the same influence everywhere.

Does the historical part of the book tell the medical historian anything new? The answer is yes. And that is the gap between what has been known on this subject to academics for a long time and what others are only finding out about now. The chapters containing information on the church’s attitude to sexual morality, on leprosy, the early history of syphilis, bubonic plague, and masturbation illustrate the age old story of reactionary view against progress. It is difficult to judge the mores of the past through the views of the present.

It is a pity that the author seems to have given such prominence to those whose views resisted progress. Nothing is mentioned of liberal pioneers in venereal diseases from Van Swieten in the 18th century, through Ricord, Fournier in the next, Abraham Flexner (for the Rockefeller Foundation), Neisser, or indeed the enormous changes brought about by the Royal Commission on Venereal Diseases in Great Britain at the time of the first world war or such notable more recent Americans such as Kampmeier, Stokes, or Earl Moore.

The chapters on America are particularly interesting from a European point of view. Learning about reactionary views always helps in developing any strategy for public knowledge and education. Well educated AIDS lobbyists have certainly had an impact in Europe as in the United States and are dearly noted in this work. The bibliography, 14 pages, is particularly good.

This is a book questioning responses and conventional morality in respect, sorrow, and anguish. It is worthy of merit. It enables the modern reader to learn about difficult aspects of morality in relation to venereal diseases and sexuality which have always had more impact on the public than the practising physician.

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Mechanics for Africa—training school in Africa to bring health awareness into curriculum

Mechanics for Africa is a training school for motor mechanics in Zambia, giving young Africans skills for life. This charitable initiative was recently launched to help impoverished Africans break out of the persistent reliance on outside aid to help them become self-reliant. Mechanics for Africa (MFA) will set up a school for motor mechanics in Ndola, Zambia, in association with Malford Baptist Church in Surrey. MFA (charity registration No 1086333) was launched recently by its patron, the Rt Hon Virginia Bottomley, MP.

Part of the holistic curriculum will help introduce the need for health awareness among ordinary local people, particularly with HIV/AIDS and other STDs, diarrhoea and resultant dehydration. Other topics to be covered will include malaria (still the biggest killer, especially among children), nutrition, first aid, hygiene, etc. All this to be part of a balanced “life skills” curriculum which will empower students to improve their lives; and those of their families by inviting them to participate in these studies.

The project asks companies, organisations, and individuals to become financially involved at various levels either through donation, tax reclaimable gifts, or loans.

MFA is the brainchild of Charles and Sharonne Watt who have worked on aid projects in southern Africa for 3 years and had the opportunity to evaluate the needs of local and wider communities.

Apart from operating the school, MFA has plans to establish a workshop and training centre for mechanics on the outskirts of Ndola (population 500,000) in the heart of Zambia’s copperbelt.

The MFA project is designed to eventually become self-funding with a commercial service for vehicle maintenance planned to operate independently and in tandem with the charity.

Mechanics for Africa is actively seeking funds and donations. Further details: Mechanics for Africa, Bridian Farm, Alford, Surrey GU6 8HR, UK (tel: 01403 752 384; fax: 01483 421 271; email: charlie@bigfoot.com; web: www.mfazambia.com).
Prevalence of *Chlamydia trachomatis* IgG antibodies in antenatal patients from Trinidad

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*Sex Transm Infect* 2001 77: 301-302
doi: 10.1136/sti.77.4.301-a

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