Correspondence

I realise that reading the text reveals that the figure is 18. Because summaries are used as abstracts by many journals, however, and consequently read by a larger number of people than would read the complete article, then a false impression will be given. Thirty five is twice the figure found in the experiment or 100% instead of 50%!

As Dr McMillan and his co-authors are using information and figures directly related to my own research, I feel I must ask that they are correctly reported.

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Reference


Reply

SIR,—We thank Mr Sargeaunt for his comments. Regardless of what zymodeme types he considers ‘pathogenic’, Mr Sargeaunt considers type I amoebae to be non-pathogenic. The statement that ‘the vast majority of homosexuals are passing only non-pathogenic amoebae’ is gravely misleading; neither clinical, histological, nor serological data were presented to substantiate this assertion. This view has been challenged in our paper: 83% of the 18 individuals excreting type I amoebae showed histological evidence of proctitis (grades B and C) which resolved after anti-amoebic treatment. Since the completion of the reported study, a further 38 homosexual men with amoebiasis have been identified. Entamoeba histolytica was the sole pathogen or potential pathogen detected in 31 of these men; 24 (77.4%) had proctitis (19 grade B; five grade C). Treatment with diloxanide furoate only was associated with resolution of the proctitis; when therapy was delayed, the proctitis persisted.

When the pathogenicity of an organism is considered, host factors cannot be ignored. As a result of careful contact tracing, we found that eight asymptomatic cyst excreters (three of whom had been infected with amoebae of zymodeme type I) had transmitted sexually the infection to others who subsequently developed symptomatic amoebiasis.

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Amoebiasis in homosexual men

SIR,—I wish to bring to your notice errors that appear in the article by Dr A McMillan et al (Gut 1984; 25: 356–60). These authors say: ‘Based on their association with dysentery or hepatic abscess formation these workers consider that only amoebae of zymodeme types II, VI, VII, and XI are pathogenic’. This statement is wrong, the authors to whom Dr McMillan is referring quite clearly state: ‘The accompanying figure shows the isoenzyme patterns for all the zymodemes so far demonstrated. Many of these zymodemes have been demonstrated in E histolytica from various areas of the world. The marker for pathogenicity is the presence of a β band and absence of an α band in PGM. This feature is confirmed, with the exception of zymodeme XIII by the advanced bands in HK’.

The accompanying figure referred to above is shown quite plainly to show that not only are zymodemes II, VI, VII, and XI pathogenic but also XII, XIII, and XIV. This mistake is inexcusable because it may lead the reader to a false impression of pathogenic amoebic infections. It is difficult to understand how such a gross error has occurred, particularly when it is known that Dr McMillan and his co-authors are aware of another published article, Entamoeba histolytica in male homosexuals, in which all 18 zymodemes were again clearly figured.

The authors write that ‘Isolates from 18 men were available for isoenzyme characterisation. Each was of zymodeme type I’. They do not relate, however, the zymodeme to any clinical, histopathological, or serological feature. In that case, what value is it then to know the zymodeme?

Leaving the above points apart I must protest in the strongest fashion possible about the summary which contains a thoroughly ambiguous statement. This quite clearly gives the impression that 35 homosexual men were infected with Entamoeba histolytica expressing zymodeme I. Surely this is not true!

Amoebiasis

References


