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HIV infection in a cohort of homosexual and bisexual men

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Epidemiological studies in the United States report rises in unprotected anal intercourse among gay and bisexual men,[1] after dramatic reductions in the mid-1980s there[2] and in the United Kingdom.[3] In 1991 rising rates of rectal gonorrhoea among homosexual men attending genitourinary medicine clinics[4] led to speculation about increasing HIV risk behaviour in the United Kingdom.

This speculation is complicated by the bias in samples of clinic attenders towards more sexually active men but may be clarified by data from samples of non-attenders.

Subjects, methods, and results

In 1987-8 we interviewed a cohort of 930 homosexual and bisexual men in 10 cities in England and Wales who had been recruited from sources other than genitourinary medicine clinics. Subsequently 77%, 65%, and 50% were re-interviewed annually. All men were interviewed about current and past sexual behaviour. Respondents in London and South Wales were asked, though not required, to provide a blood sample for testing for HIV-1 antibody; for each year 344, 290, 240, and 296, about two thirds of those eligible, did so. The results, with counselling, were available to the men at their specific request. They were linked with behavioural data only for analysis. Ethical committee approval was obtained for all localities at each stage of the study.

The median age of the original cohort was 29 (range 15 to 81) years. Full details are given elsewhere.[5] The proportion of regular clinic attenders was 31% in 1987-8 and did not vary significantly. The proportion tested before the study began was 41%, rising in 1991 to 54% of the men interviewed.

The proportion of samples positive for HIV-1 antibody was significantly higher in the clinic attenders than non-attenders (15·6% *v* 3·8%; $\chi^2=11·35$, $p=0·0018$).

Unprotected anal intercourse was more common in men in regular than in casual relationships (19% *v* 4%). The mean annual number of partners with whom anal intercourse occurred increased from 2·0 in 1987-8 to 2·7 in 1990-1, and the number of partners increased from 12·3 to 17·9 ($t=2·53$, $p=0·011$). Meanwhile, the proportion reporting oro-anal contact in the month before interview rose from 31% to 41% and reporting digital-anal contact from 42% to 57%.

Seventy three men in the cohort were antibody positive, some of whom subsequently died; 13 do not know their HIV status. Eleven (15% of all positive) men who were antibody negative in 1987-8 subsequently tested positive. The mean period between the positive result and the last negative result was 11 (range 2 to 24) months. In 10 men we could identify the year of seroconversion: our best estimates are 1987, one man; 1988, two; 1989, two; 1990, three; and 1991, two.

All 11 men reported unprotected anal intercourse and a range of other sexual acts before seroconversion. In five unprotected receptive anal intercourse was the probable mode of transmission. In two men unprotected insertive intercourse was the likely mode, but both had also practised receptive fellatio with orgasm, one with a partner known to be antibody positive and one had also engaged in insertive fisting. In the four other men both unprotected receptive and insertive anal intercourse had occurred.

In eight men, the source of infection was clearly identified as a regular partner four of whom were known to be antibody positive at the time. In only one man was the source clearly traced to a casual partner. Three of the seroconversions were not in regular clinic attenders, nor had the men attended in the year before interview. Four men had not previously been tested.

Comment

Although the small numbers make conclusive claims hazardous, these data do not substantiate a large increase in unprotected anal intercourse. One reason may be rises in oro-anal and anal-digital contact which impugn the validity of rectal gonorrhoea as a surrogate marker for HIV risk behaviour.

Almost a fifth of men who tested antibody positive in this study were not clinic attenders. Just under a sixth of positive men became infected after the government campaigns began in 1986-7. Health promotion for gay and bisexual men remains a priority, and initiatives which concentrate on HIV transmission within relationships should be encouraged.

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Relation of serum sialic acid to lipid concentrations

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Sialic acid, a constituent of plasma membrane, is present in most acute phase reactant proteins. A recent epidemiological study showed that mortality from cardiovascular diseases was higher in a population with high concentrations of serum sialic acid.[1] The increase is suspected to reflect the existence or activity of the atherosclerosis process. We investigated the relation of serum sialic acid to serum lipids, which strongly influence the occurrence and advance of atherosclerosis.

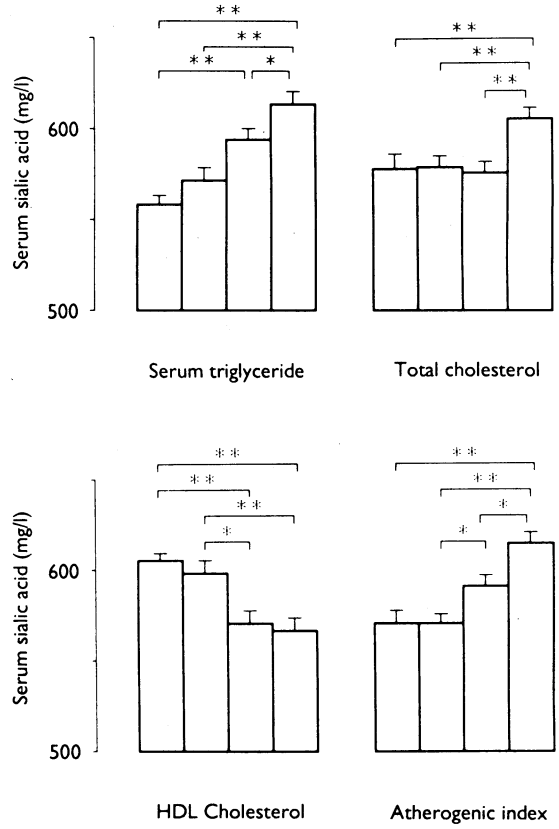
Subjects, methods, and results

Subjects were 382 men (35-54 years old) participating in a periodic medical health examination at their workplace; the response rate was 98%. Blood was sampled between 9 am and noon after subjects had fasted overnight. Serum lipids (total cholesterol, high density lipoprotein (HDL) cholesterol, and triglyceride) were analysed on an automatic analyser. Serum sialic acid was measured by the enzymatic method.[2] An atherogenic index was calculated as:

$$\text{atherogenic index} = \frac{(\text{total cholesterol} - \text{HDL cholesterol})}{\text{HDL cholesterol}}$$

Data are expressed as means±(SE). Student's *t* test was used to compare group means. The values for each serum lipid variable were arranged in ascending order and then the subjects were divided into four groups of approximately equal size. As serum sialic acid concentration is known to increase with age,[1] multiple regression analysis was performed with serum sialic acid as a target coefficient and the four items of age, serum triglyceride, total cholesterol and HDL cholesterol as explanation coefficients. As the atherogenic index is strongly affected by serum total and HDL cholesterol values, it was not included as an explanation coefficient of the multiple regression analysis. p Values $<0{\cdot}05$ were defined as significant.

Mean concentrations of serum sialic acid were compared among the fourths of each serum lipid variable (triglyceride, total cholesterol, HDL cholesterol, and atherogenic index). Mean serum sialic acid concentrations in the highest fourth of serum triglyceride, total cholesterol, and atherogenic index were significantly higher than in the other three divisions, and sialic acid concentrations were higher in each fourth of serum triglyceride and atherogenic index. For HDL cholesterol, however, sialic acid concentrations of the upper two fourths of HDL cholesterol were significantly lower than those of the lower two fourths, and the mean values of serum sialic acid were lower in each higher quartile of HDL cholesterol (figure). Standardised multiple regression coefficients between serum sialic acid and age, HDL cholesterol, triglyceride, and total cholesterol were 0·100 ($p<0{\cdot}05$), −0·146 ($p<0{\cdot}01$), 0·137 ($p<0{\cdot}05$), and 0·030 ($p>0{\cdot}05$), respectively.



*Serum sialic acid concentrations related to serum lipid variables, in fourths. Bars indicate SE; *=p<0·05, **=p<0·01*

Comment

A slight but significant positive correlation between serum total cholesterol and sialic acid in a large population and a strong positive correlation between serum triglyceride and sialic acid in patients with diabetes mellitus have been reported.[1 3] In our study, serum sialic acid concentration was significantly higher