

Xg groups and sex chromosome abnormalities in people of northern European ancestry: an addendum

A catalogue of the Xg groups of 1547 patients with various abnormalities of number or of form of their sex chromosomes, together with the groups of many of their parents, appeared in this journal in 1971 (Sanger *et al.*, 1971a). All the Xg tests were done in the MRC Blood Group Unit and covered the time from the recognition of the Xg groups (Mann *et al.*, 1962) to January 1971. The results were given in 8 tables.

The purpose of the present communication is to record that the count was brought up to October 1975 by the addition of 536 more patients and that the revised 8 tables are available on request.

The tables give the totals and analysis of the Xg groups of the following classes of propositi and their available parents: Table 1, 739 propositi with Klinefelter's syndrome; Table 2, parents of 566 XXY propositi; Table 3, 56 XX male propositi and their parents; Table 4, 975 propositi with Turner's syndrome; Table 5, parents of 610 of the Turner propositi; Table 6, 313 propositi with other kinds of sex chromosome abnormalities; Table 7 parents of 29 females with one X lacking a long arm; Table 8, normality of the Xg distribution in both parents of 647 assorted propositi.

Here follow only a few notes prompted by the increased numbers.

The Xg phenotype frequencies for normal northern Europeans are: for males Xg(a+) 0.659 and Xg(a-) 0.341; and for females Xg(a+) 0.884 and Xg(a-) 0.116 (Sanger *et al.*, 1971b).

Klinefelter's syndrome

XXY

The new total is 566 and the proportion Xg(a+) is 0.848, exactly as in the 1971 total of 395. Analysis by Fraser's method (1963) of the families in which both parents were grouped allowed the following estimate of the sources of the extra X: $X^M X^P Y$ 0.33; $X_G^{M1} X_G^{M2} Y$ 0.47; $X_G^{M1} X_G^{M1} Y$ and $X_G^{M2} X_G^{M2} Y$ 0.20. (Where both Xs are maternal it is the Xg locus rather

than the whole X which is being tracked and in this case, on the advice of Professor J. H. Edwards, Xg is written for X.)

XXY MOSAICS

The previous slight suggestion of an ultra-female distribution of Xg(a+) has disappeared and is now 0.903, close to that expected of females.

XX males

The number of propositi tested has increased from 34 to 56 and their Xg distribution now fits that of XXY rather better than XY and differs significantly from that of XX females. The Xg groups of the parents were informative about the origin of the Xs in 87 propositi: in 7 both Xs were maternal and in the eighth, one X was paternal. These results can be interpreted as support for the suggestion by de la Chapelle *et al.* (1964) that XX males arise from XXY zygotes, with subsequent loss of the Y at any rate from tissues accessible to cytogenetic analysis.

Turner's syndrome

XO

Total 424, proportion Xg(a+) 0.700 (at the 1971 total of 326 the proportion was 0.696). Both parents of 306 XO females were grouped and allowed the following estimates of the origin of the sole X: $X^M O$ 0.77 and $X^P O$ 0.23 (previously 0.78 and 0.22).

The XO/XY class of whom 34 have been grouped and 28 found to be Xg(a+) is 6 times more likely to represent a female than a male distribution; this is surprising and at present difficult to explain other than by chance of sampling.

XXX females

The previous hint that such people have an ultra-female distribution of Xg(a+) persists now when the number of propositi has doubled. Of the 80 propositi, 76 were Xg(a+), an incidence of 0.950. In 80 normal females 70.72 would be expected to be Xg(a+); for the comparison χ_1^2 is only 3.40. But should the deviation become significant it would suggest non-disjunction at the first meiotic division of oogenesis as the usual cause.

Testicular feminization syndrome, XY females

The number of propositi has increased from 66 to 79 and the distribution of the Xg groups is more ultra-male than before, 43 being Xg(a+) whereas the expected number of Xg(a+) in 79 normal males is 52.06. For this comparison $\chi^2 = 4.63$ and $P = 1$ in 33. If this deviation persists an explanation may be hard to find.

Females lacking an arm of one X

The evidence that the Xg locus when carried on a normal X is not inactivated is very strong (Ducos *et al.*, 1971; Race and Sanger, 1975). The evidence that Xg is inactivated when carried on an abnormal X (Polani *et al.*, 1970) is also strong and is greatly increased by the recent additions.

Propositi with short arm deletions or long arm isochromosomes (excluding mosaics with an XO cell line) have increased from 44 to 67, of whom 42 were Xg(a+). Long arm deletions or short arm isochromosomes (again excluding mosaics) have increased from 10 to 20, of whom 13 were Xg(a+). Both distributions are those expected of males with a single X and depart significantly from those expected of normal females: the figures for the missing short arm class are over 5 million times more like those expected of males than females, and for the long arm class 42 times more like the male distribution.

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Association of ABO blood groups and vitiligo

SUMMARY ABO blood group frequencies of 1000 vitiligo patients were studied and compared with those of blood donors and local population. The relative risk of O was significantly reduced in patients in comparison with blood donors but not with local population. This can possibly be explained by the well-known preference of O group donors in the blood bank. On a review of other studies it was felt that there may not be any real association of ABO blood groups with vitiligo.

In the past two decades much work has been done to elucidate the factors that cause vitiligo, but little is known about the genetic and hereditary factors involved in the incidence of this disease. Among the important genetic factors that have been studied are the ABO blood group antigens. The results of these studies on the association of ABO blood groups and vitiligo have, however, been conflicting. A greater incidence of AB blood group was found by El-Hefnawi *et al.* (1953) in 80 vitiligo patients as compared to their controls. Singh and Shanker (1966), in a study of 100 vitiligo patients, also observed a higher incidence of vitiligo in people with blood group AB. Srivastava and Shukla (1965) found an increased 'B' gene frequency in a study of 535 vitiligo patients, whereas Sehgal and Dube (1968) reported a decreased frequency of O group in 173 patients of vitiligo.

The varying sample sizes involved in these studies may possibly be responsible for such conflicting results. The present study, therefore, has been undertaken on a large sample of vitiligo patients to see if there is any real association of ABO blood groups and vitiligo and also to examine the nature of heterogeneity between various studies.

Subjects and methods

One thousand unrelated vitiligo patients of the Hyderabad city attending the outpatient clinic of the