needed before confident conclusions can be reached concerning the genetic nature of the Y-chromosome.

A tetraploid cell was noted in a 'brain' (supraesophageal ganglion) preparation of *WHO*, the first reported for *M. domestica* (figure 3). Such cells are well-known in other insects (e.g. polyploidy occurs readily in hymenoptera[7]) and aneuploidy is known for both sex chromosomes and autosomes in the housefly[3]. We doubt that the colchicine-hypotonic treatment preceding the air-drying step could have spuriously produced this cell, as this treatment was only applied for 30 min.

G-banding, as described here, should prove a useful tool in correlating the genetic and cytological maps of the housefly. Furthermore, our discovery of bands on the X-chromosome should reorientate thinking on the mapping of new mutants, and the possibility of sex differences in insecticide resitance. Although sex-linked markers seem rare among visible mutants[3,8], this might not be so in the case of allozymic loci.

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Recombination in the male housefly, *Musca domestica*

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Summary. Autosome II recombinations of up to 30.7% were found in male houseflies bearing a Y-chromosome translocation on autosome II. This is the highest recorded rate of recombination found in male houseflies, therefore indicating another possible source of genetic variation. The male crossover rates were related to map distances and resulted in an inaccurate estimate for one of the loci. Explanations are provided. Male crossing-over is related to cytogenetics of the Y-chromosome.

The housefly has a high degree or genetic variability which contributes to its great adaptability to new environments[2]. Recombination plays a major role in genetic variability of most organisms, but, as is the case in *Drosophila*, the housefly male has exhibited a virtual lack of crossing-over thus reducing the potential of recombination as a factor producing variability. Autosome III of the housefly male (there are 5 autosome pairs, 2n = 12) has been shown to have crossover values of 0 to 11% between the loci *bwb* (brown body) and *ge* (green eyes). These 2 loci are between 20.3 and 28.2 map units apart[3]. Crossing-over is considered fairly common for this autosome in females, with recombination frequencies of up to 40% for some loci. A very low frequency of recombinants (0.2%) has been found for autosome II in males, but actual recombination values found for the male houseflies may have been due to genetic mechanisms other than crossing-over, for example, nondisjunction[4].

In a recent examination of a Y-autosome translocation strain (identified by genetic and cytogenetic techniques[5]) from Kingsford, Australia, exhibiting holandric inheritance, recombination rates of up to 30.7% were found in males of this strain when crossed with multimarker stock females. Only a recombination involving autosome II was identified as it was this autosome which had 2 markers, the visible mutant marker or the Y-translocation. All recombinants were found in the F_2-generations.

The procedure was as follows: Kingsford *(KIN)* males were crossed with virgin females from a multimarker stock *608Q*, in which 4 autosomes were marked, each with one visible recessive gene (*ac* = alicurve, chromosome I; *ar* = aristapedia, chromosome II; *bwb* = brown body colour, chromosome III; and *ocra* = ocra eye colour; chromosome V)[6]. The F_1 male progeny (phenotypically wild type but heterozygous for the marker loci) were then backcrossed to virgin females of the homozygous multimarker stock. The experiment was repeated using a 2-marker stock, *WTIN* (*stw* = stubble wings, chromosome II; *w* = white-eye colour, chromosome III). The cross involving *608Q* showed 30.7% recombination between the aristapedia locus and the Y-chromosome translocation (table 1). The cross involving *WTIN* exhibited 9.27% recombination between the stubby wing locus and the Y-translocation (table 2). If these recombination rates are converted to map distances, the stubble wing and the aristapedia loci are 21.43 map units apart, which is a similar result to that obtained in previous studies on crossover suppressors[2]. The stubble wing locus is 9.27 map units from the point of attachment of the Y-chromosome fragment. The inconsistency of this distance with the map distances between various other markers on the same chromosome arm[2] may be explained by the loss of the segment of autosome II during the process of the reciprocal translocation with the Y-chromosome[5].

An explanation for the high rate of recombination in the *KIN* male is that the segment of the Y-chromosome lost

Table 1. Recombination in the cross, *608Q* × *608Q/KIN*

	Males	Females
Recombinant groups	*ar*	+
Number of recombinants	5	465
Total number of flies	1531	
Percent recombination	30.7	

Table 2. Recombination in the cross, *WTIN* × *WTIN/KIN*

	Males	Females
Recombination groups	*stw, wstw*	+, *w*
Number of recombinants	1	362
Total number of flies	3913	
Percent recombination	9.27	

during the reciprocal translocational may have carried a locus modifying recombination, thus the intact Y will prevent crossing-over. This may be further verified by the presence of a banding pattern found on the Y-chromosome of a wild-type strain with a normal XY complement in the male[8].

Sullivan[4] indicated that recombination, in test crosses, of heterozygous males may be due to factors (nondisjunction) other than crossing-over. Hiroyoshi[7] agrees with Sullivan[4], while Tsukamoto considers that crossing-over does not occur in the male housefly. The high recombination frequencies found in the test crosses involving the heterozygous *KIN* males are most readily explained as being due to crossing-over. Furthermore, these high values, plus the fact that autosome II was the autosome exhibiting recombination in the male, indicate that a reevaluation of recombination in male houseflies is required, involving a more detailed study of the occurrence of recombination for autosomes I, IV and V. It now seems inadequate to base the system of recombination in male houseflies on the model of the *Drosophila* male.

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Chromosome aberrations in in vitro irradiated lymphocytes from human cord blood

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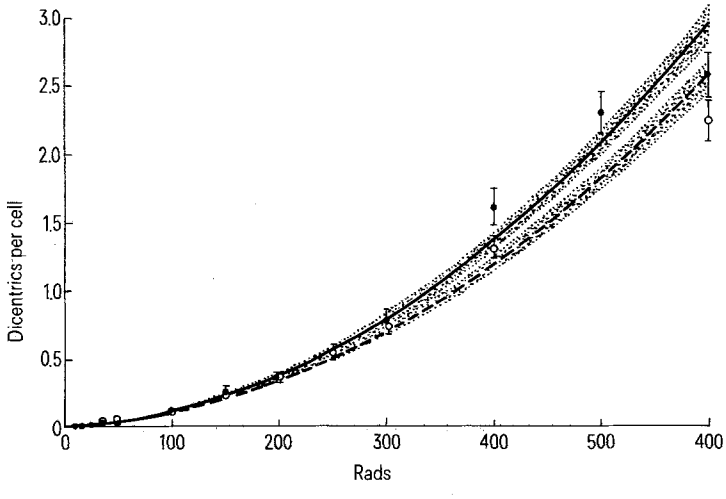
Summary. An in vitro dose effect curve of dicentric chromosome aberrations in human cord blood lymphocytes has been obtained for 250-kV X-rays. This is compared with a curve prepared in an identical manner using blood from adults. The comparison shows a marginally higher dicentric yield in blood of newborns at doses above about 250 rads.

There is a suggestion in the literature that when a peripheral blood sample from a child (< 1 year) is irradiated in vitro the induced yield of dicentric chromosome aberrations is significantly higher than when the same dose is given to adult blood. The evidence is somewhat contradictory and comes from 3 studies[2-4] of interdonor variability using samples from people ranging in age from newborns (cord blood) to 100 years. Each investigation used a single radiation dose and none examined the dose effect relationship for young children's blood in more detail.

In the present paper a dose response curve for dicentric chromosome aberrations is presented for cord blood and is compared with a curve previously produced in this laboratory using adult's blood.

Materials and methods. Blood samples from the umbilical cords of 12 healthy babies were received into 10 ml sterile heparin tubes (Stayne) in which they were exposed to X-radiation. The samples were taken following normal pregnancies in which no drugs or radiation were received by the mothers and the births were full term and spontaneous. The blood was irradiated at 37 °C with various doses (see table 1) of 250-kV X-rays HVL 1.2 mm copper, at a dose rate of 100 rad per min. After irradiation the lymphocytes were separated and cultured for 48 h in a mixture containing 4 ml Eagle's basal medium (Wellcome), 1 ml bovine serum (Difco), 0.15 ml reconstituted phytohemagglutinin (Wellcome) and the buffy coat derived from 1 ml of blood. After 45 h 0.1 ml of colcemid (200 µg/ml) (Ciba) was added. Microscope slides prepared from cultures by a standard technique were stained in orcein (Harleco) and analyzed for dicentric, centric ring and acentric chromosome aberrations. Full details of the methods for culturing and fixing cells and of the criteria used in aberration analysis have been published elsewhere[5]. The aberration yields for adult blood used in the present comparison have been published elsewhere[6]. Apart from not being performed at the same time the exposure and culture conditions were identical. We have considerable



Dicentric yield plotted against radiation dose for adult blood (open symbols and dashed line) and cord blood (solid symbols and line) and fitted to the function $Y = \alpha D + \beta D^2$. The shaded areas indicate 1 SE.