34. *Studies on the Karyotype Differentiation of the Norway Rat. X*

*Translocation between the Autosome No. 11 and the Y Chromosome in the NIG-III Strain Rat occurred after γ -Irradiation and its Genetics**

By Toshihide H. YOSIDA

National Institute of Genetics, Misima

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A remarkable case of the translocation between the autosome no. 11 and the Y chromosome was found in a male of the NIG-III strain rats born after mating between the γ -ray irradiated male and the non-irradiated female. A large part of the chromosome remaining the centromere was translocated to the short arm of the no. 11 chromosome and the remaining Y with the centromere was deformed as an extremely small element like a dot. The male with such an 11/Y translocation was mated to the normal females. By such matings the rats with several abnormal karyotypes out of the 11/Y translocation males and the normal females which were expected to be produced were obtained in their offspring. Based on the segregation in the offspring it was suggested that the male determining gene would be presented near the centromere of the Y. The present paper deals with the karyotype of the 11/Y translocation occurred in the NIG-III and its segregation in their offspring.

Material and methods. The NIG-III strain rats were established from the hybrids between a wild mutant rat with p^m gene and the Castle Black strain rat (Yosida *et al.* 1961), and after then the rats with p^m gene were selected and maintained by the brother and sister matings for 60 generations. Four males of the NIG-III rats were irradiated with γ -rays (Cs^{137}) at 700R and then mated to the 4 non-irradiated females. The chromosomes were observed from the cultured cells of the tail tip by our routine procedure (Yosida 1980).

Results and discussion. 1) Normal karyotype of the NIG-III strain: The normal karyotype of the NIG-III strain rat was similar to the other strain rat by having 42 chromosomes (21 pairs). Among them 12 autosome pairs (nos. 1 to 12) and a sex chromosome pair (X and Y) were acrocentrics or subtelocentrics, and the 8 autosome

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pairs (nos. 13 to 20) were small metacentrics. The no. 3 pair of this strain rat was acrocentric similar to the WM strain rat (Yosida 1984).

2) Translocation between the autosome no. 11 and Y chromosome: By the mating between the irradiated males and non-irradiated

Figs. 1-2. Karyotypes of the NIG-III male rat with the 11/Y translocation occurred after γ -irradiation. The large part of the Y was translocated to the short arm of the autosome no. 11 and the Y was transformed to a small dot-like element. 1: Conventional Giemsa staining. 2: G-banding staining.

females, 18 F_1 offspring were obtained. Among them one male showed a translocation between the Y and the no. 11 autosome, while the other 17 rats had the normal karyotype. The translocation was as follows; the long terminal part of the Y chromosome was broken near centromere, and the broken end was translocated to the short arm of one of the acrocentric autosome pair no. 11 (Figs. 1, 2). By such a translocation one of the pair no. 11 was transformed to the metacentric shape, and the Y to an extremely small element like a dot. All of the cells examined so far, showed the same karyotype as the above description. Namely, the pair no. 11 was heteromorphic by having translocation no. 11 and the normal partner and a normal X and a small Y. This means that these two new elements were stable and they were transferred to the daughter cells through a normal cell division. Based on the above findings, it is strongly suggested that the new metacentric no. 11 and a small dot-Y should have a functional centromere and a telomere by the reciprocal translocation between these two chromosomes. The translocation between the autosome no. 11 and the Y was demonstrated by G- and C-banding stainings. The Y chromosome is known to be heavily stained by the C-banding technique through its whole length. The Y element translocated to the no. 11 chromosome was also stained heavily (Fig. 3). By such a characteristic, the Y and 11 translocation was clearly demonstrated at the metaphase figure.

3) Transmission of the 11/Y translocation into the offspring: By mating between the 11/Y translocation heterozygous males and the normal females, 133 offspring (73♀ : 60♂) were obtained in the

Fig. 3. Metaphase of the NIG-III male rat with the 11/Y translocation by C-banding staining. Enlarged 11/Y translocation chromosome is shown at the lower and right corner. The small-Y is shown by an arrow.

Table I. Segregation of no. 11 and Y translocation in the offspring of NIG-III strain rats after mating between the translocation heterozygous males (11/11-Y) and the normal females (11/11)

Sex	Karyotypes			No. of rats
	2n	No. 11	Sex chroms.	
Female	42	11/11	X/X	62(46.8%)
"	"	11/11-Y	"	1(0.7)
"	41	11/11	X/O	1(0.7)
"	"	11/11-Y	"	9(6.7)
Male	42	11/11-Y	X/dot-Y	46(34.6)
"	43	11/11	X/X/dot-Y	13(9.8)
"	"	"	X/dot-Y/dot-Y	1(0.7)
Total				133(100.0)

F₁ to the F₃ generations. The number of females was slightly higher than that of males (54.9♀ : 45.1♂) (Table I). Among 73 females, 62 (46.8%) were of the normal female karyotype with 42 chromosomes including the normal pair no. 11 and two Xs. Ten females (7.4), however, had 41 chromosomes, among which one had the normal pair no. 11, but a single X, and the remaining 9 females showed the translocation heterozygous no. 11 and a single X. Remaining one female (0.7) had 42 chromosomes, but its karyotype was conspicuous by having a translocation heterozygous pair no. 11 and two Xs. Even though these 10 had the large part of the Y element by the translocation, their sex characters were always of femaleness, because the dot-Y element was not included.

Among 60 males, 46 (34.6) had the translocation heterozygous pair no. 11 and X and dot-Y sex elements similar to the male parent. The other 13 males (9.8), however, had 43 chromosomes, among which 40 were normal autosomes including normal pair no. 11, but the sex chromosomes were remarkable by having two Xs and one dot-Y. The occurrence of the XO females and XX/dot-Y males was considerably high in their frequencies and they were almost identical

(10:13). These sex chromosome abnormalities should have occurred by non-disjunction of sex chromosomes in meiosis of the translocation heterozygous males. The XO females might have developed by fertilization of sperms without the sex chromosomes, but the XX/dot-Y males by the sperms with X/dot-Y sex chromosomes. The remaining one male (0.7) was remarkable by having 43 chromosomes including a normal no. 11, but a single X and two dot-Ys. This male would be developed by the sperm with two dot-Ys which should have occurred by its non-disjunction in the secondary meiotic division.

By mating between the female with the translocation heterozygous no. 11 and two Xs and the 11/Y translocation heterozygous male with the X and the dot-Y sex chromosomes, two females were obtained. They had the same karyotype as the parent female. This result means that the 11/Y translocation heterozygous female with two Xs was fertile, and the large part of the Y is not affected to the sex determination and the fertilization. Based on the above evidence, it is highly probable that the male determining gene or genes would be presented on the dot-Y element, locating near the centromere.

Translocation of the autosomes and the Y-chromosome has not yet been reported so far in the mouse, but in the human translocations between the no. 14 (Laurent and Dutrillaux 1976), the no. 7 (Turleau *et al.* 1976), and no. 15 (Subert and Blehova 1974) with the Y chromosome have been reported. According to their studies, these translocations have usually been related to the sexual abnormalities. In the Norway rat the individuals with the 11/Y translocation were fertile. This seems to be a good material for the study on the sex determination mechanism in mammals. Interesting is the fact that the several chromosomal anomalies occurring sequentially by mating between the 11/Y translocation and the normal female as described above. This might be due to the irregularities of the segregation of the sex chromosomes in the meiosis. These examples seem to be a good evidence for the sequential occurrence of chromosome aberrations in mammals as suggested by the author (Yosida 1983).

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