Faizullin, L.Z. and V.A. Gvozdev.
Kurchatov's Institute of Atomic Energy, Moscow, U.S.S.R. Genetic balance and dosage compensation in D. melanogaster.

According to Muller's views, the dosage compensation of X-linked structural genes in Drosophila is maintained by compensatory genes in the X-chromosome acting negatively and proportionally to their dose (1). This mechanism implies that the increase of a compensatory gene dosage by duplications of various regions of the X-chromosome would result in the decrease of the activity of some X-linked structural genes. We determined the activities of Pgd (1-0.65) and Zw (1-63.0) structural genes for 6-phosphogluconate dehydrogenase (6PGD) and glucose-6-phosphate dehydrogenase (G6PD), respectively, measuring the specific activities (SA) of the two enzymes in crude extracts of flies carrying various duplications. The results are presented in Table 1. Three different duplications for the distal part of the X-chromosome, containing

Table 1. Relative SA of 6PGD and G6PD of hyperploid females and metafemales. (SA of diploid females = 100. Number in parenthesis indicates the number of experiments).

Genotype	Dose Pgd	Dose Zw	Relative activity %% 6PGD	Relative activity %% G6PD
2X (normal)	2	2	100	100
2X+1-9A	3		138 ± 7 (7)	
2X+1-9B	3	2	141 ± 6 (13)	98 ± 7 (8)
2X+1-14A	3		137 ± 3 (4)	
2X+16A-20	2		107 ± 3 (12)	
2X+14A-20	2		96 ± 4 (11)	
2X+9B-20	2	3	106 ± 5 (12)	150 ± 7 (11)
3X (metafemale)	3	3	149 ± 5 (10)	118 ± 3 (4)
2X+Df(1)Pgd-kz(2D3-6; 2F3-4)	2	3	99 ± 4 (12)	125 ± 11 (6)

Pgd locus (1-9A, 1-9B and 1-14A regions) result in the 1.4 fold increase of SA of the 6PGD irrespective of the duplication length. In fact, this increase is proportional to the enhancement of the gene dose. On the other hand, duplications for the right part of the X-chromosome (regions 16A-20, 14A-20 and 9B-20) not covering Pgd locus exert no influence on the Pgd activity. Therefore in contrast to Muller's results (2) we failed to detect any negative (or positive) regulatory genes for Pgd locus within either the right or the left parts of the X-chromosome. The similar results were obtained for Zw locus: the addition of the right or the left parts of the X-chromosome resulted in the increase of the G6PD activity proportionally to the Zw dose (9B-20 region) or showed no effect (1-9B).

Muller suggested that dosage compensation results from the simultaneous action of the group of compensatory genes scattered through the X-chromosome. Therefore the investigation of the influence of the additional entire X-chromosome in females on the SA of the two dehydrogenase was undertaken. Table 1 shows the 1.5 fold increase of 6PGD activity in metafemales (3X2A) as compared to normal females (2X2A) proportionally to the gene dosage. The 1.2 fold increase was observed for G6PD activity. The addition of the third X-chromosome with small Pgd^- deficiency (2D3-6;2F3-4) had no effect on 6PGD activity although G6PD increases to 25% as compared to normal females. These results may be considered as showing the equal activity of genes in each X-chromosome in females and metafemales. The obtained data are in contradiction to Muller's idea of the existance of X-linked compensatory genes able to depress the activity of the other genes of X-chromosome proportionally to the number of X-chromosomes.

The two levels of the activity of the X-chromosomes of males and females could be deter-

Table 2. 6PGD and G6PD activities of triploids (3X3A) and intersexes (2X3A). (SA is expressed as nmoles of NADP/mg protein/min. Numbers in parenthesis indicate the number of experiments.)

Genotype	6 P G D SA	6 P G D SA:X-chromosome number	G 6 P D SA	G 6 P D SA:X-chromosome number
3X3A	184 ± 8 (5)	61	263 ± 10 (5)	88
2X3A	302 ± 35 (4)	151	384 ± 47 (4)	192

(Continued at bottom of next page)

Ouweneel, W.J. and J.M. van der Meer. Hubrecht Laboratory, Utrecht, Netherlands. Determination and regulation in the haltere disc.

To study the organ map of the haltere disc, mature discs were fragmented and transplanted into mature larvae. The analysis of the metamorphosed implants showed that the prospective metathorax is located in the anterior part of the disc, including many "adventitious bristles" (appearing only in implants) and two newly discovered metathoracic papillae. The knob in the posterior part of the disc is the anlage of the capitellum; this is concentrically surrounded by the anlagen of the pedicel and the scabellum, both carrying characteristic groups of sensilla campaniformia, which were also localized. The haltere anlagen are surrounded by those parts of the prospective metathorax which will be located at the base of the haltere in the imago, including the "metathoracic bristle group" and the metathoracic spiracle. This organ map is homologous with that of the wing disc (Murphy[1]).

To investigate regulative effects in disc fragments (cf. Schubiger[2] and Bryant[3] for the leg disc), anterior and posterior fragments of mature discs were cultured in the abdomina of adult hosts, where they usually underwent cell proliferation from the wound surfaces. After the culturing period (8 to 23 days) they were transplanted back into mature larvae. Analysis of the metamorphosed implants showed that 3/4 anterior (proximal) fragments formed blastemas from which posterior (distal) structures arose by regeneration. Some structures were regenerated in duplicate. It seems that the metathorax anlage is able to regenerate a complete haltere; this has to be confirmed, however, by transection at more proximal levels. (Transplantation experiments with different anterior-posterior transection levels and with medial and lateral fragments are in progress.)

On the other hand, 1/4 posterior disc fragments formed blastemas in which the distal structures normally produced by these fragments were duplicated in mirror image. This led to the formation of bilaterally symmetrical transplants. The posteriormost anlagen were duplicated most frequently, i.e., those of the metathoracic bristle group, the capitellum, and the ventral pedicellar and scabellar sensillae groups. The strictly symmetrical structure of the transplants suggests two important, preliminary conclusions which have already been anticipated theoretically[4]: (1) if proliferation is limited, a small blastema is formed; nevertheless the fragment and blastema together seem to "split up" symmetrically ("homonomous arealization") and to produce only the most posterior anlagen in duplicate, while the more anterior anlagen originally present in the fragment drop out altogether; this would imply a partial "repatterning" within the original disc fragment. (2) If proliferation is very extensive, a blastema is formed that is larger than the original fragment; here also the fragment and blastema together seem to "split up" symmetrically, and now produce not only all the structures typical for the original fragment in duplicate, but also more proximal structures for which no anlagen were present in the original fragment. This leads us to a concept of "duplicative regeneration" which is at variance with the hypothesis of Bryant[3].

References: 1. Murphy, C. 1972, J. exp. Zool. 179:51-62; 2. Schubiger, G. 1971, Devel. Biol. 26:277-295; 3. Bryant, P.J. 1971, Devel. Biol. 26:606-615; 4. Ouweneel, W.J. 1972, Acta Biotheor. 21:115-131.

(Continued from preceding page)
mined by different ratios of the number of X-chromosomes to autosome sets. Therefore the comparison of SA of the 6PGD and G6PD in triploids (3X3A) and intersexes (2X3A) was undertaken. Table 2 shows that the SA of the two dehydrogenases are higher in intersexes than in triploids. The calculation of the level of enzymatic activity per X-chromosome showed the two-fold increase of the activity of these genes in intersexes as compared to the diploid females. Thus the similar (low) levels of the activity of X-chromosome are characteristic for the normal, hyperploid and triplo-X females. In contrast, males and intersexes (the latter in our experiments had male phenotype) have a two-times higher level of activity of X-chromosome. It is possible to suggest that the low level of X-chromosome activity is determined by the X:A ratio more or equal to 1.0 while the high level of the X-chromosome activity is expressed when X:A is less than 1.0.

References: 1. Muller, H.J. 1950, Harvey Lect. 43:165-229; 2. Muller, H.J. and W.D. Kaplan 1966, Genet. Res. 8:41-59.