

found. There is some doubt, therefore, that this double-X carries that gene. Examination of ganglia show that the chromosome is very long at anaphase (and therefore is of the double type); at metaphase, the chromosome appears V-shaped, but a tip of the V rather than the apex is directed toward the center of the plate.

(2) The tandem double-X. Pairs by forming a spiral and, by crossing over, manufactures single rod chromosomes. This compound was derived by inducing a crossover between the short arm of the Y at the tip of the X·Y chromosome and the base of  $sc^4$ . Structurally, the chromosome may be written as  $sc^4 + EN: YLsc^8$ ; the raised dot indicates the position of the centromere. The  $sc^4$  chromosome used carried  $y\ car\ m\ wa$ ; the X·Y chromosome may be represented as  $car^+ m^+ y \cdot y^+$ . Crossover studies indicate that a striking excess of single chromosomes is recovered; it is presumed that crossing over is occurring with normal frequency but that nonrandom disjunction favors the inclusion of the single X in the egg nucleus. The long arm of the Y, present at the base, has been replaced by the duplication  $BS$ . Such a chromosome should, with some low frequency, yield instances where crossing over has occurred between this duplicating fragment at the base, and the homologous region at the end of the  $sc^4$  chromosome. This chromosome would be a double ring, of interest because the recovery of single rings from it by crossing over would have to depend on certain three-dimensional properties of this type of chromosome.

Novitski, E. Useful derivatives of the X·Y chromosome.

Among a large number of rearrangements involving the X·Y chromosome found while looking for a special type of inversion,

two may be of particular interest in experiments where the inverted sequence of the X·Y chromosome is troublesome because it gives either too much or too little, crossing over when heterozygous with a normal sequence. One of these, labeled X·Y In X·Y 26, has one break in the heterochromatin, the other in section 10A. No crossovers have been found when this chromosome is with  $sc^8$  or a normal chromosome. Another, X·Y In X·Y 24, appeared to be an almost complete reinversion since it crosses over freely with a normal sequence. The crossover class with the left end of a normal chromosome and the base of In X·Y 24 is inviable in the male, however, indicating that some normally proximal genes, located distally in the X·Y chromosome, had not been shifted back into their normal position by the second inversion. To make a chromosome that would be free of this complication, In X·Y 24 was crossed to an attached-X detachment (A2) which carried the long arm of the Y chromosome. A single crossover replaced the deficient base by a normal base. The chromosome thus constituted has been tested by mating to  $y^2\ su^{wa}\ wa\ bb/0$  females. F<sub>1</sub> males are fully fertile and viable, although there must still be a small duplication at the distal end. It is carried in stock as:  $Ins\ 24L + A2R\ y/y^2\ su^{wa}\ wa\ bb$  and  $Ins\ 24L + A2R\ y\ v/y^2\ su^{wa}\ wa\ bb$ .

Oftedal, Per Genetics and histogenesis of a new tumor tu(2)49k.

A tumor stock developed spontaneously from a stock of  $ma^{49d}$  shows black aggregations in about 50%-60% of the flies in stock. The tumor may be located in

any part of the fly, but usually in the abdomen. In outcrosses 2% incidence is obtained in F<sub>2</sub>. In backcrosses the incidence is up to 10% and may rise to 34% when crossed to the stock from which it arose. The main gene is completely recessive and is located in the second chromosome, probably between  $c$  and  $px$ . It is not an allele of  $mt^A$  of Hartung (Hartung, J. Hered. 41:269). Modifiers have been located in the middle of chromosome 3, rather closely linked to  $ma$ , and in chromosome 1. The third-chromosome modifier is present in the Sb and H chromosomes of the Cy/Pm; H/Sb stock in this laboratory.

Histologically the tumors start as melanization of the larger lymphocytes, visible at 34 hours after hatching, and so complete as to sometimes obscure the presence or absence of a nucleus at 75-78 hours of age. At around 65 hours, some of the smaller lymphocytes change into spindle-shaped cells and often aggregate into clumps, showing extra- and intracellular deposits of melania. The pericardial blood-forming organ is always affected. Melanization progresses at least until 96 hours of age. Black aggregations in adults consist of cellular detritus and black masses, presumably melanin. The rectal epithelium of old tumor larvae sometimes shows nuclei reminiscent of early- and late-prophase ganglion nuclei, but of much larger size.

Ohnishi, E. Bilateral asymmetry and correlative expression of wing and hind leg in *Bd*<sup>491</sup>. The phenotype of this *Bd* is very variable, ranging from wild-type to vg-like appearance, and the grades of expression of the wings of one individual are sometimes independent of each other. Besides the excision of the wing, hind legs are crumpled and this occurs only associated with heavy expression of the wing excision. This is true also with respect to both sides of one asymmetrical individual. This correlation suggests that some cooperation exists between the imaginal discs of the dorsal mesothoracic and the ventral metathoracic, rather than a mosaic nature of this mutant.

Ohnishi, E. Tyrosinase activity during puparium formation in *D. melanogaster*. Rapid increase and decrease in tyrosinase activity during puparium formation was observed by the following techniques. Larva or pupae were ground in a glass mortar and made into a pulp with distilled water. This was centrifuged, and the supernatant fluid (without lipid layer) and precipitated cell debris were measured separately by Warburg's manometer, using catechol as the substrate. Special care was taken to use individuals of exactly the same stage. Values for  $Q_{O_2}$ , calculated from the oxygen uptake of the first five minutes, are shown in the table.

Stage	Tyrosinase activity ( $Q_{O_2}$ )		
	Extract	Cell debris	Sum
larva moving on the wall .....	trace	trace	-
larvae immobile (ca. 1 hr before puparium formation) .....	8.2	14.9	23.1
prepupa with white puparium .....	12.2	15.5	27.7
prepupa 1.5 hr after puparium formation .....	3.7	10.1	13.8
prepupa 3.0 hr after p.f. ....	1.6	6.4	8.0
prepupa 6.0 hr after p.f. ....	2.2	4.4	6.6
prepupa 12 hr after p.f. ....	0.7	1.4	2.1

$Q_{O_2}$  = microliter  $O_2$ /mg body weight hour

In spite of the very high activity of the white prepupa preparation by the grinding method, an almost inactive preparation was obtained by the following procedure. White prepupae were immersed in 0.75% NaCl solution and dissected by needles under a binocular microscope, with special caution not to injure the tissues, and the puparium was torn into 3-4 pieces, setting free body fluid into the NaCl solution. Remaining tissues and puparium were ground and suspended in water. This diluted body fluid and suspension were both almost inactive. When both were mixed and left to stand for 10 minutes, then separated by centrifuging, some activity appeared. Considering these and other factors, it may be concluded that tyrosinase in vivo is probably in an inactive state, and it is activated by an activator of unknown nature present in the tissues. Details will be shown elsewhere.