

Schwalm, F.E., H.A. Bender and D. Klingele. University of Notre Dame, Indiana. Ultrastructural organization of the eggs of the female sterile mutant- $lz^{61f}$  (*D. melanogaster*).

localized heterogeneously in  $lz^{61f}/lz^{61f}$  eggs. Similarly, the floccular bates spheres have merged and their contents form large pockets. These pockets are surrounded by a single layer of mitochondria (fig. 1). Alpha spheres are restricted to distinct regions of the egg where almost no other components are found. Large spaces are filled with endoplasmic reticulum which frequently assumes the form of annulate lamellae (fig. 1). Polar granules, similar to those found by Mahowald (1968) and in *Coelopa* eggs (currently under investigation in our laboratory), occur in different areas inside the egg, remote from the posterior pole and widely separated from each other (fig. 2).

These observations suggest that disorganization of the egg at termination of oogenesis could account for the lack of development. A more detailed study of oogenesis in this mutant has been initiated.

Ultrastructural organization of eggs from the  $lz^{61f}$  mutant one day after oviposition was studied. The distribution of components appears to be highly disorganized as compared to that in normal eggs (King 1960, Okada and Waddington 1959). Mitochondria, which are distributed evenly throughout the wild type eggs, are

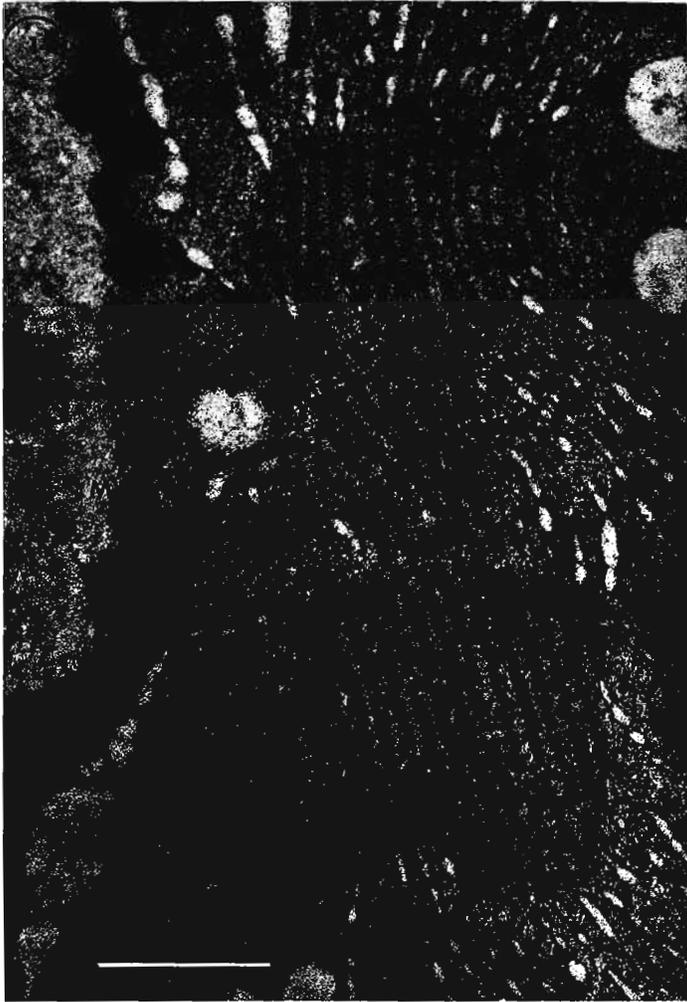


Fig. 1. Electronmicrograph from a longitudinal section of a  $lz^{61f}$  egg. Row of mitochondria (M) on margin of floccular space. Extensive cytoplasmic area, rich in endoplasmic reticulum (ER) continuous with annulate lamellae (AL).

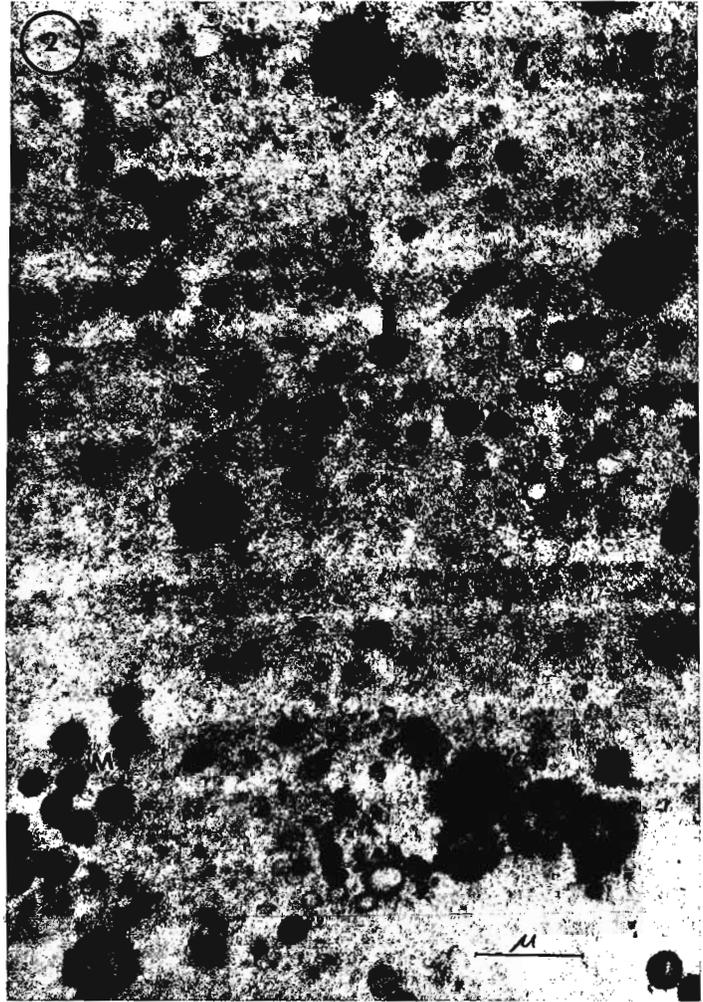


Fig. 2. Electronmicrograph from central region of  $lz^{61f}$  egg. Granular bodies (Gb), presumably identical with polar granules in wild type eggs. Mitochondria (M).

References: King, R. 1960, Growth 24: 265. Okada, E. and Waddington, C.H. 1959, J. Embryol. Exp. Morphol. 7:583. Mahowald, T.P. 1968, J. Exp. Zool. 167: 237.  
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Schwinck, I. University of Connecticut, Storrs, Connecticut. Phenogenetic inhibition and enhancement of drosoppterin formation in various mutants of *D. melanogaster*.

Earlier it was found that the concentration dependent phenocopy effect of the xanthine dehydrogenase inhibitor 4-hydroxy-pyrazolo(3,4-d)pyrimidine (HPP) on drosoppterins in cinnebar (cn) eyes decreases the amount of drosoppterins slightly below the level of the rosy (ry)

strain but never below 10% of the cn control value. We now asked the following: Is a low level drosoppterin synthesis uncontrollable by the xanthine dehydrogenase metabolites? Or will these metabolites decrease the drosoppterin synthesis further in other eye color mutants which normally have a rather small amount of drosoppterins and a functional xanthine dehydrogenase? Therefore, HPP was fed to larvae of a control cn strain and of the following eye color mutants: claret (ca), orange (or<sup>66k</sup>), pink-peach (p<sup>p</sup>), raspberry (ras<sup>2</sup>), and rosy (ry<sup>2</sup>); all strains also contained cn in order to block the ommochrome synthesis and thus to facilitate the visual classification and the extraction of drosoppterins in acidified ethanol. At various breeding temperatures (18°C, 22.5°C, 27°C), larvae were raised on control food and on HPP-food (0.005 M HPP), and drosoppterins extracted from whole heads (1 head/.1 ml or for low values 2 heads/.1 ml), and the absorption at 485 mμ determined in a Beckman microcuvette procedure. Our data demonstrate clearly that the HPP can further decrease the drosoppterin formation in all mutant strains except ry, although to a different extent. Furthermore, HPP-feeding also causes the temperature dependent semi-lethality and delay in development which is so characteristic for the ry mutants and the HPP-caused phenocopy in cn;ry<sup>+</sup> animals. The statistic evaluation of over 6 day old flies of the 22.5°C growth series shows highly significant differences (p=0.001) of the means of drosoppterin quantities for control versus HPP-food for the genotypes cn;ca and cn,or<sup>66k</sup> and cn;p<sup>p</sup> and ras<sup>2</sup>;cn. However, there is always some residual drosoppterin synthesis, although on different low levels for the different mutant strains.

Phenylalanine crystal implantation into pupae can increase the drosoppterin synthesis in maroon-like and rosy eyes and in the HPP-caused rosy-like phenocopy of cn genotype, as published earlier. This suggested the following two working hypothesis: (A) Phenylalanine is involved in a control mechanism interacting with the xanthine dehydrogenase metabolites and, therefore, acts specific in the maroon-like and rosy mutant and the phenocopy. (B) Phenylalanine acts at a later, more general step in the drosoppterin biosynthesis; in this case it should also increase the drosoppterin formation in other eye color mutants which have an active xanthine dehydrogenase. The implantation of large phenylalanine crystals into abdomen of late pupae already forming drosoppterins in their eyes or into 0-1 hr old flies resulted in a much better long-term survival compared to implantation in younger pupae. Obviously, a smaller increase is expected because 1/2 to 2/3 of the eye drosoppterins are deposited before the onset of the experimental phenylalanine supply. Nevertheless, for cn;ca and cn,or<sup>66k</sup> and cn;p<sup>p</sup> an increased drosoppterin synthesis to two - to three-fold amounts of the control value was found, which is almost as extensive as in the cn;ry<sup>2</sup> flies used as a control in this experimental series. In contrast, the ras<sup>2</sup>;cn flies did not show a phenylalanine dependent increase of drosoppterin synthesis, although in this mutant strain 3/4 of the normal drosoppterin formation occurs after the eclosion of the flies and thus would be under the influence of the phenylalanine implant in the experimental series. These data suggest that phenylalanine interacts with some late step on the drosoppterin pathway (hypothesis B), resulting in some mutants in a phenocopy distinctly different from the "normal" eye color phenotype.

These epigenetic metabolite control mechanisms thus drastically alter the eye color phenotype: (a) the inhibitor can decrease the drosoppterin quantity to as low as 10% of "normal", and (b) the enhancer can cause a several-fold increase in drosoppterin formation in various eye color mutants. These results are to be reported in detail elsewhere.

References: Schwinck, I., 1965, Zeitschrift f. Naturforschung 20b: 322-326. Schwinck, I., 1967, Zoolog. Anz. Suppl. 30: 382-390. Schwinck, I., 1968, Intern. Cong. of Genetics, Tokyo, Vol. I: 125. Schwinck, I., 1969, Genetics 61: s53. (Supported by USPHS Grant GM-10256 and a Grant from the University of Connecticut Research Foundation).