

It appears that *clw* mutant expression is possible only in combination with unstable singed-strong alleles. But normal *clw*⁺ phenotype is expressed in *sns*, *sn*^m and *sn*⁺ alleles. The recombination in double mutant X-chromosome is free; the polytene chromosomes seem normal. We tried to "divide" two mutations by crossing over in *sn*49 *clw*/ct 1z females but failed; each time crossover with *sn*49 allele had *clw* expression. To explain this unusual situation we assumed that mutant state for two closely linked genes is related to insertion of a hypothetical IS-like segment into the region of these loci. The insertion segment is capable of changing its orientation or excision from the host chromosome. As shown in Fig. 1, insertion of the IS into orientation "1" blocks the normal expression of *sn* and *clw*. According to this suggestion, it is possible to predict all observed allelic transitions:

- (1) Regularly recurring transitions from the normal state to the original double mutant are due to the capacity of the IS to change its orientation, remaining in the same site;
- (2) Incorrect excision of the IS from the chromosome gives rise to a stable mutant *sn* allele and normal wing phenotype;
- (3) Precise excision of the IS produces stable wild type;
- (4) Intralocus transposition of IS segment is possible, resulting in the appearance of a novel *sn*^m derivative and *clw*⁺ state.

Unfortunately we couldn't identify *clw* mutation with known club-like wing mutations in the X chromosome. In the region of the *sn* locus (21.0) there are two mutations acting on wings: cut (20.0) and kinked femur (lost). But ct/*sn*49 *clw* flies are normal.

A similar case of simultaneous changing of two mutations was described earlier (Demerec and Slyzinska 1937). In T(1;4) *w*^{mt} 258-18 translocation the distal region of the X chromosome is transposed to the heterochromatin area of chromosome 4 with unstable mutant expression of white and roughest genes. Here mutant condition of the white gene (*w*, *w*^{ch}, *w*^{cr}) was observed each time in *rst* facets, but in *w*⁺ sectors *rst* and *rst*⁺ facets were observed. So there is definite similarity between some position-effect inducing factors and instability phenomena, as Demerec suggested (Demerec and Slyzinska 1937).

References: Berg, R.L. 1974, DIS 51:100; Ivanov, Yu.N. 1974, DIS 51:71; Golubovsky, M. D., Yu.N. Ivanov and M.M. Green 1977, Proc. Nat. Acad. Sci. USA 74:2973; Golubovsky, M.D. 1978, DIS 53:171; Demerec, M. and H. Slyzinska 1937, Genetics 22:641.

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Lyon-I, Villeurbanne, France. Mitochondrial activity during metamorphosis in *D. melanogaster*.

Oxygen consumption shows drastic changes during development in *Drosophila* (Fourche 1969). Recently, it has been stated that the variations in respiration at the larval stage may partly be explained by changes in either mitochondrial content or mitochondrial activity (Rezvoy,

Fourche and Guillet, in press). Both were found to correspond to the feeding periods (Fourche 1967a) and the modifications in hormone balance. The present paper investigates the role of mitochondria in the control of respiration during metamorphosis.

The strain used in these experiments was a wild strain Algeria. Each batch included about 1000 pupae isolated within three hours of puparium formation to provide homogeneous batches. Age was counted from the middle of the isolation period. The pupae were kept at 25°C until they were used at the appropriate age. Mitochondrial isolation and oxygen measurement by means of a Clark electrode are described elsewhere (Rezvoy, Fourche and Guillet, in press). Mitochondrial proteins were estimated by the Folin-phenol method of Lowry et al. (1951).

In the 98 hour old larvae, the mitochondrial protein content was 8.3 µg per larva; two hours after puparium formation it was only 5.5 µg. It increased after 60 hours and reached 7.6 µg at emergence (Fig. 1).

The Q O₂ (µl O₂/hr/mg mitochondrial protein) was measured at state 3; the substrate was sodium succinate. In the 98 hour old larvae, the Q O₂ was 71 µl hr⁻¹mg⁻¹. After puparium formation Q O₂ followed a U-shaped curve; the lower value was 15 µl hr⁻¹mg⁻¹ after 36 hours. Then it increased until emergence: 71 µl hr⁻¹mg⁻¹ (Fig. 1).

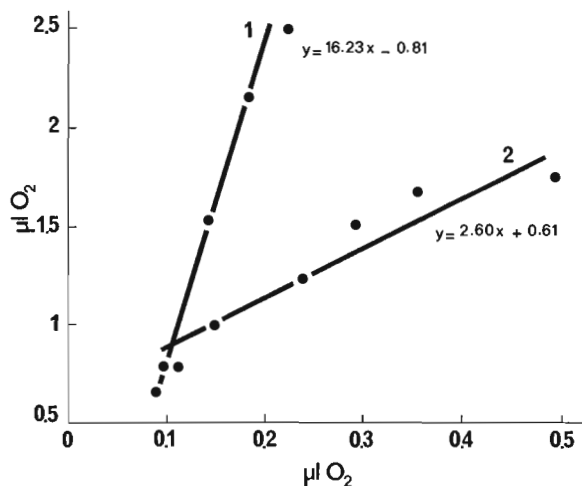


Fig. 1. Evolution of mitochondrial content (a) and mitochondrial activity (b). 1 - puparium formation, 2 - larvo-nymphal apolysis.

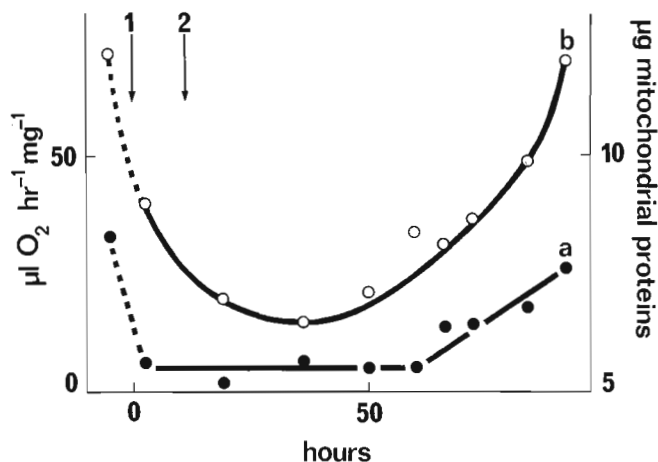


Fig. 2. Abscissa: oxygen consumption of the mitochondrial population. Ordinate: oxygen consumption of the whole pupa. 1 - descending phase, 2 - ascending phase.

Before puparium formation the respiratory rate of the larvae decreased (Fourche 1967b); a concomitant disappearance of mitochondria and a decrease in mitochondrial activity were observed. The oxygen consumption of the pupa was also shown to follow a U-shaped curve (Fourche 1969). During the descending phase of this curve, there was only a decrease in the mitochondrial activity without any change in the mitochondrial content. In contrast, the ascending phase was followed first by an increase in activity and subsequently by increases in both mitochondrial activity and content in order to answer a greater energy demand.

The results may be summarized as follows. The oxygen consumption of the mitochondrial population of a pupa (specific respiratory activity \times mitochondrial content of a pupa) also follows a U-shaped curve. If these values are compared with the respiratory rate of the whole pupa at 25°C (Guillet and Fourche 1973), a strong correlation between them can be seen. There are two linear relationships (Fig. 2), one for the descending phase of the U-shaped curve and one for the ascending phase. However, in the first case the mitochondrial oxygen consumption constitutes a smaller fraction of the total respiration. One of the most likely interpretations involves the presence of degenerated mitochondria in the pellets which may already be observed at the end of the larval stage: less than 1% (Rezvoy, Fourche and Guillet, in press). It is likely that the degenerated mitochondria have a very low oxidative activity as was shown for cytochrome C oxidase (Bulos et al. 1972). These degenerated mitochondria are probably connected with histolysis processes which begin after ecdysone release (Hodgetts et al. 1977).

Finally, it may be said that the mitochondria play a role in the control of respiration both in larva and pupa. They are able to modulate oxygen consumption either by changes in mitochondrial activity or the quantity of mitochondrial proteins or both.

References: Bulos, B., S. Shukla and B. Sacktor 1972, Arch. Biochem. Biophys. 149:461; Fourche, J. 1967a, Arch. Anat. Microsc. Morph. Exp. 56:141; Fourche, J. 1967b, J. Insect Physiol. 13:1269; Fourche, J. 1969, Ann. Biol. 8:334; Guillet, C. and J. Fourche 1973, Ann. Zool. Ecol. Anim. 5:533; Hodgetts, R., B. Sage and J. O'Connor 1977, Dev. Biol. 60:310; Lowry, O.H., N.J. Rosebrough, A.L. Farr and R.J. Randall 1951, J. Biol. Chem. 60:310.