Table 1. Fecundity of Ceresan treated and control D. melanogaster flies.

Concentrations	Total fecundity	Mean daily egg production/female
Control	6910	34.55 ± 0.23
25 mg	5016	25.08 ± 0.23*
35 mg	3934	19.67 ± 0.22*
45 mg	3736	18.68 ± 0.23*

* $P < 0.05$, by Analysis of Variance.

It is clear from Fig. 1 that, in controls, fecundity suddenly declines after the first day and gradually increases, whereas in different concentrations of Ceresan, egg laying pattern is abnormal and the fecundity is altered day to day, with many fluctuations. An abnormal egg laying behavior was also shown by hens fed with 0.5 ppm mercury, which laid a greater percentage of eggs outside nest boxes compared to controls (Heinz 1976).

Perusal of Table 1 indicates that Ceresan has a significant effect upon fecundity of D. melanogaster. Analysis of variance computed to compare the fecundity of treated flies with that of controls has shown that fecundity is significantly reduced even by the lowest concentration of 25 mg of Ceresan tested ($P < 0.05$). A dose-related reduction in fecundity is also evident from Table 1. Such an effect of mercury on fecundity is also known in other animals (Heinz 1974; Spann et al. 1972).

Ramel and Magnusson (1969), analyzing the genetic effects of mercurial compounds on D. melanogaster, state that "Mercurials given in the food to Drosophila larvae or adult flies obviously reach the gonads, where they cause chromosome disturbances presumably of the similar nature as the ones observed cytologically in plant cells". A chromosome breaking action of mercury has also been shown by Levan (1945). Further, Ramel (1969) also points out that Phenyl mercury causes more chromosome breakage than methyl mercury compounds. Phenyl mercury is also shown to cause somatic mutations, pollen sterility and chromosome fragmentation in plants (Mac Farlane 1950). The authors are of the opinion that chromosome disturbances caused by Phenyl mercury in the gonads and the germinal cells may be one of the major causes for the effects on reproduction in Drosophila.

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References: Gruwez, G., C. Hoste, C.V. Lints and F.A. Lints 1971, Experientia 27:1414-1416; Heinz, G. 1974, Bull. Environm. Contamination Toxicol. 11:386-392; Heinz, G.H. 1976, J. Wildl. Manage 40:710-715; Levan, A. 1945, Nature 156:167-177; Lints, F.A. 1971, Gerontologia 17:39-51; Mac Farlane, E., W.E. 1950, Genetics 35:122-123; Ramel, C. 1969, Hereditas 61:208-230; Ramel, C. and J. Magnusson 1969, Hereditas 61:231-254; Spann, J.W., R.G. Heath, J.F. Kreitzer and L.N. Locke 1972, Science 175:328-331.

Golubovsky, M.D. and I.K. Zakharov. Institute of Cytology and Genetics, Novosibirsk 90, USSR. Simultaneous reversions of two sex-linked unstable mutations.

Since 1973 an outbreak of mutability of singed bristle locus was observed in natural populations of D. melanogaster. Most of the new alleles were unstable and putatively interpreted as insertions (Berg 1974, Ivanov 1974, Golubovsky et al. 1977, Golubovsky 1978). In 1975 in the progeny of one male from a wild Far East population we found a remarkable case of simultaneous appearance of two mutations in the same X chromosome: singed and club wing (clw). In all male progeny the original sn49 allele had strong mutant expression, but club wing phenotype varied in penetrance and clearly was expressed in only 10-11% of males carrying the clw allele (at 25°C). In special tests 225 males with phenotypically normal wings crossed with $\overline{XX}$/Y females produced 1408 club-wing F_1 males out of 13829 tested, or 10.2%. At the same time, males with mutant club-like wings produced 11.7% of club-like sons among 4839 tested. It is evident that the rate of expression of clw phenotype in the progeny does not depend on parental phenotype. Instability of the two mutations was tested in successive generations. We isolated some allelic derivatives of the original sn49 alleles, as stable and unstable (Table 1). The revertants from $sn49^s$ (strong mutant expression) to sn^+ occurred with a frequency of 3×10^{-3}. They had absolutely normal bristles and wings, but some were stable (as $sn49^+$ 1-1), others unstable (1-4 and 7-3) (see Table 1). The last ones in turn were also capable of producing two types of "contra-revertants": (1) with original bi-mutant condition and (2) with mutant singed bristle phenotype and normal wing (clw^+) (as $sn49^s$ 18-1). The moderate sn^m derivaties were discovered to be quite unstable. The total scheme of allelic transitions is given in Fig. 1.

Table 1. The mutation frequency of unstable sn49 allele and its derivatives in the progeny of crosses with $\overline{XX}/Y$ females.

Allele sn49 and its derivatives	Wing phenotype	Bristle phenotype of F_1 sn^s	sn^m	sn^+	Direction and frequency of mutations
$sn49^s$ *	club-like	14434	5	47	$sn^s - sn^+$ 3.2×10^{-3} $sn^s - sn^m$ 3.4×10^{-4}
$sn49^s$ (18-1)	normal	9223	0	0	stable
$sn49^+$ (1-1)	normal	0	0	10172	stable
$sn49^+$ (1-4)	normal	2	3	11152	$sn^+ - sn^s$ 1.8×10^{-4} $sn^+ - sn^m$ 2.7×10^{-4}
$sn49^+$ (7-3)	normal	11	1	12042	$sn^+ - sn^s$ 9.1×10^{-4} $sn^+ - sn^m$ 0.8×10^{-4}
$sn49^m$ (1-23)	normal	0	3465	36	$sn^m - sn^+$ 1.0×10^{-2}

* "s" means strong singed bristle phenotype; "m" - moderate; "+" - normal.

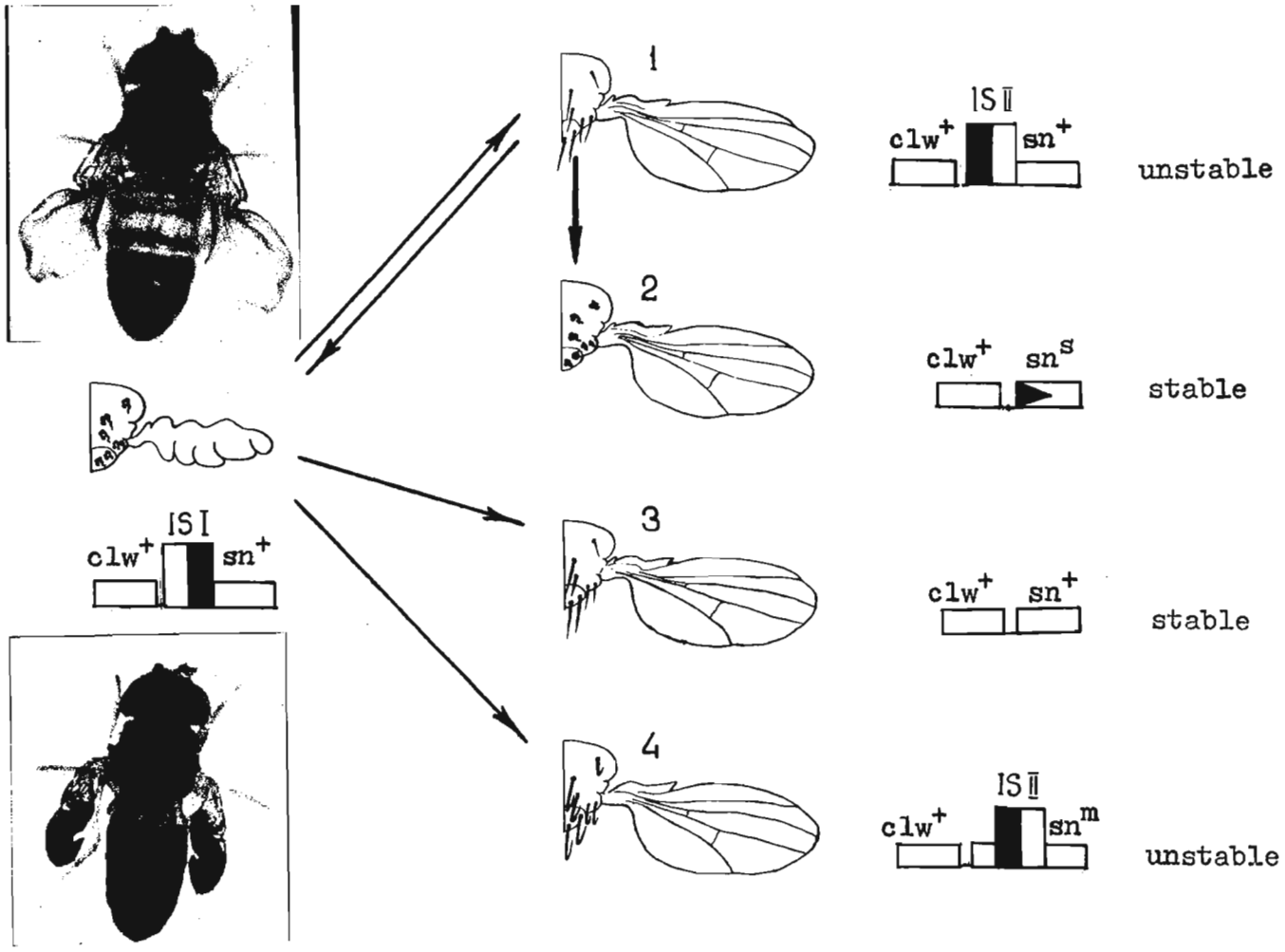

Fig. 1. Simultaneous reversions of two closely linked sn and clw mutations and possible explanation on the basis of insertion. Left - photo of original double mutant (two expressions of clw phenotype); right - observed allelic transitions to stable and unstable derivatives. For details see text.

It appears that clw mutant expression is possible only in combination with unstable singed-strong alleles. But normal clw^+ phenotype is expressed in sn^s, 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 sn49 clw/ct 1z females but failed; each time crossover with sn49 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/sn49 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.

Guillet, C. Université Claude Bernard 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 O_2 (µl O_2/hr/mg mitochondrial protein) was measured at state 3; the substrate was sodium succinate. In the 98 hour old larvae, the Q O_2 was 71 µl $hr^{-1}mg^{-1}$. After puparium formation Q O_2 followed a U-shaped curve; the lower value was 15 µl $hr^{-1}mg^{-1}$ after 36 hours. Then it increased until emergence: 71 µl $hr^{-1}mg^{-1}$ (Fig. 1).