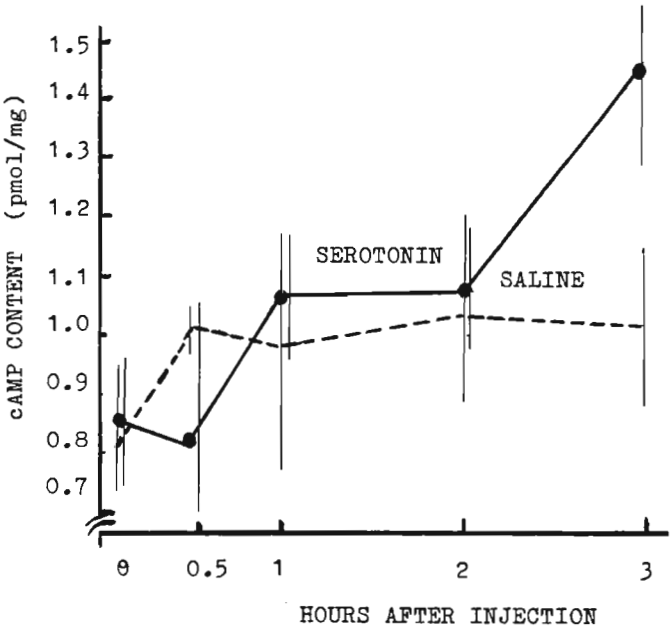

Figure 1. The dynamics of the increase in cAMP content following serotonin injection.

of serotonin effects on both the locomotor activity and cAMP content. N-acetylserotonin could either compete with serotonin for binding with the same receptor of serotonin-sensitive adenylate cyclase or affect any other factors involved into regulation of cAMP content. It is also possible that in our experimental design, when serotonin is injected into Drosophila abdomen, its effects could be amenable for registration only after 2 hr, since the substance has to be transported to the proper sites of its primary action.

References: Kamyshev, N., G. Smirnova, E. Savvateeva, A. Medvedeva & V. Ponomarenko 1983, Pharm. Biochem. Behav. 18: 677-681; Evans, P.D. 1980, Insect Physiol. 15: 317-473; Savvateeva, E. & N. Kamyshev 1981, Pharm. Biochem. Behav. 14: 603-611.

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Apparent neutrality of amylase in **Drosophila pseudoobscura** grown on starch and maltose media.

In order to further understand the forces which affect genetic variability in natural populations, we have studied the amylase locus in **Drosophila pseudoobscura.** Amylase hydrolyzes starch to maltose. There are two common alleles at this locus in natural poulations, $Amy^{1.00}$ and $Amy^{.84}$. We previously found that the $Amy^{1.00}/Amy^{1.00}$ homozygote is significantly more viable when in direct competition with the other two genotypes under stressful conditions (25°C, low amount of starch) (Seager & Anderson, submitted to Evolution).

We used the same 40 lines with which we measured viability (20 independently derived lines of the two homozygotes, all homokaryotypic for the Arrowhead inversion) to establish two starch and two maltose population cages. For each set of cages initial frequencies of $Amy^{1.00}$ were near .90 or .15. In order to approximate the conditions under which viability differences had been found, the cages were kept at 25°C and we attempted to keep both starch and yeast amounts relatively low. The food contained 6% starch or maltose, 2% killed brewers yeast, 1.5% agar, and propionic acid.

The generations in the cages were discrete. Each generation we obtained adult and zygotic allele frequencies by electrophoresing 200 adult males and 200 adult females from the cages, and an equivalent number of adults raised under nearly optimal conditions from egg samples. The cages were continued for 10 generations with each generation lasting about a month. After generation 8 in the maltose cages and 7 in the starch cages, zygotic samples were no longer obtained. The starch cages were increasingly difficult to maintain and at generation 8 the population in starch cage I decreased substantially.

Table 1. Frequency of $Amy^{1.00}$ in population cages of **D.pseudoobscura** maintained on starch or maltose medium. The other allele present was $Amy^{.84}$. 800 alleles were sampled at each life stage in each generation. Z = zygotic frequency and A = adult frequency.

Generation	Maltose I		Maltose II		Starch I		Starch II	
	Z	A	Z	A	Z	A	Z	A
0		87.9		16.7		87.5		15.8
1	91.1	96.3	25.3	22.5	77.2	82.5	33.1	32.2
2	98.3	95.6	24.8	32.4	78.6	81.0	33.7	30.6
3	95.9	97.1	29.0	24.0	80.0	83.2	22.6	26.3
4	95.1	97.6	24.1	25.7	82.2	83.2	34.2	29.7
5	97.0	90.3	29.6	28.0	82.7	85.7	28.2	26.8
6	96.3	93.8	30.1	31.2	83.2	83.6	29.1	25.2
7	97.4	93.8	32.0	31.0	87.5	83.4	24.3	22.5
8	95.5	94.3	33.1	31.7		92.3*		24.7
9		92.9		33.0		93.8		15.7
10		91.5		38.3		96.7		21.6

* population size drastically reduced.

The results are shown in Table 1. There is little consistent allele frequency change nor any evidence of selection in any of the cages. In particular the starch cages do not show any more change than the maltose cages. No evidence of selection has been detected using an experimental system designed to maximize the chance that selection will occur. Results from other experiments have been contradictory. Some have also failed to detect selection involving amylase (Yardley et al. 1977, Powell & Amato 1984) while others have seen selection (Anderson et al. 1979, Powell & Andjelkovic 1983). Taken together, these experiments emphasize how complex selection can be and show that the question of whether variation is selected or neutral may have no easy answers.

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EM mapping of rearrangements in the 24-25 sections of **D.melanogaster** 2L chromosomes.

Three series of chromosome rearrangements designated as dp^{hx}, sc^{19-x} and cl^{hx} were induced by X-ray irradiation (4000 R) of Oregon R and $T(1;2)sc^{19}$ males and then isolated by means of Dp(2;2)B3 de dp cl duplication; the first two series as well as Df(2L)M11 and Df(2L)M-z^B were balanced over Dp(2;1)B19 (Reuter & Szidonya 1983; a detailed description rearrangements and induction and their genetic analysis will be given elsewhere). Heterozygotes for Batumi L wild stock were used in electron microscopic (EM) mapping of deficiencies. Techniques of squashed chromosome preparations for EM have been described earlier (Semeshin et al. 1979). Revised Bridges' maps (Lindsley & Grell 1968) and EM data obtained on Batumi L were used for determination of rearrangement limits.

In the 24AF region Bridges described 30 bands including 8 doublets (Figure 1a). Some of the doublets, such as 24A1-2, D1-2, D5-6, F1-2, F6-7 and the 24C9, D3 and F3 thin bands were described by Saura (1980). However, in our EM studies all the doublets are visible as single bands; the 24B3, C6, C9, D3, F3, F5 and F8 thin bands were not detected (Figure 1b-e). Contradicting to Saura's data we could not visualize 4 bands in the 24F3-8 interval. In this region there are 2 zones with the diffused, slightly puffing material which are adjacent to the 24F1-2 and 25A1-4 thick bands (Figure 1f) and actively incorporates ^{3}H-uridine (Zhimulev & Belyaeva 1974). We observed the 24E2' thin band which was not described earlier (Figure 1e,f,g). Altogether, 24 bands were found in the 24 section of 2L chromosome.

The 25AC region was mapped earlier (Semeshin et al. 1985). As for the EM map of the 25DF region, it coincides with that of Bridges as well as with Saura's data, if Bridges' doublets are considered as single bands; the 25D1-2 "doublet" consisting of two separate bands is an exception (Figure 1f). The complete EM map of the 24 and 25 sections of the 2L chromosome is presented in Figure 6.

Df(2L)dp^{h19}. As is seen from Figure 1g the deficiency presumably deletes the 24F1-2 to 24F6-7 region. We also can not exclude the possibility that the proximal and distal breakpoints are located in the left edge of 25A1-4 complex and in the 24F1-2 band, respectively, and the remaining parts of these bands are fused.

Df(2L)M-z^B. According to Bridges' data this deficiency deletes the 24E2-F1 to 25A1-2 region (Lindsley & Grell 1968). However, Duttagupta & Dutta Roy (1984) consider that the 25A1-2 band remains intact and is not affected by the deficiency. EM data clearly show that the Df(2L)M-z^B deletes the 24E1-2 to 24F6-7 region (Figure 1h), but its proximal breakpoint can not be determined accurately. Because of the underreplication and the break in normal chromosomes the appearance of the 25A1-4 complex is rather variable (Figure 1b-f; see also Zhimulev et al. 1982) which often looks like a wedge. Similar pictures are observed in the chromosomes with deficiency (Figure 1h). However, we can not exclude that the left part of the 25A1-4 complex is removed and therefore attribute it to the uncertainty of mapping.

Df(2L)dp^{h28} deletes the 24D8 to 24F6-7 region (Figure 2a). We are not sure whether the 24D7 thin band is retained and the part of the 25A1-4 band is deleted; this material is attributed to the uncertainty of mapping.

Df(2L)M11 clearly deletes a part of the 25A1-4 complex because of the different thickness of the band in normal and deleted homologues (Figure 2b). The distal breakpoint of the deficiency is located just to the right of the 24D5-6 band (Figure 2c). Thus, the 24D8 to 24F6-7 region and the left part of the 25A1-4 band are included in the deleted material.

Df(2L)ed dp^{h1}. This deficiency also removes the left part of the 25A1-4 band (Figure 2d). Figure 2e shows clearly that the 24C3 band is preserved. Therefore, the deficiency deletes the region from the 24C5 band to the left part of the 25A1-4 complex. The 24C4 thin band is attributed to the uncertainty of mapping.