

Once the delayer effect of Butyrate had been shown, the following step was to compare the results of overfeeding experiments in crowded media (70 larvae in 0.5 ml. of Lewis' medium) with those of non-crowded (70 larvae in 5 ml. of Lewis' medium) supplemented with Sodium Butyrate. Only two doses of Butyrate were chosen for this kind of experiment: 50 mM and 100 mM which are those doses judged most suitable for the effect being sought. A total of five replicae were made. Table 2 shows the overfeeding in crowded media which served as control for the overfeeding in non-crowded media supplemented with 50 mM and 100 mM of Sodium Butyrate. The times of overfeedings were 8th, 10th, 12th, 14th and 16th day from the seeding day. In Table 2 larval stop is evident from the regression analysis. As regards total survival, the 50 mM concentration shows better survival than the crowded cultures, the opposite being true for the 100 mM concentrations. The regression of outer mean development over overfeedings shows larval stop in both concentrations, 50 mM and 100 mM, though the development at 50 mM is closer than 100 mM to crowded conditions.

Altogether the results reveal that Sodium Butyrate mimics quite accurately the result obtained in crowded cultures with respect to larval stop, delayed development and survival. Butyrate is known to inhibit cellular deacetylases of histones leading to an active state of chromatin (Weisbrod 1982). Thus, in one way or another the phenomenon of larval stop must be related to the regulation of gene expression, probably in relation to the genes responsible for Juvenile hormone and Ecdysone production which are controlling all the development.

References: Botella, L.M., A.Moya & J.L.Mensua 1983a, DIS 59:23-24; Botella, L.M., C. Gonzalez & J.L.Mensua 1983b, EDRC, Cambridge; Mensua, J.L. & A.Moya 1983, Heredity 51:347-352; Moya, A. & J.L.Mensua 1983, DIS 59:90-91; Weisbrod, D. 1982, Nature 297:289.

Bouletreau, M., P.Fouillet, E.Wajnberg and G.Prevoist. University of Lyon, France. A parasitic wasp changes genetic equilibrium in *D.melanogaster* experimental populations.

Parasitism has long been suspected to be involved in genetic equilibrium and polymorphism of natural populations (Day 1974; Clarke 1979; Price 1980). However the lack of experimental evidence, at least for animal populations, makes this hypothesis rather speculative.

We compared the evolution of the allelic frequency at the sepia locus in experimental populations of *D.melanogaster* either free of parasites, or constantly kept under parasitic pressure by the larval endoparasite *Leptopilina boulandi* (Nørdlander 1980).

The cage populations were of the overlapping generations type, with a weekly introduction of four cups each containing 25 gm of fresh yeast medium (David & Clavel 1965), and a turnover based on a two weeks periodicity. Wild *Drosophila* and parasite strains originated from Tunisia. The mutant stock sepia has been kept under laboratory conditions for many generations.

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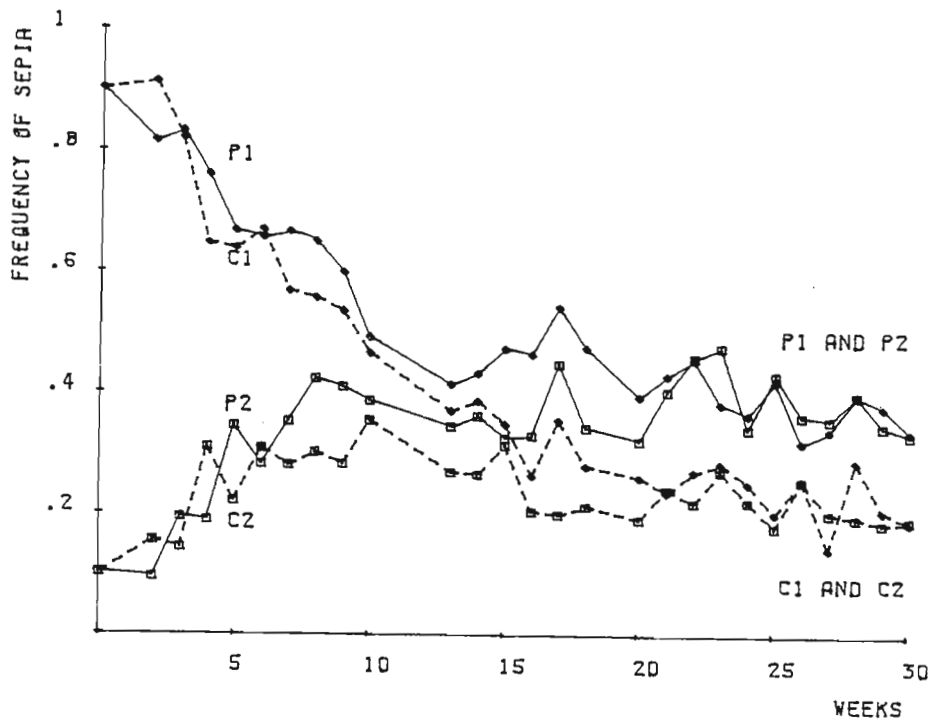


Fig. 1. Allelic frequencies at the sepia locus in control cages (C1 & C2) and in parasitized ones (P1 & P2).

Four cages were initiated simultaneously. The frequency of the recessive allele *se* was .9 in cage C1 (control) and P1 (parasitized); .1 in cages C2 and P2. Eggs were sampled weekly in each cage and after proper development, emerging flies were examined and the allelic frequency estimated by the square root of homozygous *se/se* frequency.

From the second week onwards, 200 *Leptopilina boulardi* couples were weekly introduced in cages P1 and P2. The biology of this wasp is very similar to that of its relative, *L.heterotoma* (= *Pseudeucoila bochei*). (see Van Lenteren 1976): females lay their eggs inside late 1st or early 2nd instars of *D.melanogaster*. Parasitized larvae grow up and pupate. At 25°C adult wasps emerge from the host's puparium on day 18 or 20 after. Since the developmental time of the parasite widely exceeds the fortnight's stay of cups in cages, no parasite could emerge inside cages and the weekly introduction of a new batch of adult parasites in cages P1 and P2 ensured a constant level of infestation all over the experiment.

Figure 1 shows the genetic evolution of the four experimental populations. Control populations C1 and C2 show a typical convergent evolution towards a .20 frequency equilibrium of the *se* allele, which is a classical value (Anxolabehere 1976). Parasitized cages P1 and P2 also show a convergent evolution. They reach their genetic equilibrium at the same time as control cages do, but the allelic frequency of *se* is much higher: .35. This striking difference obviously results from the presence of parasites in cages P1 and P2. The 1.8 fold increase in the equilibrium frequency of the less fitted allele demonstrates the possibility for a parasite, here a "parasitoid", to strongly affect the genetic makeup of the host population.

Further experiments are being carried out to clarify the underlying mechanisms. Preliminary results suggest that they are more complex than a trivial preference of the parasite for hosts of a given genotype.

References: Anxolabehere, D. 1976, *Evol.* 30:523-534; Clarke, B. 1979, *Proc.Soc.Lond.* 205:453-474; David, J. & M.F.Clavel 1965, *Bull.Biol.Fr.Belg.* 99:369-378; Day, P.R. 1974, Freeman San Francisco 238 pp.; Nørðlander, G. 1980, *Ent.Scand.* 11:428-453; Price, P.W. 1980, Princeton University Press 237 pp.; Van Lenteren, J.C. 1976, *Neth.J.Zool.* 26:1-83.

Casares, P. Universidad de Oviedo, España.  
Interspecific inhibition between *D.melanogaster* and *D.simulans* during oviposition process.

The purpose of this communication is to present a new oviposition behaviour observed in competition studies. One of the aspects studied in my doctoral thesis ("Competencia interespecifica entre *D.melanogaster* y *D.simulans*", unpublished) was to determine if the female

fecundity of one of these species could be modified by the presence of virgin females of the other species. To achieve this, male and female virgins of both species were separately aged to 5 days. Then, groups of pairs of each species were mated and later the males were discarded. With these newly mated females, two experimental units were achieved. In experiment-1, four tests were simultaneously initiated with the following females per vial: Test-M, with 8 mated *melanogaster* females; Test-M(S), with 8 mated *melanogaster* females + 8 virgin *simulans* females; Test-S, with 8 mated *simulans* females; Test-S(M), with 8 mated *simulans* females + 8 virgin *melanogaster* females.

The females were allowed to lay eggs for 24 hours in vials filled with standard baker's yeast medium. Then, the 8 or 16 females were transferred to vials with fresh food. After 48 hours, the females were individually assessed for fertility. Any replication with dead or sterile females was discarded. The number of eggs laid throughout the period 0-24 hrs (first vial) and 24-48 hrs (second vial) were recorded and likewise, the number of pupae and adults produced. All experiments were carried out at 21.5°C and constant light. The results are shown in Table 1.

In *D.melanogaster*, no different fecundity was found between tests M and M(S), that is, the presence of virgin females of *D.simulans* did not affect the *melanogaster* oviposition process in a two day period. In *D.simulans*, however, a remarkable reduction of fecundity was apparent when virgin *melanogaster* females were present in the vial. This inhibition of laying makes the progeny of test S(M) 83% of the progeny obtained in test S. Undoubtedly, this inhibitory behaviour during oviposition must be originated through some effect derived from the presence of virgin females. A possible objection to this could be the different adult density of tests S and S(M) with 8 and 16 females, respectively. This was solved