PARTLY COLORED LARVA, AN AUTOSOMAL RECESSIVE LEthal MUTATION IN THE FLOODWATER MOSQUITO Aedes vexans

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ABSTRACT. A first mutation in a laboratory strain of the floodwater mosquito *Aedes vexans* is described. *Partly colored larva* (pel) is inherited monofactorially as a recessive autosomal lethal, causing only dark coloration of the head capsule, the saddle on abdominal segment X and the respiratory siphon during the 4th larval stage. All mutants die in the 4th larval (49%) or pupal stage. The sex-linked mutant *white eye* (w) was used in crosses to show autosomal inheritance.

INTRODUCTION

Many mutations affecting larval color pattern in mosquitoes have been described. Some mutants concerning fat body color have been observed in *Culex pipiens* Linn. (Laven 1957) or in *Aedes aegypti* Linn. (Craig and Gillham 1959). Mutations altering larval epidermal and fatty tissue color have been isolated in *Culex pipiens* (Laven and Chen 1956, Dennhöfer 1973, McClendall 1978), in *Culex tarsalis* Coq. (Apperson 1979), in *Anopheles quadrimaculatus* Say (Seawright and Anthony 1972) and in *Anopheles albimanus* Wied. (Rabban et al. 1976, Seawright et al. 1984).

In the floodwater mosquito *Aedes vexans* Meigen, about which genetic information is rare, a mutant affecting epidermal color, *partly colored larva* (pel), has been found. The subject of this paper is the study of the inheritance of that color mutant with respect to the only other genetic marker so far known in *Aedes vexans*, *white eye* (w, Friederich and Deschner, unpublished data).

MATERIALS AND METHODS

Partly colored larvae were originally discovered in experiments involving inbreeding of a laboratory colony of *Aedes vexans* mating in rather small cages (46 × 37 × 42 cm). The adults were normally maintained at 24 ± 1°C, 75± 8% RH and under a controlled lighting cycle (LD 16:8). Females were offered blood from a human host and isolated individually for oviposition.

Eggs of each ovipositing female were stored on filter paper in glass petri dishes. To get 100% egg hatch, eggs were first stored for 1 month at 20°C, transferred for 3 months to 5°C and then stored for 2 weeks at 25°C (Friederich 1983) before application of the hatching stimulus (vacuum method modified after Gjul- lin et al. 1941).

The ensuing batches of larvae were cultured as single families. Families containing mutants were saved and the wild type sibs were massed to produce the next generation. This process of selection was repeated in each generation and according to the mode of inheritance (see below) the mutant was so maintained by heterozygote × heterozygote matings.

The *white eye* (w) mutant used represents a sex-linked recessive trait with a distance of 16.21 ± 7.16 map units to the sex determining factor (Friederich and Deschner, unpublished data).

The statistical procedures of Mather (1957) were applied on single families as well as on the data obtained by pooling all the families.

RESULTS AND DISCUSSION

The *pel* mutant occurred spontaneously during inbreeding of the laboratory strain. *Pel/pel* homozygotes were only detectable in the 4th larval stage. During the 4th larval stage 49% of the mutants died, the remainder perished as pupae. No *pel/pel* adults have been observed. Therefore, no homozygous stock was available.

Figure 1 shows the differences in epidermal pigmentation between mutant and normal lar-
vae. Some regions of pcl larvae, like the head capsule, the saddle on abdominal segment X and the respiratory siphon, contain more pigment than normal. Other regions, like the sternites and tergites, the setae of the thorax and abdomen and the tracheae, seem to have less pigment than normal. All these differences could also be observed on exuviae from molted 4th stage larvae. Pleiotropically, white eye influences eye color and also the color of the fat body which contains lesser dark pigment than normal larvae.

Crosses were conducted to establish the mode of inheritance (Table 1). In the progeny of reciprocal crosses of pcl heterozygotes with wild type or white eye (F1) the phenotype pcl was not expressed, but appeared in the F2 (Crosses B-E) and subsequent generations (Cross A). Moreover no sex linkage could be observed in the crosses with the sex-linked recessive mutation white eye (w; Cross D and E, Table 1). Therefore pcl is a recessive autosomal lethal. The phenotype is only expressed in the 4th larval stage in sclerotized regions. Penetrance is complete and there is very little variation in expressivity. The lethality may also occur in the prelarval stages because an overall tendency for a slightly lower than expected number (25%) of partly colored larvae was observed. Out of 994 4th larvae in 26 families, 23.5 ± 3.5% larvae expressed the pcl phenotype.

Pcl shows no similarity to other recessive autosomal lethal mutants reported in mosquitoes: mel in Culex pipiens (Laven and Chen 1956, Tadano and Barr 1975), b in Anopheles stephensi (Mason and Davidson 1966), bl in An. quadrimaculatus (Seawright and Anthony 1972) or bl in An. albimanus (Rabbani et al. 1976).

The phenotype of the sex-linked factor Black larva (Bl) in Culex pipiens showing complete dominance in the male and incomplete dominance in the female (Vandehey 1967) somewhat resembles pcl. But in Bl only an increased

### Table 1. Crosses indicating that partly colored larvae (pcl) in Aedes vexans is recessive, autosomal. All larvae showing pcl phenotype died in the 4th larval or pupal stage. (w = white eye; m, M = sex determining factors.)

<table>
<thead>
<tr>
<th>Cross*</th>
<th>Parental genotypes</th>
<th>Number of families**</th>
<th>Progeny** phenotype in families with pcl</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Parental genotypes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Female × Male</td>
<td>with pcl</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>pcl+ × pcl</td>
<td>12</td>
<td>293 — 90 —</td>
</tr>
<tr>
<td></td>
<td>pcl × pcl</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>pcl+ × pcl</td>
<td>9</td>
<td>42 — 13 —</td>
</tr>
<tr>
<td></td>
<td>pcl × pcl</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>pcl+ × pcl</td>
<td>7</td>
<td>81 — 23 —</td>
</tr>
<tr>
<td></td>
<td>pcl × pcl</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>m pcl × m pcl</td>
<td>26</td>
<td>230 81 75 28</td>
</tr>
<tr>
<td></td>
<td>m pcl × m pcl</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>m pcl × m pcl</td>
<td>6</td>
<td>22 10 4 2</td>
</tr>
<tr>
<td></td>
<td>m pcl × m pcl</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

* Crosses B–E yielded no pcl larvae in F1, but in F2 after inbreeding of F1.

** F1–F4 in Cross A, F2 in crosses B–E.

As is evident from the data obtained from crosses A-E (Table 1) and from the statistical analysis (Table 2), partly colored larva shows a recessive monofactorial inheritance. The mutant pcl demonstrates no sex linkage since no significant distortion of sex-ratio was observed in the surviving sibs of families with pcl/pcl larvae.

### Table 2. Statistical analysis of data from Table 1.

<table>
<thead>
<tr>
<th>Cross</th>
<th>pcl+</th>
<th>w</th>
<th>pcl</th>
<th>w pcl</th>
<th>Expected segregation</th>
<th>X²</th>
</tr>
</thead>
<tbody>
<tr>
<td>A–E*</td>
<td>759</td>
<td>—</td>
<td>235</td>
<td>—</td>
<td>3:1</td>
<td>0.9068 (P = .35)</td>
</tr>
<tr>
<td>D, E</td>
<td>252</td>
<td>91</td>
<td>79</td>
<td>30</td>
<td>9:3:3:1</td>
<td>0.9839 (P = .81)</td>
</tr>
</tbody>
</table>

* Without regard to eye color.
pigmentation of the larval head capsule is observed. Also contrary to pcl, in Bl all larval instars are pigmented. Bl is no lethal factor; however it shows a pleiotropic effect, namely, a decreasing developmental rate.

The recent description of a new, recessive, autosomal mutation in Anopheles albimanus (Seawright et al. 1984), amber (am), corresponds very much to that of pcl. Amber in An. albimanus appears like pcl in the 4th larval stage. Moreover the head capsule and the saddle of abdominal segment X has another color as in normal larvae. Am however lacks the recessive lethal effect present in pcl.

Crossings of pcl with translocations and inversions have to be made in the future to assign the pcl locus to one of the two autosomes. It should be of value in any genetic experimentation.

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References Cited
