Studies on *Thrips tabaci* Lind.: A New Stage in its Life Cycle

Incubation of *T. tabaci* eggs outside caster-oil leaves led to the discovery of a new stage in its life cycle and the correction of the incubation period[1]. Studies on duration of the egg and prenymph stages are therefore planned in this paper, in association with a brief description of egg, prenymph, process of hatching and the way the prenymph escapes from the egg pocket in the plant leaf.

Materials and methods. Eggs were extracted from the leaf by triangular razer blade inserted in a wooden handle, and were then placed in petri dishes for incubation at 22 ± 1 °C and 100% relative humidity. Eggs incubated inside cotyledons were obtained by confining caster oil seedlings with the adult thrips in containers designed by the authors (under publication).

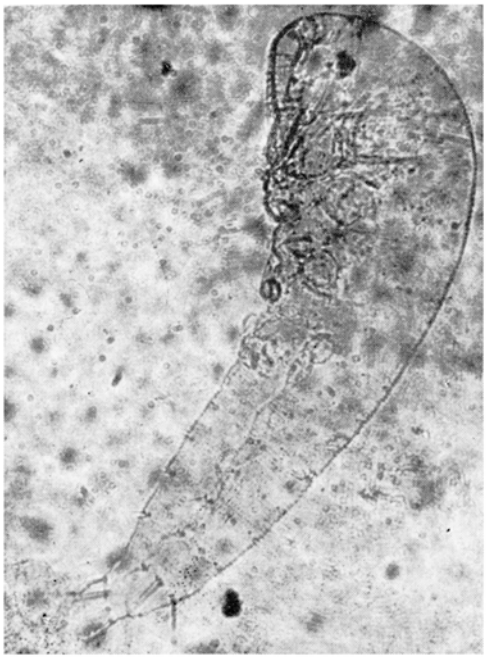

Fig. 1. The new stage in life cycle of *T. tabaci*.

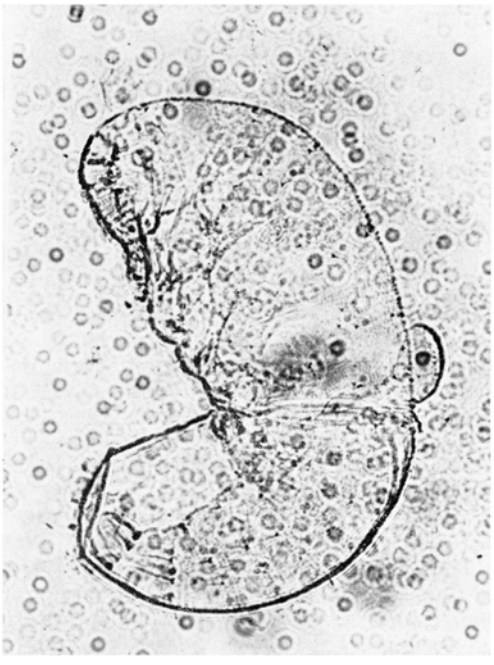

Fig. 2. Process of hatching by rupturing the upper side of the egg.

Table 1. Duration of egg and prenymph stages in and outside the plant leaf at 22±1°C and 100% relative humidity

No. of eggs	Duration of egg stage (days)		Duration of prenymph stage (days)	
	In plant	In vitro	In plant	In vitro
1	4	4.5	1	2
2	4	4	1	2
3	4	4	0*	2
4	4	4	1	2.5
5	3	5	2	1.5
6	3	4	1	1.5
7	4	4	2	2.5
8	4	4	1	1.5
9	4	4	0	1.5
10	5	4	1	2

For incubation period: 3.9 ± 0.54 in plant leaf and 4.15 ± 0.32 days in petri dishes.
For duration of prenymph: 1.0 ± 0.118 in plant leaf and 1.90 ± 0.38 days in petri dishes.

* Less than 24 h, the observation period.

[1] A. A. W. A. GAWAAD and A. Y. SHAZLI, in preparation.

Results. The egg, kidney-shaped at the time of oviposition, elongates and acquires a different shape 24 h after its deposition. The hatching end of the embryo is bent in line with the curvature of the prenymph back.

The embryo does not secrete any fluid for digesting the chorion, as no change in the chorion colour takes place during occlusion. The chorion rim is elastic, because it is not torn during hatching (Figure 2).

The prenymph bears, in addition to 2 pink eyes, 2 circles of long bristles which characterize the immature stages of *T. tabaci*, especially the pre-pupa and pupa (Figures 1 and 3). Probably it does not feed as it lived normally on filter papers treated with distilled water in petri dishes.

Table 2. Hatchability percentage of *T. tabaci* eggs in petri dishes at 22+1°C and 100% relative humidity

No. of eggs	No. of eggs hatching	hatch-ability (%)	No. of eggs	No. of eggs hatching	hatch-ability (%)
6	6	100	13	9	69.23
14	13	92.86	11	10	90.91
10	10	100	10	7	70
11	9	81.82	17	14	82.35
11	9	81.82	10	9	90

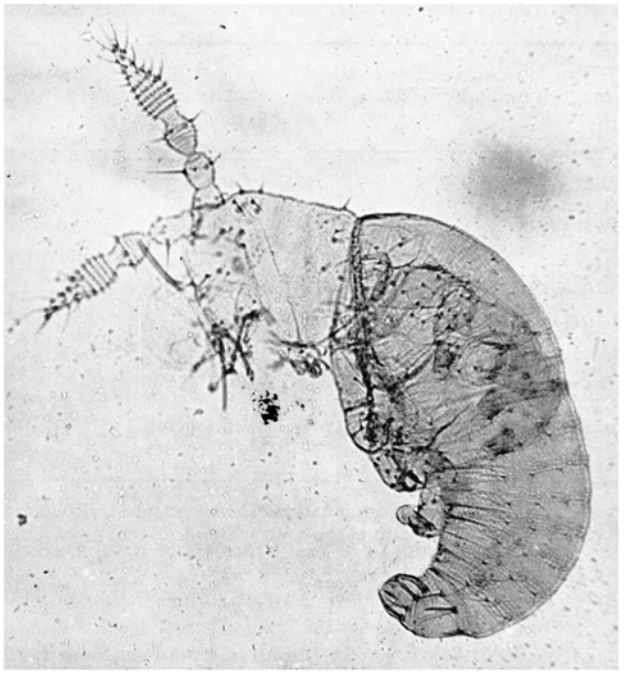

Fig. 3. The prenymph in moulting; antennae, head and mouth parts of nymph are freed of casted skin.

The function of this stage is restricted in reaching the leaf surface. It pushes its way through the leaf tissue by wiggling as it gradually pops up the leaf surface. When it frees its head and thorax, it moults along the dorsal midline; the first instar actively surfaces itself after freeing its legs one by one. It presses with the legs on the leaf surface to draw the rest of its body outside the egg pocket. Once the antennae and legs are normally spread, or inflated by the insect blood pressure, it moves very fast, leaving its exuvium in the leaf pocket.

Duration of egg and prenymph stages. The periods occupied by the egg and prenymph stages according to observations in plant leaves and in petri dishes are shown in Table I, with 85.9% of hatchability in petri dishes in Table II. In petri dishes the prenymph lasts for a longer period (1.9 $\pm$ 0.3 days) than in the leaf tissue (1 $\pm$ 0.118 days). Some plant factors may be held responsible for shortening embryonic and prenymphal development i.e. thegmotactic response, osmotic pressure, nutrients or plant hormones.

Résumé. La durée d'une phase nouvelle de laprénymphe de *Thrips tabaci* et le mécanisme d'éclosion de l'oeuf est décrit.

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Effect of Heat on Serum Thyroxin and Thyrotropin and its Modification by Dihydrotachysterol[1]

The fact that injections of 350–500 mg/kg/d dihydrotachysterol (DHT) elevate the serum level of thyrotropin (TSH) and thyroxin (T-4) in rats kept at room temperature has been reported by Tal and Sulman[2]. The present work was undertaken in order to compare the serum levels of TSH and T-4 in rats kept at room temperature (22 °C) and at higher temperatures (34 °C and 37 °C).

Materials and methods. 3 groups of 24 male albino rats of the Hebrew University 'Sabra' strain, 21-day-old, weighing 40–45 g, were housed 6 per cage in 3 rooms at 22 $\pm$ 1 °C, 34 °C and 37 °C $\pm$ 1 °C respectively. Care was taken to assure identical temperatures in all cages which were kept on shelves. The rats received standard laboratory pellets and water ad libitum. Group 1 (12 control rats at 22 °C) was injected i.p. daily from day 21 to 42 with 0.1 ml olive oil. Group 2 (12 DHT rats at 22 °C) received 500 µg/kg/day of DHT in 0.1 ml olive oil i.p. during the same period. Group 3 (12 control rats at 34 °C) received the same treatment as group 1. Group 4 (12 DHT rats at 34 °C) received the same treatment as Group 2. Group 5 (12 control rats at 37 °C) served as control to group 6 (12 DHT rats at 37 °C). They received the same treatment as groups 1 and 2, respectively. Groups 1–4 were sacrificed on the 42nd day of their life, while groups 5–6 were killed at an age of 25 days. This early date of killing was made necessary by the control rats (group 5) dying after that time, whereas group 6 survived for normal periods. Blood samples were taken from all rats and tested for TSH (McKenzie method[3]) and T-4 (Tetralute-Ames method[4]). Calcium was assayed by the atomic absorption method.

Results. The Table shows that TSH and T-4 levels in the DHT-treated rats kept at room temperature (22 $\pm$ 1 °C) rose significantly above those of the control rats kept at the same temperature: TSH increased from 1.0 mU/ml to 2.1 mU/ml and T-4 from 2.5 µg/100 ml to 6.0 µg/100 ml (groups 1–2).

Exposure of control rats (group 3) to a higher temperature (34 $\pm$ 1 °C) increased TSH levels slightly (from 1.0 mU/ml to 1.3 mU/ml), but caused a marked decrease in T-4 levels (from 2.5 µg/100 ml to 1.1 µg/100 ml). Mortality and weight loss of these rats was rather high (60%). The decrease in the T-4 level and death could be completely counteracted by DHT injections (500 µg/kg/day group 4).

Exposure of control rats to still higher temperatures (37 $\pm$ 1 °C – group 5) caused the death of all animals within 5–6 days. Blood samples of these rats taken on the 4th day of exposure to 37 °C revealed very low levels of TSH (0.1 mU/ml) and of T-4 (0.42 µg/100 ml), lower than those of rats reared at 34 °C. This reaction was completely abolished in group 6, where daily treatment with (DHT 500 µg/kg/day i.p.) counteracted this fall and prevented death.

Discussion. The effect of DHT on T-4 secretion was studied by Djojosoebagio and Turner[5]. Early in 1971 we suggested[2] that the enhanced TSH and T-4 secretion by DHT runs via the hypothalamus-pituitary-thyroid axis rather than via the thyroid directly. On the other hand,

[1] This study was generously aided by a grant from the Dr. Gerald Kaplan family, Kfar Shmaryahu (Israel).

[2] E. Tal and F. G. Sulman, Neuroendocrinology, in press (1971).

[3] J. M. McKenzie, Endocrinology *62*, 865 (1958).

[4] B. E. P. Murphy, C. F. Pattee and A. Gold, J. clin. Endocrin. *26*, 247 (1966).

[5] S. Djojosoebagio and S. Turner, Proc. Soc. exp. Biol. Med. *116*, 1099 (1964).