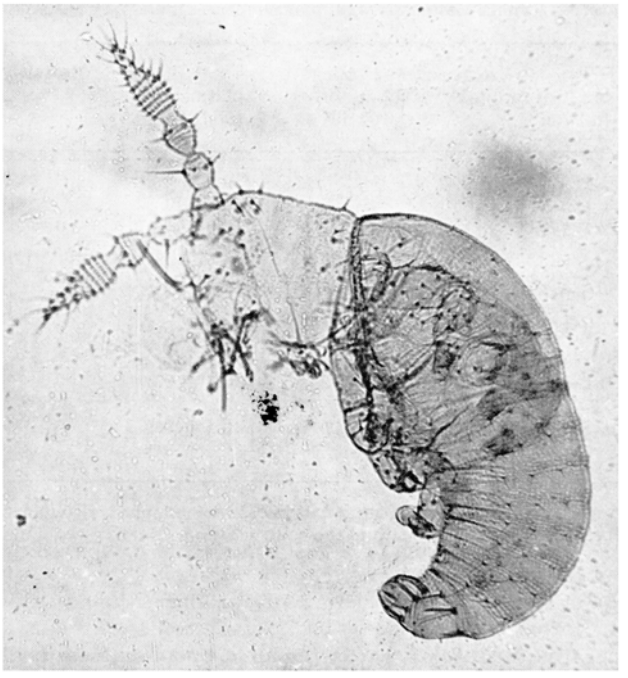

Fig. 3. The prenymph in moulting; antennae, head and mouth parts of nymph are freed of casted skin.

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,

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Effect of heat stress and DHT on thyrotropin (TSH) and thyroxin (T-4) serum level

Group No.	Treatment DHT and temperature	Body wt. on day of start (g)	Body wt. on day of end (g)	TSH levels of serum (mU/ml)	T-4 levels of serum (μg/100 ml)	Total plasma Ca^{++} (mg/100 ml)	Mortality (%)
1	Control 22 °C	44 ± 5	120 ± 6	1.0 ± 0.04	2.5 ± 0.1	10.3 ± 0.1	0
2	DHT i.p. 22 °C 500 μg/kg/day	45 ± 5	125 ± 5	2.1 ± 0.06	5.94 ± 0.029[a]	10.6 ± 0.1	0
3	Control 34 °C	45 ± 5	70 ± 10	1.3 ± 0.2	1.1 ± 0.03[a]	10.5 ± 0.2	60[a]
4	DHT i.p. 34 °C 500 μg/kg/day	45 ± 5	110 ± 5	1.9 ± 0.3	2.3 ± 0.12[a]	10.5 ± 0.1	0
5	Control 37 °C	45 ± 5	47 ± 5	0.1 ± 0.2[a]	0.42 ± 0.02[a]	10.4 ± 0.2	100[a]
6	DHT i.p. 37 °C 500 μg/kg/day	45 ± 5	50 ± 3	0.5 ± 0.1[a]	0.92 ± 0.05[a]	10.5 ± 0.1	0

Each of the 6 groups consisted of 12 male rats. Group 1–4 was 21 days old when treatment started and 42 days old when stopped. Group 5-6 was 21 days old when treatment started and had to be killed after 4 days because of imminent death of the control group. (Means ± S.E.M.). [a] Significant ($P < 0.001$).

BALOGH et al.[6] and BAJUSZ[7] found high BMR levels in rats kept at temperatures above 33 °C. Our findings of low T-4 levels at high temperatures may explain the high mortality at these temperatures, as the rats cannot then cope with their high metabolic rate while lacking enough T-4 for negative feedback. It is, therefore, suggested that a certain optimal level of T-4 is a prerequisite for survival of animals at high temperatures. This view is also based on the rather high T-4 levels in the control rats kept at 34 °C (group 3), which could adapt themselves to heat, whereas the control rats kept at 37 °C (group 5) died early showing extremely low T-4 levels. No significant difference could be detected between the TSH levels at 22 °C and 34 °C (groups 1 and 3), while a highly significant drop in plasma TSH was noticed at 37 °C (group 5) as compared with the former. This drop in TSH at 37 °C was partly set off by DHT injections (group 6). The quantities of DHT injected in this experiment (500 μg/kg/day) did not affect plasma Ca levels. Still, DHT increased both TSH and thyroxine levels at all 3 experimental temperatures tested. This increase was significant in spite of the steep drop in blood TSH and thyroxine obtained at 37 °C. This shows that the thyroxine-TSH negative feedback is partly impaired by an ambient heat of 34 °C and completely abolished at 37 °C.

Zusammenfassung. Dehydrotachysterol (DHT 500 μg/kg/24 h) erhöht den TSH- und T-4-Spiegel in Rattenserum bei normalen und hohen Temperaturen, ohne den Calcium-Spiegel zu verändern, und verhindert den Tod von Ratten, die bei 34° oder 37 °C gehalten werden. Das Überleben der Ratten nach DHT-Injektionen erklärt sich durch erhöhte T-4-Produktion, die einen negativen «Feedback» auf den Hypothalamus ausübt und so die lethale Überproduktion von TSH verhindert.

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Effect of Decreased Dietary Protein upon Mammary Gland Growth in Rats

Experimental and clinical studies have shown that chronic and acute starvation, caloric restriction, vitamin and mineral deficiencies alter the function of endocrine glands. Restricted food intake resulted in reduced activity of the thyroid gland[1,2]. SINGH et al[3]. reported that decreased dietary protein, i.e., protein-free died and 5% protein diet, reduced thyroid hormone secretion rate (TSR) in rats. It has also been reported that absence of dietary protein caused decrease in FSH secretion which eventually affects estrogen and progesterone secretion. Several workers[4–6] have shown that thyroid hormone administered in intact rats or ovariectomized rats treated with estrogen and progesterone increased the amount of mammary gland growth. Estrogen, progesterone and prolactin are the main hormones essentially required for mammary gland development. In the present study, the effect of decreased dietary protein upon mammary gland growth by DNA estimation has been studied.

Materials and methods. 106 virgin female rats (approximately 70 days old) of the Sprague-Dawley Rolfsmeyer strain (purchased from Rolfsmeyer Co., Madison, Wis-

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