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Short Communications

Hyperthermia after intrahypothalamic injections of thyrotropin releasing hormone (TRH) in the pigeon[1]

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Summary. Thermoregulatory responses to intrahypothalamic injections of thyrotropin releasing hormone (TRH) were recorded from unanesthetized pigeons exposed to 6 °C, 20 °C and 32 °C. Our results suggest that TRH is a non-specific excitatory neuromodulator or neurotransmitter for heat production in the pigeon.

There is a considerable amount of evidence showing that TRH is distributed not only in the hypothalamus but also throughout the nervous system[3–5]. Besides stimulating the release of thyrotropin (TSH) from the adenohypophysis, TRH is also known to affect prolactin secretion[4,6].

Several studies have confirmed the thermoregulatory effects of TRH in mammals[3,4]. However, similar effects are not well known in birds. Intracerebral administration of TRH has been shown to elevate the body temperature (T_b) in the fowl by activating heat production and decreasing thermodispersive mechanisms[7].

The aim of the present study was to determine the effect of intrahypothalamic administration of a large range of different dosages of TRH on temperature regulation in the pigeon. In addition, the effects of season and various ambient temperatures (T_a) on the thermoregulatory responses were considered.

Materials and methods. Using pentobarbital anesthesia, a guide cannula was implanted stereotaxically[8,9] into the brain of the pigeon *(Columba livia)*, with the tip located either in the preoptic (PO/AH) area or in the posterior hypothalamus. 22 birds weighing 275–425 g were used in the study. 10 pigeons were used in November (group A), 5 with the guide cannula in the PO/AH area and 5 with the

Effects of intrahypothalamic injections of TRH on shivering in pigeons at ambient temperatures used in the winter (W) and in the spring (S)

T_a (°C)	Dosage (ng)	Season	Shivering (μV) Before injection	Maximum increase	
6	100	W	29.0±5.67	9.0±1.20	×
	200	W	31.5±8.89	12.0±1.99	×
	200	S	22.5±7.96	12.0±3.24	×
	500	W	27.1±7.14	13.7±3.46	×
	500	S	20.3±5.96	9.5±2.60	×
20	50	W	14.6±5.44	12.3±1.35	×
	50	S	11.8±3.07	7.5±1.85	
	100	W	21.3±5.53	18.8±6.20	×
	200	W	12.8±2.90	8.8±2.24	×
	200	S	7.8±3.71	14.5±3.62	×
	500	W	19.0±6.47	24.4±5.55	×
	500	S	7.8±3.12	11.7±2.21	×
32	100	W	0	11.8±5.08	×
	200	W	0	6.3±2.88	×
	200	S	0	4.8±0.95	×
	500	W	0	8.1±3.33	×
	500	S	0	4.3±1.03	×

Values are mean±SE. × Significant difference ($p<0.05$ or less) compared with corresponding controls (Student's t-test).

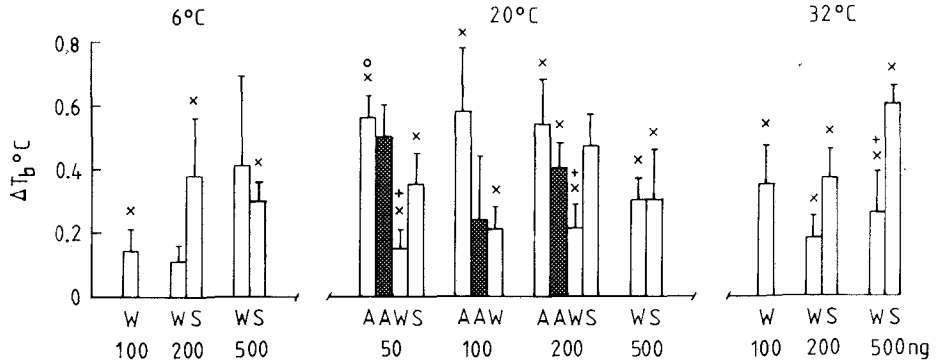

Figure 1. Mean maximum increases in the body temperature (T_b) after TRH (50–500 ng) in pigeons at different seasons (A = autumn, W = winter, S = spring) at ambient temperatures of 6 °C, 20 °C and 32 °C. Bars indicate SE of the mean. Open columns: responses to TRH in the anterior hypothalamus. Filled columns: responses to TRH in the posterior region in the autumn at 20 °C.
× Statistically significant difference from corresponding controls ($p < 0.05$ or less).
+ Statistically significant difference ($p < 0.05$ or less) for the same dosage between responses in the winter and in the spring.
○ Statistically significant difference ($p < 0.001$) for the same dosage between responses in the autumn and in the winter. Statistical analysis based on Student's t-test.

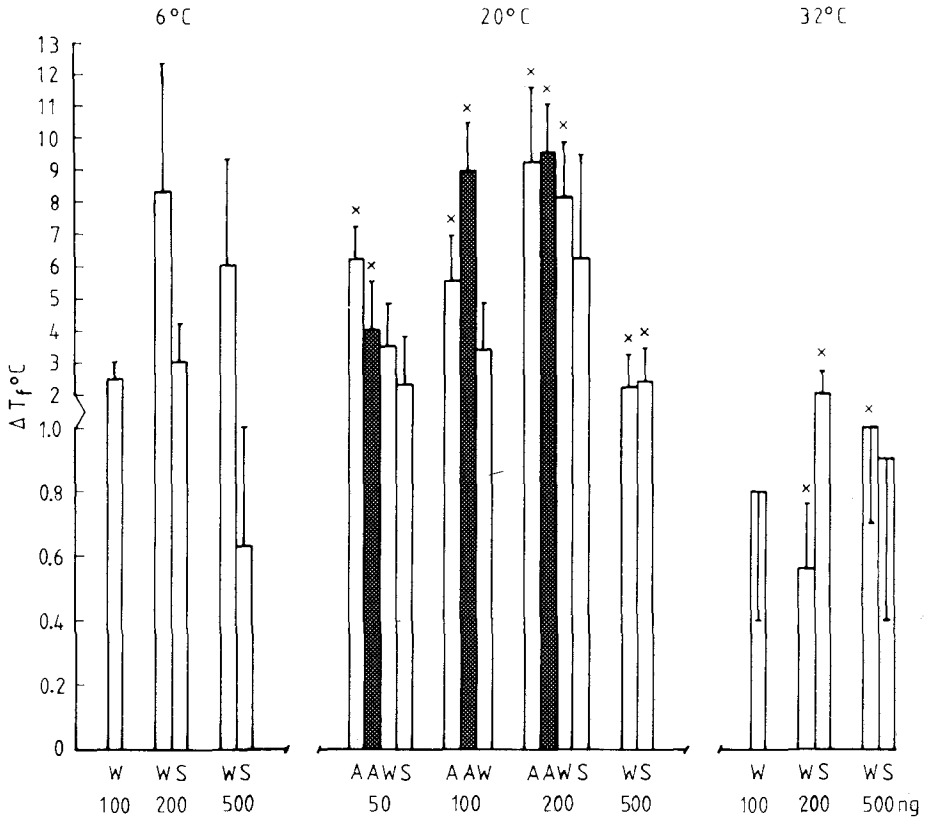

Figure 2. Mean maximum increases in the foot temperature (T_f) after TRH (50–500 ng) in pigeons at different seasons (A = autumn, W = winter, S = spring) at ambient temperatures of 6 °C, 20 °C and 32 °C. Bars indicate SE of the mean. Open columns: responses to TRH in the anterior hypothalamus. Filled columns: responses to TRH in the posterior region in the autumn at 20 °C.
× Statistically significant difference ($p < 0.05$ or less, Student's t-test) from corresponding controls.

cannula in the posterior hypothalamus. 8 pigeons were used in January-February (group W) and 4 in March-April (group S) with the cannula located in PO/AH area. The modified coordinates[8] were A 7.5–8.0 mm or 4.8–5.5 mm, L 1.2–2.0 mm and 9.0–10.0 mm below the surface of the skull. At least one week elapsed between the operation and the beginning of injections.

TRH (L-pyroglutamyl-L-histidyl-L-proline amide, Sigma) was dissolved in distilled water and injected in a volume of 1 µl. In November dosages used were 50, 100 and 200 ng at a T_a of 20 °C, in January-February 50, 100, 200 and 500 ng at T_a's of 6 °C, 20 °C and 32 °C and in March-April 50, 200 and 500 ng at T_a's of 6 °C, 20 °C and 32 °C. The thermoneutral temperature in the pigeon ranges from 24 °C to 30 °C[10]. Temperatures were measured 1.5 cm deep in the rectum and from tarsometatarsal area of the foot with Ellab NRM4 and NRM6 probes and an Ellab Z94-B recorder. Shivering was measured from the breast muscle with 3 monopolar stainless electrodes and a 2-channel signal processing system. After amplification, frequencies below 10 Hz and

above 500 Hz were filtered out and the EMG signal was passed into the integrator for rectification (time constant 10 sec). The averaged potential indicating shivering intensity was recorded with a Rikadenki (Tohshin) potentiometer. The direct EMG signals were monitored continuously with a Tektronix 502A dual beam oscilloscope. The details of the measurement procedures used in this study have been described earlier[11,12].

Results and discussion. As shown in the table the intrahypothalamic administration of TRH induced or potentiated shivering at all ambient temperatures tested. Shivering was stimulated within a few seconds and the maximum was attained in 2–6 min. This was followed by an elevated T_b (fig. 1). The foot temperature increased in all cases (fig. 2). No correlations in the thermoregulatory responses were seen among various dosages, ambient temperatures and injection sites, although a slight dependence was recorded for the seasons (fig. 1). Injections of 1 µl of distilled water did not induce any significant changes in the measured variables.

For the body temperature, our results are in agreement with those observed in the fowl[7]. Intrahypothalamic infusion of TRH (100–500 ng) was shown to increase body temperature of the fowl maximally by 0.8 °C. In the present study the rise in the foot temperature seems paradoxical since it suggests an increased peripheral heat loss. It is, however, in contrast with the responses obtained in the fowl[7].

It is now well known that intrahypothalamic microinjections of various putative neurotransmitters and neuromodulators cause hypothermia in the pigeon in most cases[13,14]. As shown in the present study, a hyperthermic response after TRH injections was always recorded. An explanation for hyperthermia may be drawn from the evidence which shows that TRH is an analeptic agent, at least in mammals, i.e. it has the ability to counteract the depressant effects of other drugs[5]. TRH increases muscle tone and motor activity and activates heat production mechanisms i.e. shivering and tremor[7]. Accordingly, the increase in shivering activity after injection of TRH is associated with the stimulation of excitatory neuronal pathways between cold-sensors and heat production effectors. This idea is supported by the fact that the rise in body temperature is independent of ambient temperature or dosage. A similar observation has been made in rats, where intracerebrally administered TRH increases body temperature independent of the dosage[15].

It was also of interest to note that the intensified shivering was always associated with an activation of a part of the heat loss effectors (vasodilation). The maximum foot temperature was recorded about 20 min later than maximum shivering. It is suggested that the primary function of TRH is to increase shivering and overall activity, followed by a compensatory response through the vasomotor tone to lower the body temperature.

Taken together, our results suggest that TRH is an non-specific excitatory neuromodulator or neurotransmitter in heat production in the pigeon.

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Olfactory receptor systems for sex pheromone mimics in the American cockroach, *Periplaneta americana* L.

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Summary. By application of a differential saturation EAG technique, the olfactory receptor system for compounds active as sex pheromones in the American cockroach was elucidated. The interaction of sex pheromone mimics with receptors responsive to a sex pheromone (periplanone-B) was revealed. As suggested by the single cell recording studies, the presence of sex pheromone receptors responsive specifically to sex pheromones (periplanone-A and -B) was shown, as well as the presence of general odor receptors which are functionally different from the sex pheromone receptors.

The visible sexual excitement behavior observed in the male American cockroach (*Periplaneta americana* L.) following exposure to the monoterpenoid sex pheromone mimic, (+)-trans-verbenyl acetate[2], and its more active analogs such as (+)-verbanyl propionate (VaP)[3] is indistinguishable from the behavior induced by the natural sex pheromones, periplanone-A (PA)[4] and periplanone-B (PB)[5]. However, the chemical structures[2–5] and the threshold dose values[3,6] required for inducing the sexual behavior are quite different between the mimics and the pheromones. This fact stimulated us to question whether the mimics act upon the same receptors as the natural pheromones. The present study was undertaken to examine the receptor system associated with the monoterpenoid sex pheromone mimics by applying a differential saturation electroantennogram (EAG) technique to male antennae of the cockroach. This technique, developed for beetles[7,8] and moths[9], involves continuous antennal exposure to a high concentration of a compound through an airstream (primary odorous stimulation) until the insect receptors are