



Title	Effect of Thyroid Hormone on Thermal Sweating
Author(s)	Matsumoto, Takaaki; Kosaka, Mitsuo; Yamauchi, Masaki; Ohwatari, Nobu; Yang, Guo-Jie; Nakamura, Koichi; Yamashita, Shunichi; Izumi, Motomori; Nagataki, Shigenobu
Citation	熱帯医学 Tropical medicine 30(3). p225-231, 1988
Issue Date	1988-09-30
URL	http://hdl.handle.net/10069/4532
Right	

This document is downloaded at: 2019-02-16T14:07:03Z

Effect of Thyroid Hormone on Thermal Sweating*

Takaaki MATSUMOTO¹, Mitsuo KOSAKA¹, Masaki YAMAUCHI¹,
Nobu OHWATARI¹, Guo-Jie YANG¹, Koichi NAKAMURA¹,
Shunichi YAMASHITA², Motomori IZUMI² and Shigenobu NAGATAKI²

1 *Department of Environmental Physiology, Institute of Tropical Medicine,
Nagasaki University, 12-4 Sakamoto-machi, Nagasaki 852, Japan*

2 *First Department of Internal Medicine, Nagasaki University
School of Medicine, 7-1 Sakamoto-machi, Nagasaki 852, Japan*

Abstract: Hyperhidrosis with hyperthyroidism and dry skin with hypothyroidism are clinically well-known phenomenon, however, up to now there is no general agreement concerning the effect of thyroid hormone on thermal sweating based on the objective quantitative data. In the present study thermal sweating responses in the patients suffering from hyperthyroidism and hypothyroidism were examined by means of capacitance hygrometer-sweat capture capsule method which can determine much finer fluctuation of sweat rate. In environmental control chamber (25°C, 60% rh) local heat load (43°C water bath, 30 min) was applied on the lower legs to induce thermal sweating. Sweat-onset time after heat load in hyperthyroidism (n=5) and control group (n=9) were 10.6 ± 5.2 and 15.6 ± 5.0 min (Mean \pm SD), respectively. Total sweat volume induced by 30 min heat load in both group were 66.06 ± 26.24 and 47.95 ± 31.25 mg/capsule, respectively. Increase of oral temperature at sweat-onset time in both group were 0.20 ± 0.16 and 0.10 ± 0.08 °C, respectively. In the two patients suffering from hypothyroidism more than a few years no sweat was observed during 30 min heat load. In one case sweat-onset time was 56 min where 0.50°C rise in oral temperature, and in the other case no sweat was induced finally even though 0.90°C increase in oral temperature during 51 min heat load. In contrast remarkably short sweat-onset time and large amount of sweat were observed in the two thyroidectomized patients who stopped replacement therapy of triiodothyronine (T3) two weeks ago, although, whose thyroid function (T3, T4, FT4 and TSH) were revealed the same as the value of long-term hypothyroidism group. From the present results, following two suggestions can be drawn; one is thyroid hormone enhances thermal sweating, the others is thyroid hormone action at the intra-cellular level might be changed relatively slow compared to the regulation of thyroid hormone concentration in serum level. Further researches are necessary to clarify the effect of thyroid hormone on thermal sweating and the action site of thyroid hormone on sweating mechanism.

Key words: Hyperthyroidism, Hypothyroidism, Thermal sweating, Capacitance hygrometer-sweat capture capsule method

Received for Publication, July 22, 1988.

Contribution No. 2127 from the Institute of Tropical Medicine, Nagasaki University.

* Presented in the 65th Annual Meeting of Physiological Society of Japan, Wakayama, April 4-6, 1988.

INTRODUCTION

Although hyperhidrosis is one of the most valuable and important clinical symptoms of hyperthyroidism, there is very little research on sweating in hyperthyroidism and there is no general agreement concerning the effect of thyroid hormone on thermal sweating. The earlier reports (Krikler, 1966; Shanks *et al.*, 1969) were based on the subjective assessments or on the impression of the observers. More recently, Judith *et al.* (1972, 1973) estimated sweat rate by continuously monitoring total body weight loss and reported that sweat rate in thyrotoxic patients was little different from those of normal subjects at 29°C. They also examined sweating rate in experimental thyrotoxicosis induced by the oral administration of triiodothyronine for two weeks and found little increase at 29°C but significant increase at 34°C compared to control period. On the other hand, there is no report of thermal sweating in patients with hypothyroidism.

In the present investigation thermal sweating in the patients with hyperthyroidism and hypothyroidism was determined by using of capacitance hygrometer-sweat capture capsule method (Fan, 1987) which can make more accurate measurement of local sweat rate to further clarify the effect of thyroid hormone on thermal sweating.

MATERIALS AND METHODS

The subjects in this study were five untreated hyperthyroidism patients (3 males and 2 females, 23–45 years old), four untreated hypothyroidism patients (all female, 25–55 years old, 2 of them were suffering from Hashimoto's thyroiditis more than a few years and another 2 were thyroidectomized patients of thyroid cancer and their replacement therapy of triiodothyronine (T3) was stopped two weeks before experiment because of ¹³¹I therapy for metastasis of thyroid cancer) and 9 normal volunteers (all male, 20–40 years old). The thyroid hormone data of the subjects were summarized and shown in Table 1. All subjects of the control group were revealed normal thyroid function. In the hyperthyroidism group elevated serum concentrations of T3 (triiodothyronine) and T4 (thyroxine) and decreased TSH (thyroid stimulating hormone) were determined. The circulating hormone levels of short-term hypothyroid patients were almost equal to those of long-term hypothyroid patients; decreased T3 and T4 and increased TSH levels.

On the experimental day the subjects were submitted to sit on the chair quietly in the controlled climatic chamber (25°C, 60% rh) for 30 min to equilibrate to the temperature of the chamber. Attachment of sweat capture capsules onto the chest and abdominal skin were made. Ten minutes after the beginning of recording, heat load (43°C hot water, 30 min) was applied on bilateral lower extremities to induce thermal sweating. Local sweat rate of the skin covered by the capsule were measured continuously by using of capacitance hygrometer-sweat capture capsule method (Fan, 1987). Changes in temperature of oral cavity and skin (chest, back and forearm) were simultaneously measured with thermistors.

Table 1. Results of thyroid function tests in each group

	T4 $\mu\text{g/dl}$	T3 ng/dl	TSH $\mu\text{U/ml}$
Hyper (n=5)	27.3 \pm 4.1	589.3 \pm 90.6	ND
Hypo-Ⓐ (n=2)	0.9 1.8	19.6 29.3	91.9 112.3
Hypo-Ⓑ (n=2)	0.4 1.5	11.2 18.2	114.2 132.0
Control (n=9)	10.0 \pm 1.5	112.5 \pm 17.8	1.1 \pm 0.4

Mean \pm SD. Hyper: hyperthyroidism, Hypo-Ⓐ: long-term hypothyroidism, Hypo-Ⓑ: short-term hypothyroidism. Individual data were shown in the group of Hypo-Ⓐ and Hypo-Ⓑ. ND: not detectable.

The present study was performed in 2 p.m.–4 p.m. from January to March to avoid the influence of circadian and seasonal variation of thermal sweating.

Values are presented as means \pm SD. Statistical differences were assessed by the Student's t-test for unmatched data.

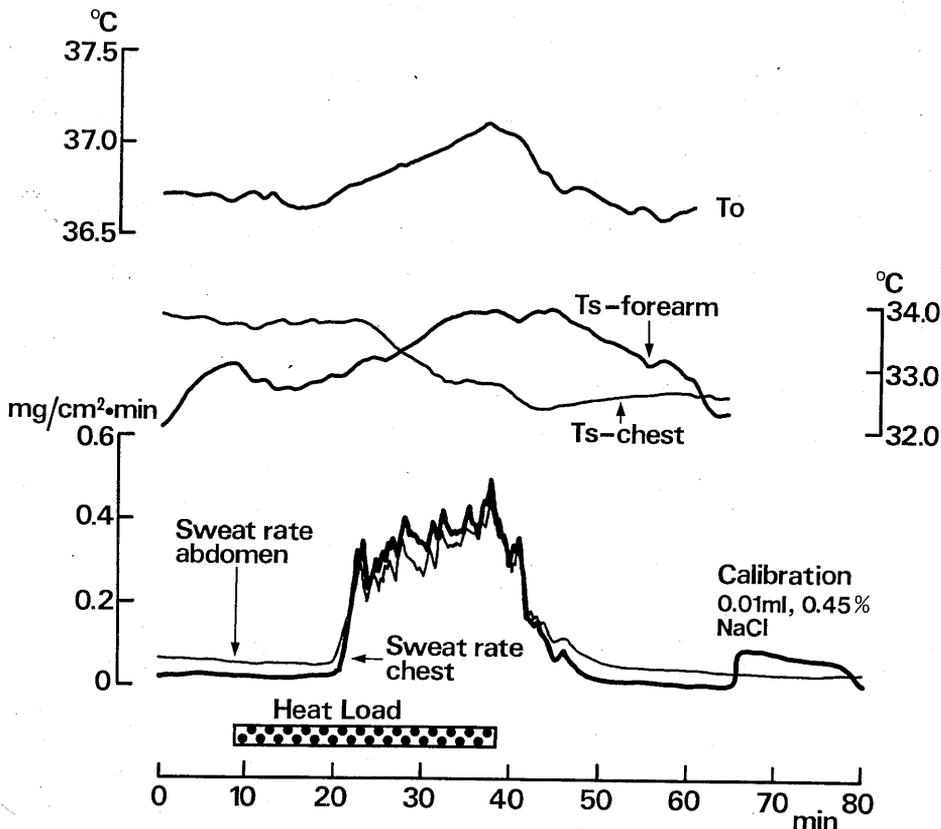


Fig. 1. Typical recording of a control subject. To: oral temperature, Ts: skin temperature. For details see text.

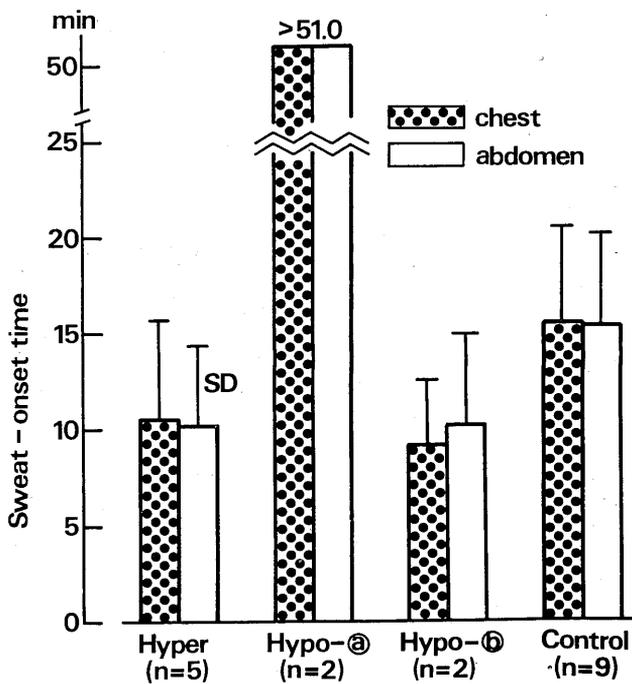


Fig. 2. Comparison of sweat-onset time. Hyper: hyperthyroidism, Hypo-a: long-term hypothyroidism, Hypo-b: short-term hypothyroidism. For details see text.

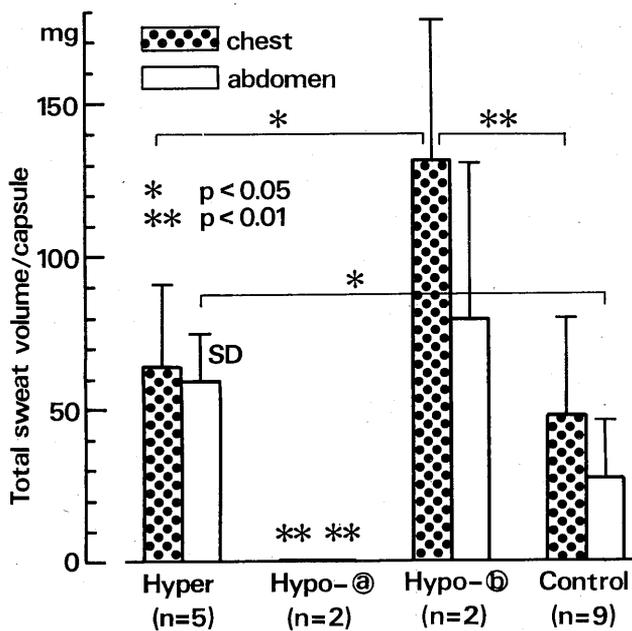


Fig. 3 Comparison of total sweat volume induced by 30 min heat load. Hyper: hyperthyroidism, Hypo-a: long-term hypothyroidis, Hypo-b: short-term hypothyroidism. For details see text.

RESULTS AND DISCUSSION

Typical recording of a control subject is shown in Fig. 1. Rise in forearm skin temperature by heat load was observed first and rise in oral temperature followed. Then sweat occurred at the sweat-onset time of 12.0 min (chest) and 11.6 min (abdomen). Sweat-onset threshold of oral temperature was 36.77°C (0.06°C increased). Considerable fall in chest skin temperature due to sweating (evaporative heat dissipation) was observed. At the end of experiment, calibration of sweat volume was done and local sweat rate was expressed in the order of $\text{mg}/\text{cm}^2\cdot\text{min}$. Total sweat volume induced by 30 min heat load was 73.86 mg/capsule in the chest and 58.03 mg/capsule in the abdomen (Fig. 1).

Sweat-onset time and total sweat volume in the four groups (hyperthyroidism, long-term hypothyroidism, short-term hypothyroidism and control) were summarized and shown in Figs. 2 and 3, respectively. In hyperthyroidism, sweat-onset time (10.6 ± 5.2 min) was short compared to that in control (15.6 ± 5.0 min), though there were no significance. In the previous reports sweat-onset time had not been determined because a body balance was indirectly used to measure sweat rate (Judith *et al.*, 1972, 1973). Total sweat volume (66.06 ± 26.24 mg/capsule) in hyperthyroidism was large compared to that in control (47.95 ± 31.25 mg/capsule), though non-significant in the chest. Judith *et al.* (1973) described that surprisingly sweat rate of thyrotoxic patients were so little above normal under fairly normal environmental condition. However, in the present study there was a significant difference in the comparison of sweat volume determined in abdominal capsule: 59.33 ± 15.81 (hyperthyroidism) vs. 27.62 ± 18.73 mg/capsule (control), $p<0.05$.

Up to now there was no report of sweat test in hypothyroidism. However, our observation is the first study to confirm that no sweat was induced by 30 min heat load in patients with hypothyroidism (long-term) (Fig. 3). Therefore, heat load was continued moreover. Thermal sweating was observed at 56 min of sweat-onset time in one case, but no sweat was induced in the other case by the continuation of heat load until the experiment was discontinued at 51 min because of her complaint of discomfort.

On the other hand, shorter sweat-onset time and larger amount of sweat were observed in patients with short-term (less than two weeks) hypothyroidism in a striking contrast to those obtained in long-term hypothyroidism, although these two groups showed the same levels of circulating thyroid hormones (Table 1).

Changes in oral temperature and mean skin temperature (average of determined values at three regions) were shown in Table 2. Initial temperature in both of oral and skin in hyperthyroidism were significantly higher than those in control ($p<0.05$). There were no difference in oral and mean skin temperature between hypothyroidism and control before heat load.

Change in oral temperature due to 30 min heat load is considered to be a balance of heat uptake on the heated local skin and heat dissipation owing to sweating (evaporative heat loss) and to non-evaporative heat loss. It was $0.57\pm 0.13^{\circ}\text{C}$ in hyperthyroidism and larger than that in control ($0.41\pm 0.12^{\circ}\text{C}$), $p<0.01$ (Table 2). Under ther-

Table 2. Comparison of oral temperature and skin temperature

	Ts-mean before	To before	Δ To of sweat-onset	Δ To of 30 min heat load
Hyper (n=5)	34.47 \pm 0.81	37.20 \pm 0.18	0.20 \pm 0.16	0.57 \pm 0.13
Hypo-Ⓐ (n=2)	32.99 \pm 1.38	36.83 \pm 0.06	0.50, >0.90	0.18 \pm 0.06
Hypo-Ⓑ (n=2)	32.98 \pm 0.49	36.64 \pm 0.23	0.21 \pm 0.11	0.80 \pm 0.01
Control (n=9)	33.29 \pm 0.67	36.87 \pm 0.24	0.10 \pm 0.08	0.41 \pm 0.12

Hyper: hyperthyroidism, Hypo-Ⓐ: long-term hypothyroidism, Hypo-Ⓑ: short-term hypothyroidism, Ts: skin temperature, To: oral temperature.
Mean \pm SD, * p<0.05, ** p<0.01, *** p<0.001

mononeutral condition (25°C) vasodilation of skin vessels is already occurred in thyrotoxic patients whose deep body temperature and skin temperature are higher than control in order to dissipate excess heat produced by increased metabolism. Therefore larger amount of heat is considered to be reversely taken into the body when ambient temperature is above skin temperature or local heat load is applied. In other words, dry heat loss mechanism acts under thermoneutral condition for normal subjects, and sweating appears at shorter onset time when a little heat load (i. e. exercise and increase in ambient temperature) is applied, which does not induce thermal sweating in normal subjects.

Increase in oral temperature by 30 min heat load was 0.18 \pm 0.06°C in long-term hypothyroidism and 0.80 \pm 0.01°C in short-term hypothyroidism, respectively. In hypothyroidism (long-term) a little rise in oral temperature and no thermal sweating were induced by 30 min heat load, it might be due to weakness of vasodilation on heated skin. And considerable increase in oral temperature of 0.50°C or above 0.90°C could induce thermal sweating, this fact suspects that thyroid hormone acts directly on the controlling mechanism of sweating.

In short-term hypothyroidism (less than 2 weeks) thyroid function test revealed the same value as long-term hypothyroidism, however, sweat volume and rise in oral temperature were larger and sweat-onset time was shorter than those of control. The difference of thermal sweating between long- and short-term hypothyroidism suggests that the action of thyroid hormone intra-cellular level may change more slowly than the regulation in plasma level. Gibinski *et al.* (1972) reported that sweat rate did not decrease after several weeks thyrostatic treatment in thyrotoxic patients.

From the present results it is suggested that thyroid hormone may enhance thermal sweating with direct involvement in the regulation mechanism of sweating. However, further studies should be done to clarify the effect of thyroid hormone on thermal sweating and the action site of thyroid hormones on sweating mechanism.

ACKNOWLEDGEMENT

The authors greatly appreciate the excellent secretarial assistance of Misses Junko Kawashima and Junko Hayashima.

REFERENCES

- 1) Fan, Yu-Jen (1987): Determination of acclimatization by capacitance hygrometer-sweat capture capsule method. *Trop. Med.*, 29 (2), 107-121.
- 2) Gibinski, K., Powierza-Kaczynska, C., Zmudzinski, J., Giec, L. & Dosiak, J. (1972): Thyroid control of sweat gland function. *Metabolism*, 21, 843-848.
- 3) Judith, A. A., Jenkinson, D. J. & Roddie, I. C. (1972): The effect of β -adrenoceptor blockade on human sweating. *Br. J. Pharmac.*, 47, 487-497.
- 4) Judith, A. A., Daphne C. L., Roddie, I. C. & Wallace, W. F. M. (1973): Studies on thermal sweating in clinical and experimental thyrotoxicosis. *Clin. Sci. Mol. Med.*, 45, 765-773.
- 5) Krikler, D. M. (1966): Adrenergic receptor blockade. *Lancet*, 268.
- 6) Shanks, R. G., Hadden, D. R., Lowe, D. C., McDevitt, D. G. & Montgomery, D. A. D. (1969): Controlled trial of propranolol in thyrotoxicosis. *Lancet*, i, 993-994.