tern with low levels from the 'flash' (1L), through the second (6D) scotophase, and continuing until the end of the 7L photoperiods. As in the rat[7], it appears that the changes induced by a short period of light, in this case 1 h, have effects on melatonin secretion which last through the succeeding 6-h period of darkness. Thus, it is the length of the first scotophase which is critical in determining the secretory pattern of melatonin. This agrees with the conclusions of Ravault and his colleagues[15] who found that the effects of a 1-h 'flash' of light on prolactin secretion in sheep were dependent on the length of time between the end of the main photophase and the start of the 'flash' – the period during which, in our experiment, melatonin levels were elevated.

Plasma samples were also assayed for prolactin and cortisol in the current experiment and the results are reported elsewhere[16]. Prolactin secretion was stimulated by skeleton long photoperiod ($P < 0.001$), with no obvious circadian rhythm. Plasma cortisol was significantly lower in skeleton long photoperiods than in short days ($P < 0.05$), but again there was no clear circadian rhythm. Thus, the changes in plasma levels of melatonin which are reported in the current work do not immediately affect the secretion of prolactin or cortisol, although the pineal gland is involved in their control; pinealectomy blocks the effect of skeleton long photoperiod on prolactin and cortisol in growing sheep[17].

The results of this experiment support current evidence that it is the temporal pattern of melatonin secretion rather than the overall amount of melatonin present over 24 h which may be involved in the photoperiodic mechanisms in sheep[8].

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Effects of intraventricular administration of insulin on thyrotropin secretion in rats

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Summary. Intraventricular administration of insulin stimulates increases in the levels of thyrotropin-releasing hormone and thyrotropin in rats.

Insulin is found in the rat hypothalamus[3] and stimulates increases in the levels of growth hormone and corticotropin[4,5]. Studies on the effect of insulin on thyrotropin (TSH) secretion have revealed that the peripheral administration of insulin induced elevation of plasma TSH levels in rats[6,7]. However, sites of the effects on TSH secretion are unclear at present. Therefore, the authors investigated intraventricular administration of insulin on TSH release in rats.

Materials and methods. Animals. Male (Wistar strain) rats weighing 200 g were employed. They were housed in temperature (22 °C) – and humidity (60%) – controlled quarters and fed a diet of laboratory chow and water ad libitum. *Drugs.* Ovine insulin was kindly supplied by Shimizu Pharm. Co., Ltd. (Japan)[2]. Synthetic thyrotropin-releasing hormone (TRH) was purchased from the Protein Research Foundation (Japan). *Experimental design.* All experiments were conducted in a temperature-controlled room (22 °C). After overnight fasting, the rats were anesthetized with pentobarbital (50 mg/kg). One hundred nU of insulin was dissolved in 1 µl of saline, and injected intraventricularly using a 10 µl Hamilton syringe as described earlier[8]. The rats (5 at a time) were then decapitated with a guillotine at 5, 10, 30, 60 and 90 min after the insulin injection. Trunk blood was collected in heparinized tubes kept on ice. The hypothalami were obtained by the method previously described[9]. For the control, saline was injected. *Assay methods.* TRH, thyroxine (T_4) and 3,3',5-triiodothyronine (T_3) were measured by means of a specific radioimmunoassay for each[10,11]. TRH content in the hypothalamus was expressed with reference to a total amount of dissected hypothalamic section. TSH was determined by the rat TSH radioimmunoassay kit[12] supplied by the National Institute of Arthritis, Diabetes and Digestive and Kidney diseases Research Materials Distribution Program (NIAMDD). Blood glucose levels were measured by the autoanalyser method. *Statistics.* Mean and standard error of the mean was calculated for each group. Student's t-test was used to evaluate the differences between the control and experimental groups.

Results (table). The hypothalamic immunoreactive TRH (ir-TRH) content decreased significantly after insulin injection, whereas its plasma concentration increased significantly. The plasma TSH levels increased significantly with a zenith at 30 min after the injection. The plasma T_3 levels increased signi-

Effects of intraventricular administration of insulin on immunoreactive TRH contents in the hypothalamus, plasma immunoreactive TRH, TSH, T_4, T_3 and blood glucose levels

After drug administration	0	5	10	30	60	90 min
Saline-treated						
TRH contents in the hypothalamus (ng)	4.1 ± 0.3	4.1 ± 0.2	3.9 ± 0.2	4.1 ± 0.3	4.0 ± 0.3	4.2 ± 0.3
TRH in plasma (pg/ml)	6.4 ± 2.0	6.3 ± 1.9	5.2 ± 2.0	6.1 ± 2.1	5.6 ± 1.8	6.4 ± 2.0
TSH in plasma (ng/ml)	270 ± 24	264 ± 24	266 ± 25	270 ± 26	264 ± 24	266 ± 25
T_4 in plasma (μg/dl)	4.9 ± 0.3	4.9 ± 0.4	5.0 ± 0.3	5.1 ± 0.4	5.2 ± 0.3	4.9 ± 0.2
T_3 in plasma (ng/dl)	51 ± 3.6	52 ± 3.8	49 ± 3.4	47 ± 3.4	48 ± 4.0	47 ± 3.2
Blood glucose (mg/dl)	88 ± 4.5	85 ± 4.6	80 ± 4.0	80 ± 3.8	78 ± 3.9	77 ± 3.7
Insulin-treated						
TRH contents in the hypothalamus (ng)		3.6 ± 0.2	3.0 ± 0.3[c]	3.4 ± 0.3	3.8 ± 0.3	4.2 ± 0.3
TRH in plasma (pg/ml)		10 ± 3.4	30 ± 6.4[b]	16 ± 3.6[c]	11 ± 3.4	7.0 ± 2.6
TSH in plasma (ng/ml)		290 ± 25	340 ± 27	480 ± 29[a]	360 ± 29[c]	260 ± 23
T_4 in plasma (μg/dl)		5.2 ± 0.3	5.1 ± 0.3	5.2 ± 0.4	5.4 ± 0.4	5.6 ± 0.4
T_3 in plasma (ng/dl)		50 ± 3.6	55 ± 4.0	58 ± 4.2	72 ± 4.6[b]	86 ± 4.2[a]
Blood glucose (mg/dl)		86 ± 4.2	82 ± 3.8	78 ± 4.1	77 ± 3.9	75 ± 3.6

Values are expressed as the mean ± SE in each group of 5 rats. Differences from the saline-treated group are shown by [a] $p < 0.001$, [b] $p < 0.005$ and [c] $p < 0.05$.

ficantly, but that of T_4 did not. The blood glucose levels did not change significantly.

Discussion. It has been reported that the peripheral administration of insulin stimulates TSH release in rats[6,7], and the authors found that intraventricular injection of insulin increased plasma ir-TRH and TSH levels. This suggests that insulin may act on the hypothalamus. The hypothalamic ir-TRH content and its plasma concentration may expressed a balance between TRH release, synthesis and degradation, so the inactivation of TRH immunoreactivity by plasma or hypothalamus in vitro was investigated. The inactivation of TRH immunoreactivity by plasma or hypothalamus after insulin injection did not differ from that of the control[13]. These data suggest that insulin may affect TRH release or synthesis. The question can be raised as to whether the effect of insulin on TSH release might be a direct action of insulin on the hypothalamus or mediated via a fall in blood glucose levels. The present data indicate that the effects of insulin on TSH release are not mediated via a fall in blood glucose levels. Plasma T_3 levels increased significantly after insulin injection, confirming the results obtained by peripheral administration of insulin[6,7]. The present study revealed that plasma ir-TRH levels increased followed by increases in plasma TSH and T_3. The time course of these changes is similar to that with exogenous TRH administration[14]. These data suggest that insulin acts directly at the hypothalamus level to stimulate TRH and TSH release.

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Can postictal suppression of the perforant path – fascia dentata responses account for the ECS-induced anterograde amnesia in rats?

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Summary. Electroconvulsive shock (ECS) decreases fascia dentata responses to entorhinal stimulation by 50% in unanesthetized rats. Synaptic potentials and population spikes return to pre-ECS level during 1 h and 3 h, respectively. This recovery rate is compared with the dynamics of ECS-induced anterograde amnesia.

The assumption that the hippocampus plays an important role in memory processes[3] is based not only on results of lesion studies but also on conditioning-elicited changes of hippocampal evoked potentials[4,5] and on anterograde and retrograde amnesia caused by epileptic activity of this structure[6,7]. Hesse and Teyler[8] demonstrated in anesthetized rats that hippocampal afterdischarge suppresses the long-term potentiation elicited in CA1 stratum radiatum by low frequency tetaniza-