eluated with a mixture of ethyl-acetate:methanol (7:3). Concentrations were determined by Unicam Sp 500 spectrophotometer at 530 mμ wavelength. In every instance, at least 6 controls and 5 to 6 adrenals of unilaterally adrenalectomized rats were incubated together. The quantity of aldosterone is easily measurable in this way. Therefore, every point of the Figure illustrates the production of at least 5 adrenals. It is seen from the data of the Figure that, in accordance with literary data[4], corticosterone production of compensatory hypertrophic adrenals shows a definite increase. On the other hand, production of aldosterone remains unchanged.

Although regulation of aldosterone production is, from many points of view, not yet entirely clarified, a regulation of humoral nature must certainly be considered, as shown in the first place by YANKOPOULOS' cross-circulation experiments[8]. The fact that in compensatory hypertrophy no increased aldosterone production was found, suggests that the regulating system of aldosterone production does not become mobilized by the effect of a 50% decrease. The increase of corticosterone production is doubtless the consequence of endogenous ACTH hyperproduction. The present experimental findings are evidence against the aldosteronotrope role of ACTH.

Zusammenfassung. Die Aldosteronproduktion der kompensatorisch-hypertrophisierten Nebennieren wurde im Rattenexperiment untersucht. Die Corticosteronproduktion zeigte eine wesentliche Zunahme, dagegen blieben die Aldosteronwerte unverändert, was gegen eine ACTH-Regelung der Aldosteronsekretion spricht.

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Food Intake Patterns from Weaning to Adulthood in Male and Female Rats with Hypothalamic Lesions[1]

The classical studies of HETHERINGTON and RANSON[2–5], of BROBECK[6,7], and ANAND and BROBECK[8,9] described the role of the hypothalamus in the control of food intake; these findings were confirmed by numerous workers[10].

Aside from a study by KENNEDY[11], who investigated the effect of ventromedial lesions on food intake in weanling rats, these studies were conducted in adult rats. In contrast, however, studies of food intake pattern in rats that had lesions placed in various parts of the hypothalamus shortly after weaning are not known to this author.

The rats reported here were used initially for a study concerning the effect of hypothalamic lesions on growth. The present note deals with the food intake pattern observed in these animals from the time of operation (25 days of age) to the age of 98 days. Charles River rats of both sexes received bilateral electrolytic lesions using a modified Horsley-Clarke stereotaxic instrument. All rats were housed in individual cages in a room, kept at 24°C which was successively light and dark for 12 h periods. A synthetic diet, which yielded 4.2 Cal/g, and water were available *ad libitum*. Food intake was measured either for three consecutive days weekly or for four consecutive days every two weeks. Variations in the size of the groups as well as in the periods of measurement occur because some animals were used for experiments other than described in this note.

(*A*) *Food Intake Pattern of Male Rats with Hypothalamic Lesions Compared to Intact Controls.* The only group with a consistently lower food intake throughout the entire period of this investigation is that with lesions in the area of the dorsomedial nuclei (Group 5M, Table I). In none of the other groups was there a significant difference from the control values before the ninth week of life (sixth week post-operative). Food intake diminished temporarily in rats with lesions in the supraoptic area insufficient to produce diabetes insipidus (Group 2M). But ablations in both the ventromedial area (Group 3M) and lesions in the region of the mammillary-dorsal premammillary nuclei (Group 7M) were associated with significantly increased food ingestion at comparable ages. This finding correlates with puberty (50–60 days[12]). It is noteworthy that rats with ventromedial lesions did not display the extreme degree of hyperphagia which is so characteristic for older animals with ablations in this structure[3,6,7]. Furthermore, lesions in the mammillary-dorsal premammillary area (Group 7M) were associated with continued hyperphagia in the post-pubertal period.

(*B*) *Food Intake Pattern of Female Rats with Hypothalamic Lesions versus Intact Controls.* In female rats with lesions in the region of the supraoptic nuclei a slight, but significant, hyperphagia occurred during the tenth week of life (Group 2F, Table II); in male rats this had transpired during the ninth week. On the other hand, lesions in the area of the dorsomedial nuclei (Group 5F) did not cause the hypophagia shown by male rats with similar lesions. Ablations involving the mamillary-dorsal-premammillary nuclei of females (Group 7F) resulted, as in the males, in increased food intake which appeared during the tenth week of life. Rats with lesions in the

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Table I. Food intake of male hypothalamic and intact control rats (g/day)

Week	Group						
	1	2	3	4	5	6	7
5	(44)[b] 11.8 ± 0.52[a]	(20) 11.7 ± 0.64	(9) 11.6 ± 0.81	(3) 12.3 ± 1.52	(3) 8.5 ± 0.29	(9) 11.5 ± 1.44	(3) 14.3 ± 2.53
6	(33) 13.5 ± 0.31	(21) 12.9 ± 1.79	(5) 12.3 ± 1.15	—	(8) 9.3 ± 0.72	—	—
7	(21) 17.0 ± 2.55	(9) 17.1 ± 1.12	(5) 15.8 ± 1.53	—	(5) 9.9 ± 1.87	—	—
8	(40) 19.1 ± 0.40	(23) 17.7 ± 3.60	(6) 18.4 ± 1.27	(3) 16.5 ± 2.43	(8) 14.7 ± 1.39	—	—
9	(33) 20.6 ± 0.65	(11) 17.4 ± 1.26	(8) 23.4 ± 1.09	(3) 15.8 ± 5.34	(3) 16.8 ± 1.17	(9) 18.6 ± 1.20	(3) 25.8 ± 1.89
10	(25) 19.2 ± 1.02	(12) 22.5 ± 1.95	(3) 16.8 ± 2.42	(8) 16.9 ± 1.33	—	—	—
11	(30) 21.1 ± 0.74	(12) 20.6 ± 1.62	(6) 22.6 ± 0.91	—	(8) 17.5 ± 1.46	(9) 20.1 ± 1.62	(3) 26.4 ± 3.02
12	(24) 20.6 ± 0.67	(9) 19.1 ± 1.82	(4) 18.9 ± 1.03	(3) 22.4 ± 3.32	(3) 15.2 ± 0.67	—	—
13	(22) 21.3 ± 0.65	(7) 21.8 ± 2.26	(3) 20.3 ± 1.20	—	(5) 21.0 ± 4.62	(9) 19.4 ± 1.24	(3) 25.8 ± 1.18
14	(5) 24.2 ± 0.42	(5) 21.6 ± 1.38					

[a] Mean ± S.E.M. ····· $P < 0.05$. [b] n —— $P < 0.01$.

Table II. Food intake of female hypothalamic and intact control rats (g/day)

Week	Group						
	1	2	3	4	5	6	7
5	(12)[b] 12.1 ± 0.96[a]	—	(5) 14.3 ± 0.93	(6) 10.4 ± 1.70	(2) 9.9 ± 0.85	(3) 9.6 ± 1.87	(6) 12.2 ± 1.71
6	(6) 10.7 ± 0.83	(3) 10.7 ± 1.00	—	—	—	—	—
7	(12) 15.6 ± 1.12	—	(5) 14.3 ± 1.24	(6) 15.1 ± 2.24	(2) 7.8 ± 4.50	(3) 12.4 ± 0.88	(6) 16.5 ± 0.73
8	(6) 12.4 ± 0.69	(3) 15.3 ± 2.31	—	—	—	—	—
9	(6) 18.2 ± 2.48	(4) 17.0 ± 2.37	—	—	—	—	(3) 21.7 ± 2.11
10	(12) 16.7 ± 0.53	(3) 13.7 ± 1.45	(5) 20.9 ± 1.47	(2) 17.9 ± 1.15	(2) 12.5 ± 3.20	(2) 13.2 ± 1.50	(3) 21.1 ± 1.74
11	(6) 17.9 ± 1.99	—	—	(4) 17.8 ± 3.71	—	—	(3) 20.7 ± 1.88
12	(12) 17.9 ± 0.64	(3) 14.2 ± 2.22	(5) 23.0 ± 2.10	(2) 21.4 ± 4.65	(2) 16.0 ± 2.00	(2) 10.0 ± 6.70	(3) 21.7 ± 1.25
13	(6) 17.1 ± 1.00	—	—	(4) 17.6 ± 2.74	—	—	(3) 18.7 ± 0.67
14	(12) 17.6 ± 0.62	(3) 15.8 ± 3.28	(5) 22.1 ± 1.54	(3) 20.0 ± 6.30	(2) 16.0 ± 3.00	(2) 16.7 ± 0.35	(3) 20.5 ± 1.80

[a] Mean ± S.E.M. ····· $P < 0.05$. [b] n —— $P < 0.01$.

ventromedial area (Group 3F) showed no significant hyperphagia until the 12th week of life.

It appears that lesions placed in certain areas of the hypothalamus of the weanling rat in both sexes result in derangements of food intake patterns which, depending upon sex and location, either cause a transitory or persistent increase or decrease of food intake. Puberty seems to be a period in the life cycle during which some lesions cause hypophagia and in others hyperphagia. Then, following puberty, this effect either persists or subsides. Other hypothalamic lesions have no effect on feeding behavior. How significantly the endocrine correlates of puberty, coupled with the hypothalamic lesions, are responsible for these changes in food intake is open to speculation. It is conceivable that the striking endocrine alterations of puberty may affect energy balance or the degree of utilization of ingested food for anabolic processes. This may then involve a feed-back mechanism in relation to appropriate centers regulating caloric intake.

In young rats with lesions in the ventromedial area the absence of a degree of hyperphagia which transcends that correlated with growth may be consistent with the thesis that growth *per se* provides maximal hyperphagia. Growing animals, as KENNEDY[11] has shown, eat about twice as much as mature rats in proportion to their body weight; their food intake appears to be unaffected by hypothalamic lesions until the age at which the food intake relative to body weight begins to decrease. Lesions in the posterior hypothalamus thus far reported did not cause hyperphagia[11]. But such lesions did result in hyperphagia in both sexes in the present study. On the other hand, HETHERINGTON and RANSON[5] found that lesions in the caudal hypothalamus dorsolateral to the mammillary bodies did cause obesity in rats weighing about 95–130 g. Food intake, however, had not been measured in their study.

The findings of the present study suggest that effects of hypothalamic lesions on food intake cannot be properly evaluated without taking into account the age of the rat at the time lesions are produced. Ablations which may cause changes in food intake if placed after maturity may not do so shortly before puberty or at still an earlier age. The converse of the foregoing may also obtain. Fluctuations in energy requirements during growth and puberty may cause transient fluctuations in food intake which can be evident only in continuous studies from weaning through adulthood[13].

Zusammenfassung. Änderungen in der Futteraufnahme von Ratten mit hypothalamischen Läsionen sind nicht nur vom Locus, sondern auch vom Geschlecht und dem Alter abhängig, in welchem sie beigebracht wurden. Pubertät scheint eine Zeit zu sein, in welcher Änderungen in der Futteraufnahme eintreten. Läsionen im hinteren Hypothalamus, die bisher nicht als hyperphagisch bekannt waren, wenn sie in erwachsenen Ratten plaziert wurden, verursachten Hyperphagie in beiden Geschlechtern, wenn die Operation kurz nach Ablaktation durchgeführt wurde.

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Acute Effect of Thyrotrophic Hormone on the Concentration of Succinic Acid Dehydrogenase and Sulfhydryl Groups in the Thyroid Gland of the Rat[1]

A great variety of methods have been evolved for the measurement of thyroid function[2]. A feature that most of them have in common is that they require a fairly long-term effect on the thyroid gland. Some methods have been developed, however, to elicit the acute changes in thyroid function. They are based on the determination of intracellular colloid granules[3,4], percentage of epithelium[2], radioactive P^{32} uptake[5], or radioactive I^{131} uptake[6].

It has been suggested recently that the determination of succinic acid dehydrogenase[7] and sulfhydryl groups[8] can be used to measure thyroid function in long-term tests. The purpose of this work is to ascertain whether these methods can be applied in the measurement of acute changes in thyroid function.

Sixty-four Long-Evans male rats were employed as the test animals. They were divided into 8 groups of 8 animals each. The rats of 7 groups were given 15 IU of thyrotrophic hormone (Ambinon, Organon) subcutaneously, the eighth group serving as controls. The thyrotrophic hormone (TSH) groups were killed 1, 2, 4, 6, 8, 12, and 16 h after the injection simultaneously with the controls. To eliminate possible diurnal variation the injections were arranged so that the animals of all the groups were sacrificed at the same time before noon. The animals were killed by rapid decapitation, the thyroid glands were removed and weighed by torsion balance. One lobe of the thyroid was homogenized and the succinic acid dehydrogenase concentration was determined by the method of VILLAREAL and BURGOS[9]. The SH-groups were determined by the mercury-orange method of BENNETT and WATTS from the other half of the gland[10]. 'Student's' *t*-test was used in the statistical analysis of the results[11].

The results are tabulated. The Table shows that the succinic acid dehydrogenase concentration of the thyroid gland rises fairly sharply under the influence of TSH. It reaches its maximum within 4 h of the injection and returns to the control level in 12 h. The increase is statistically significant. The elevation in the concentration of the

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