similar results were obtained from in vivo and in vitro experiments. It is also characteristic that full inhibition of the hormonal activity was never obtained, independently of the dilution of specific sera and the time of inactivation. There always remained some residual hormonal activity.

Discussion. These results indicate that the different biological activity of the parathyroid hormone may be connected with the different active centers of hormone. Moreover the immunological activity is connected to a high degree with a fragment of hormone which is probably the same as this one responsible for the calcium mobilization phenomenon[1].

Parathyroid hormone has a great immunological specifity. It was not possible to obtain any form of the immunological reaction with non-specific sera. In all experiments the biological inactivation of hormonal materials was obtained only when the specific antisera were used. It is not in agreement with the former results of KOOH and FRASER[9], which observed the inhibition of the guinea-pig, rabbit and rat parathyroid hormone activity after administration of anti-bovine PTH serum.

The influence of the parathyroid hormone inactivation on the phosphorus release was also investigated but no significant differences were observed. It is in agreement with the opinion that phosphorus metabolism is independent from the parathyroid glands, but rather depends on the dietary content of the mineral[10,11].

Résumé. Des préparations hormonales de parathyroïde humaine, bovine et porcine furent traitées par des sérums anti PTH spécifiques et non spécifiques. Les expériences in vitro et in vivo ont montré que l'inhibition de l'activité biologique du PTH se produit seulement sous l'influence des sérums spécifiques. L'inactivation immunologique n'inhibe jamais complètement l'activité du PTH.

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[9] S. W. KOOH and D. FRASER, Can. J. Physiol. Pharmac. *46*, 441 (1968).
[10] C. E. DENT and C. M. HARPER, Lancet *1*, 559 (1962).
[11] B. J. FAIRHURST, Lancet *1*, 302 (1963).

X-Irradiation and Thyroid Activity in a Teleost, *Mystus vittatus* (Bloch)

Some investigators have reported similarity of dose-survival time curves after X-irradiation between mammals and few teleosts[1-4]. Extensive studies on the thyroid physiology of mammals in response to ionizing radiation, and very little work in this field on teleostean thyroid, present an uneven picture of the thyroid activity in this group. Therefore, in this experiment an attempt has been made to study the effects of varying doses of X-irradiation on thyroid gland activity in a freshwater catfish, *Mystus vittatus*. This species was specially selected for the present program because some aspects of its thyroid activity under natural and varied experimental conditions are well known[5-10]. Thyroidal radioiodine (^{131}I) uptake and its histology were taken as parameters for the measurement of thyroid activity, which were done by the procedure described earlier[6].

210 adult males of *M. vittatus* were utilized in this experiment. 180 specimens were divided into 6 batches of 30 each. Their pharyngeal area containing thyroid follicles were exposed to varying doses of X-rays and their thyroid activity at regular intervals of 1, 2, 3, and 4 months were studied (Table). The X-ray apparatus was operated at 250 kvp, 30 ma, with 0.5 mm Al filter. The temperature for experimental and control groups was kept more or less uniform and it ranged from 22 to 24 °C. Exposure of pharyngeal region to the dose of 0.8 kR (Batch 1) initially accelerated the thyroidal radioiodine uptake. Specimens of batch 2 showed slight reduction in thyroid activity

[1] H. QUASTLER, Radiation Res. *4*, 303 (1956).
[2] N. EGAMI, H. ETOH, C. TACHI and Y. HYODO, Zool. Mag., Tokyo *71*, 313 (1962).
[3] Y. HYODO, Annotnes zool. jap. *37*, 104 (1964).
[4] H. ETOH and N. EGAMI, Annotnes zool. jap. *38*, 114 (1965).
[5] T. P. SINGH and A. G. SATHYANESAN, Acta zool. *49*, 47 (1968).
[6] T. P. SINGH, Z. Zellforsch. mikrosk. Anat. *87*, 422 (1968).
[7] T. P. SINGH, Gen. comp. Endocr. *11*, 1 (1968).
[8] T. P. SINGH, Z. Zellforsch. mikrosk. Anat. *94*, 172 (1969).
[9] T. P. SINGH, Experientia *25*, 431 (1969).
[10] T. P. SINGH, Gen. comp. Endocr. *12*, 556 (1969).

Thyroid activity of *Mystus vittatus* in response to varying doses of X-rays

Batch[a]	Dose in kR/fish	Maximum of ^{131}I uptake after irradiation at various intervals (%)			
		1 month (mean ± S.E.[b])	2 months (mean ± S.E.)	3 months (mean ± S.E.)	4 months (mean ± S.E.)
1	0.8	16.00 ± 1.49 (8)	19.23 ± 2.00 (7)	20.15 ± 1.55 (6)	19.77 ± 1.20 (6)
2	1.6	15.40 ± 0.86 (6)	13.32 ± 0.54 (7)	11.80 ± 0.77 (6)	10.00 ± 1.15 (6)
3	2.4	13.50 ± 1.20 (6)	10.24 ± 1.10 (6)	9.79 ± 0.40 (7)	7.50 ± 0.48 (6)
4	3.2	9.37 ± 0.76 (5)	5.64 ± 0.53 (6)	4.58 ± 0.53 (5)	3.00 ± 0.22 (5)
5	4.0	6.29 ± 0.49 (5)	4.00 ± 0.28 (5)	2.17 ± 0.54 (5)	2.18 ± 0.44 (6)
6	4.8	4.13 ± 0.32 (5)	2.37 ± 0.18 (5)	1.70 ± 0.23 (5)	
7	Sham-irradiated control	14.56 ± 1.22 (8)	13.67 ± 0.88 (6)	15.00 ± 1.39 (6)	16.24 ± 0.76 (6)

[a] Each batch had 30 specimens. [b] Mean with S.E. Number of fishes used for the evaluation of thyroid activity at various intervals are given in parentheses.

after 90 days of irradiation. The same response in thyroid activity was produced in 60 days post-irradiation with 2.4 kR as evident by I^{131} uptake (Batch 3). The rate of reduction in thyroid activity was observed to be directly proportional to the increasing doses of X-rays. The thyroid activity in the specimens of batch 6 after exposure to 4.8 kR was depressed very quickly and registered an uptake of only about 4% of the total injected dose on 30th day post-treatment as compared to 15% uptake of sham-irradiated control. Radiation-induced damage in the thyroid gland histology was reflected by follicular atrophy, loss of epithelium and colloid and connective tissue fibrosis. There was no significant change in the thyroid follicles of the specimens of batch 2 when compared to controls till 90 days after irradiation. A slight follicular atrophy was observed after 120 days of treatment. Although the thyroidal radioiodine uptake was considerably depressed in batches 4, 5 and 6 within 30 days of treatment, significant damage in thyroid histology became apparent at about 60 days after irradiation. The appearance of histological damage in thyroid gland was slower than its radioiodine uptake response after X-irradiation. It seems that radioiodine uptake test for thyroid activity is more sensitive than histological response which has also been advocated by FONTAINE et al.[11]. The serum-PBI measurements were also done for the evaluation of thyroid activity but the result has been communicated elsewhere. The state of activity of thyroid gland as reflected by radioiodine uptake was also supported by serum-PBI test. These tests – thyroidal I^{131} uptake and serum-PBI for determination of thyroid activity – appear to be more sensitive, quick and reliable.

Further experiments in this line are currently in progress. X-irradiated specimens (frequency 2.4 to 5 kR) showed very little or no response to TSH administration. It appears that thyroid dysfunction induced by X-irradiation is primary and not the result of TSH deficiency. Probably X-rays basically interfere with biological activity of epithelial cells of thyroid gland and inactivate and or kill enzymes responsible for initiating the trapping of circulating iodine and synthesis of thyroid hormone, or cell permeability of the follicular cells is lost and thus they are unable to perform one of their primary tasks, i.e., to maintain 'Iodine pump' which provides the raw material 'iodine' required in thyroid hormone synthesis[12].

Zusammenfassung. Eine niedrige Dosis Röntgenstrahlen führt zu einer mässigen Steigerung der Radiojodaufnahme in der Schilddrüse von *Mystus vittatus*, während mit steigender Röntgendosis die Radiojodaufnahme gehemmt werden konnte.

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[11] M. FONTAINE, J. LELOUP and M. OLIVEREAU, C. r. Seanc. Soc. Biol., Paris *147*, 255 (1953).

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Induction of Obesity in Obese-Hyperglycaemic Mice on Normal Food Intake

It has been alleged that the pathogenesis of the syndrome of obesity and hyperglycaemia in the mouse differs from that of hypothalamic obesity. The basic difference is that in the latter, the regulation of food is deranged, resulting in hyperphagia and obesity (regulatory obesity), whereas in the obese hyperglycaemic mice (*obob*), hyperphagia is secondary to a primary metabolic defect (metabolic obesity)[1,2]. The search for this primary metabolic defect has been intensive, but as yet unsuccessful. STAUFFACHER et al.[3] suggested that the muscle tissue of *obob* is inherently insensitive to insulin and that this insensitivity could conceivably be causally related to the development of the syndrome. We confirmed the presence of insulin-insensitive muscle in *obob*, but concluded that this was secondary to obesity as it disappeared following reduction of body weight[4]. There is also evidence supporting the view that the adiposity of *obob* precedes the occurrence of hyperinsulinaemia, and hyperglycaemia[5].

Thus, it would appear that the *obob* can increase the mass of their adipose tissue in the absence of hyperglycaemia, hyperinsulinaemia, and insulin resistance. The hypothesis that they can also increase it in the absence of excessive food intake was tested in the following experiment: 5 entire litters consisting of the offspring of mice heterozygous for the mutant gene (*Obob*) were taken at weaning (21 ± 2 days). They were weighed, caged individually, and given Thomson's rat diet, in pellet form, for 2 days. Following this, they were given the same food but in powder form, and in amount slightly less (by 0.3 g) to the mean daily food intake of lean mice of the same age and sex. Uneaten food, if any, was measured daily and thus the daily food intake for each mouse was determined.

There were no visibly obese animals at the start of the experiment. However, by about the second month of age, 4 out of the 37 animals had the typical appearance of *obob*, despite the fact that their body-weights did not differ significantly from that of the remaining 33 animals. The use of the binomial theorem demonstrated that there was no significant difference between actual and expected number of *obob* in each litter and in the 5 litters combined ($p > 0.05$). During the experimental period in which food intake was controlled, the body weight gain of these 4 animals exceeded that of the remaining 33 by 48% ($p < 0.001$), despite the fact that their food intake was identical (Figure).

After this period, all animals were allowed to have free access to food for a further 2 months during which time their body weight increased steeply to reach a new plateau. By the end of this period the difference between lean and obese animals' body weight had doubled (Figure).

This experiment demonstrates that the body weight of mice homozygous for the obese gene (*obob*) and that

[1] J. MAYER, Ann. N.Y. Acad. Sci. *63*, 15 (1955).

[2] J. MAYER, Metabolism *6*, 435 (1957).

[3] W. STAUFFACHER, A. E. LAMBER, D. VECCHIO and A. E. RENOLD, Diabetologia *3*, 230 (1967).

[4] C. CHLOUVERAKIS and P. A. WHITE, Metabolism *18*, 998 (1969).

[5] C. CHLOUVERAKIS, E. DADE and R. A. L. BATT, Metabolism *19*, 687 (1970).