procedure. To test the effect of the experimental manipulations on the protective immunity of immunized mice to sporozoites, a second variety of control experiment was done. In this experiment 2 sporozoite-immune and 2 non-immunized mice received 150–250 mg of noninfected liver fragments IP and 1 h later received 50,000 sporozoites i.v. The non-immunized mice in this experiment subsequently developed parasitemias while the sporozoite-immune mice remained uninfected. It is therefore clear that the manipulations involved in this type of transplantation are not sufficient to destroy the specific immunity to sporozoites in these mice.

In the single report of apparently successful transmission of mammalian liver EES, Rossan et al.[7] transplanted liver fragments containing developing EES of Plasmodium cynomolgi from sporozoite infected monkeys to the peritoneums of recipient monkeys at various times during the incubation period of the EES. These liver transplants were only occasionally infective to the recipient monkeys from the 4th to the 6th day post-injection of sporozoites into the donors. However, although the blood of the liver donors contained no infective parasites at these times, it was not technically possible for Rossan et al.[7] to eliminate the possibility of transfer of sporozoites with the liver fragments.

Because we can now totally immunize mice against infection with sporozoites, we have been able to experimentally exclude the possibility that infectivity of inoculated liver is caused by transferred sporozoites. By the results which we have obtained in these preliminary experiments we have demonstrated that it is possible to transmit rodent malaria by transplantation of developing EES from rats to both rats and mice. This procedure may be performed rapidly and provides a practical percentage of EES infected animals for applied experimental studies on this little understood stage of the mammalian malaria parasite. Because we have demonstrated experimentally that neither precocious merozoites nor latent sporozoites were responsible for the transmission of the plasmodium infection, we must conclude that the infectivity was due to viable immature EES in the transplanted liver fragments. We further conclude that at least some of these immature EES underwent continued development to mature merozoites and subsequently successfully invaded the blood streams of their recipient hosts.

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Effects of bilateral lesions of the nuclei habenulae on plasma thyroxine levels in Japanese quail

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Summary. Electrolytic bilateral lesions of the nuclei habenulae were made in male, adult photostimulated quail. Habenular destruction led to a marked decrease in the plasma thyroxine level (40%), whereas sham operated birds did not differ from controls. This result appears to be somewhat different from those obtained in mammals and the mechanisms of habenular-thyroid interrelationships are unknown.

The functional importance of the habenular complex is not completely understood. It was suggested[2–4] that the nuclei habenulae may be involved in thyroid regulation, in mammals. Various experimental data appear to indicate that habenular nuclei and surrounding regions exert an inhibitory effect upon the whole pituitary-thyroid system. However, it was found in other studies that bilateral ablation of the habenula does not interfere with thyroid activity[5,6]. On the other hand, bilateral habenular lesions were found to impair the pineal inhibitory response to a flash of light in unanaesthetized resting quail[7]. It was, therefore, interesting to investigate the habenular involvement in neuroendocrine regulation in birds. The aim of the present study was to explore the thyroid activity in quail, after destruction of the habenular nuclei, by determining the plasma thyroxine level. Adrenal cortical investigations in the same lesioned birds have been discussed elsewhere[8].

Material and methods. 20 adult male quail were used in this experiment. Birds were reared under controlled temperature (26 °C) and artificial lighting (18 h light: 6 h dark; light on: 06.00 h, light off: 24.00 h). Medial and lateral nuclei were located stereotaxically[9] under Equi-Thesin (Jensen Salsbery) anaesthesia. Lesions were produced by passing a direct current (250 μA for 25 sec) through a platinum electrode, 0.14 mm in diameter. In order to destroy completely the median and lateral habenula, it was necessary to place 2 lesions on either side, because these nuclei constitute a rather flat and thin structure. Stereotaxic coordinates are given in the table. After surgery, birds were left at rest in the same environmental conditions. Blood was collected (between 09.00 h and 10.00 h, to avoid any circadian interference) 4 weeks after lesioning. Upon completion of the experiments, the head was perfused with saline followed by 10% formalin-saline. The brain was subsequently frozen and serially cut in 40 μm sections for staining by the Kluver-Barrera technique[10]. Body weight was noted on the day of surgery and at autopsy. Plasma thyroxine levels were determined by the competitive protein binding radioassay developed in birds by Astier et al.[11].

Lesioned birds were compared to intact (no treatment) and sham-operated quail. Sham-operation was performed by fixing the head in the stereotaxic frame, trepanning the skull and lowering the electrode to the habenular stereotaxic coordinates without delivering any current through the electrode.

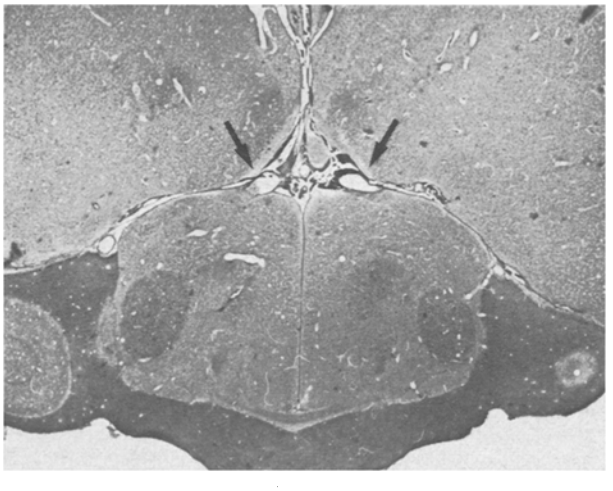
Results and discussion. Results are shown in the table. In intact and sham-operated animals, plasma thyroxine level was found to be quite similar to the values reported in photostimulated male quail by Peczély, (unpublished results: from 3.3 to 4.4 ng/ml vs 4.1 ng/ml in present

Effects of electrolytic lesions of the habenular nuclei on thyroid function in quail

Group	Stereotaxic Coordinates A	L	V	Parameters of electrolytic lesions	Plasma thyroxine level (ng/ml)
Intact (6)*	–	–	–	–	4.15 ± 0.33**
Sham-operated (6)	+ 4 + 3.3	± 0.5 ± 0.6	+ 3.6 + 3.7	–	4.40 ± 0.88
Lesioned (8)	+ 4 + 3.3	± 0.5 ± 0.6	+ 3.6 + 3.7	250 μA – 25 sec	2.49 ± 0.45***

* number of animals; ** mean ± SEM; *** $p < 0.01$ vs controls.
A, anterior; L, lateral; V, vertical, stereotaxic coordinates.

investigations) and in photostimulated drakes[12,13]. Plasma thyroxine was markedly decreased (almost 50%) in quail bearing habenular lesions. Body weight did not differ between lesioned birds (188 g) and controls (177 g). Plasma testosterone levels (Herbuté, Peczély and Jallageas,[1] unpublished results) and adrenal cortical activity[8] were not altered after habenular lesions. Testes were fully developed (4,317 mg). To our knowledge there is no information suggesting that thyroid function might be affected by the habenula in birds. In mammals, Mess[2,14] reported that lesions in the region of the habenular nuclei suppress an iodine deficiency goiter and inhibit the thyroid response to low-level goitrogen administration. Bogdanove[15] also noted that the response to small amounts of propylthiouracil was impaired after habenular lesions. It was suggested[16,17] that the nuclei habenulae are involved in an inhibitory mechanism for TSH secretion. However, Yamada[18] and Matsuda[19] failed to obtain any significant alteration in thyroid function after destruction of the habenular nuclei. Whatever the habenular-thyroid relationships are in mammals, our results suggest that such interrelations are rather different in birds since we obtained an important decrease in thyroxine level after habenular lesions in quail. Histological controls show that electrolytic damage was restricted to the habenular region. The pineal stalk and pineal body were not affected by the lesioning (figure). Fibre connections between the habenular nuclei and lateral preoptic-hypothalamic, septal and interpeduncular regions have been described in birds[20–22] as in mammals.



Histological location of the habenular lesions (→) frontal plane (A 3.6).

Further studies are to be undertaken in order to provide more precise information concerning the habenular influence on the pituitary-thyroid axis in birds and the neural and neuroendocrine involvement in such a regulatory mechanism.

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