increase of the HVA content in the brain stem is not due to hypothermia but rather to a direct action of the drug[1]. This direct effect may be diminished by hypothermia, as seen in the experiments carried out with 10 mg/kg chlorpromazine in different environmental conditions (Figure). The following possible mechanisms of action of chlorpromazine in inducing an increase of cerebral HVA can probably be excluded: (a) Release of dopamine: The endogenous dopamine of the brain stem does not decrease even after repeated administration of chlorpromazine. (b) Diminution of the HVA elimination from the brain: Chlorpromazine does not counteract the decrease of the endogenous HVA in the brain stem which had been elevated by previous administration of the monoamine releaser Ro 4-1284. Furthermore, in rats, as previously reported for rabbits and cats[1,2], chlorpromazine causes no increase of cerebral 5-HIAA, which might be expected if the drug had an unspecific effect on the outflow of HVA. (c) Acceleration of dihydroxyphenylalanine-dihydroxyphenylpyruvic acid transaminase, which might yield HVA without intermediary formation of dopamine: The MAO inhibitors pargyline and iproniazid abolish the chlorpromazine-induced HVA increase although the drugs do not seem to interfere with the transaminase[10]. This indicates that, in the experiments with chlorpromazine alone, the HVA rise might be due to an increased oxidative deamination of dopamine.

The above findings support the hypothesis that the chlorpromazine-induced increase of the cerebral HVA level is caused by an accelerated turnover of dopamine, possibly due to a compensatory enhancement of the dopamine synthesis in consequence of a blockade of dopaminergic receptors in the extrapyramidal centres. Experiments with various other psychotropic drugs are in agreement with this assumption. Accordingly, only neuroleptics which may impair the function of the extrapyramidal centres (e.g. phenothiazines and butyrophenones) increase the cerebral HVA, whereas drugs which interfere relatively little with the extrapyramidal system (thymoleptics, tranquilizers, hypnotics) do not change the HVA level in the brain stem[11].

Zusammenfassung. Im Stammhirn von normothermen Ratten bewirkt Chlorpromazin einen Anstieg von Homovanillinsäure (HVS), der bei Hypothermie verringert wird. Dieser Anstieg geht nicht mit einer Verminderung der Dopaminkonzentration einher und bleibt nach Hemmung der Monoaminoxydase aus. Die durch Vorbehandlung mit einem kurzwirkenden Monoaminfreisetzer (Benzochinolizinderivat Ro 4-1284) erhöhte endogene HVS zeigt nach Chlorpromazin keinen verzögerten Abfall.

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Gonadotrophic Control of Lamina propria and Laminar Collagen of Testis

It has been observed earlier that testicular tubules of toad (*Bufo melanostictus*), during cold torpor, contain huge amounts of sudanophil lipids[1]. Further investigation has indicated these lipids to be ketosteroids and of a similar nature to those found in interstitial cells (unpublished). It is postulated on the basis of the following experiments that these lipids, accumulated in interstitial cells, have pierced through the tubular wall into the tubules, which have been considerably weakened due to the probable absence of gonadotrophin from the pituitary, causing atrophic changes in the testis during the torpid state[2].

Toads, in torpid state, acquired during cold weather (January) were divided into two groups of 24 each, with an average body weight of around 30 g. One group received 25 I.U.[3] of PMS gonadotrophin[4] per animal/day for 7–10 days parenterally through the dorsal lymph sac[5]. After the experiment, testes from each group of animals were fixed in 4% formol, and after the usual paraffin procedure and sectioning they were stained by the PAS-allochrome technique[6] for collagen and lamina propria and compared with those of normal active toads at breeding time.

Figure 1 shows a thin branching disintegrated lamina of testis during torpid state with hardly any trace of collagen around it. String-like elastic recoiled lamina probably indicates the tearing away at high tension. On

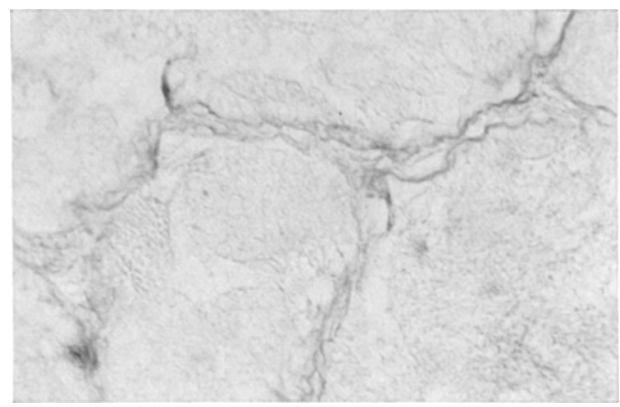

Fig. 1. Toad's testis (cold torpor). Note disintegrating torn lamina and absence of collagenous support. ×192.

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gonadotrophin treatment considerable thickening and repairing of lamina was noted together with disappearance of the lipid from the tubules, which almost resembled those of normal testis in the breeding season (Figure 2). Collagenous support, probably of soluble form, is clearly seen in normal testis, on both sides of lamina. Gonadotrophin was also found to replenish this collagen around lamina propria, indicating that integrity of lamina and laminar collagen probably remains under gonadotrophic control of the pituitary. Increased utilization of testicular hormone may influence the connective tissue formation like oestrogen[7-9].

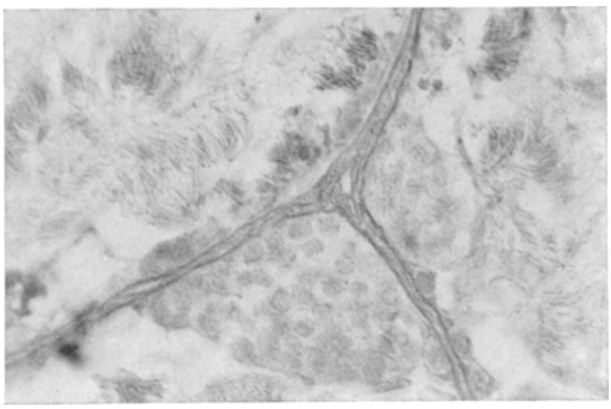

Fig. 2. Toad's testis (breeding season – active season). Note single layer of well-formed lamina supported on both sides by distinct collagen. × 192.

Résumé. Le lipide dans des tubules testiculaires de crapaud à l'état engourdi s'accumule peut-être, en pénétrant dans la lamina propria. Cette lame, comme il a été noté, était faible à l'absence de gonadotropine PMS qui rend forte la lamina propria et aussi la structure collagéneuse qui supporte la lamina propria.

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Tumour Immunity in Rats Following Injection of Homologous Ribosomes

Several authors have published the results of the antitumour effect of ribonucleic acid (RNA) from normal tissues[1-3]. It is probable that, under the influence of RNA from the normal tissue homologous to the tumour, the ability of autonomous growth of the latter is decreased.

The experiments we performed are related to another mechanism whereby RNA from normal tissues can be used to influence tumour growth.

It has been shown[4] that the anti-ribosomal sera precipitate the homologous as well as the heterologous ribosomes, an antigen common to all the ribosomes being RNA itself, and that an autoimmune response can be obtained following injection of liver ribosomes in animals[5].

The results presented here show that tumour homotransplants are rejected in rats in which autoimmunity has been produced with homologous ribosomes.

Male Wistar rats weighing 180–200 g were injected subcutaneously with a 1:1 mixture of ribosome suspension (50 mg of protein) and complete Freund's adjuvant (Difco). Control rats received the adjuvant only.

The ribosomes were prepared according to Zamecnik and Keller[6]. Protein was determined by the method of Lowry[7] and phosphorus as described by Lindberg and Ernster[8].

After 70 days the rats were injected intraperitoneally with 2 million cells of Yoshida ascites tumour. Blood was taken from the ribosome-immunized and control rats, before the injection, for hematological and serological[9,10] tests.

All control animals died between the 14th and the 18th day after tumour transplant, whereas 4 of 15 immunized rats died at the same time, and 11 survived indefinitely. Some of the ribosome-immunized animals had developed a slight anaemia and increased erythrocyte sedimentation rate, but no clear evidence for humoral or cellular factors in the inhibition of the growth of tumour transplant has been obtained.

Riassunto. Ratti trattati con ribosomi omologhi di fegato in «Freund adjuvant» sviluppano una condizione di refrattarietà al successivo trapianto di cellule di epatoma ascitico di Yoshida.

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