Until now the corticotroph cells of reptiles have evidently been interpreted as undifferentiated cells; such a mistake can also easily occur in other vertebrates because of the fineness and scarcity of the specific granules[11-13]. On the contrary, they are clearly distinguishable from the X cells, which are also situated in the rostral zone of the pars anterior, not only by their different affinity to stains, as has already been mentioned, but by their morphological features. Normally the X cells are cylindric and the supranuclear zone of their cytoplasm appears full of closely packed granules; these granules are small but easily discernible with the light microscope, and are definitely orangeophilic with the staining methods used. The rostral localization of the corticotroph cells in the animal studied agrees with that which has been observed in birds[5] and fishes[7]. In the latter, cytoplasmic vacuolation as well as nuclear and nucleolar hypertrophy have also been noticed as an effect of metopirone administration[14]. The affinity of cytoplasmic zones for iron haematoxilin can be explained by the usual abundance of ergastoplasm in this cellular type[12].

In conclusion, a third type of non-mucoproteinaceous secretory cells, responsible for corticotrophin production and not previously described in reptiles, must be admitted in the anterior pituitary gland of *Cnemidophorus l. lemniscatus*.

Resumen. Mediante la administración de metopirona pudo demostrarse en la hipófisis anterior de *Cnemidophorus l. lemniscatus* la existencia de un tercer tipo de células secretoras no mucoprotídicas, responsables de la producción de corticotrofina y no identificadas hasta ahora en reptiles. Estas células se encuentran en la porción rostral del lóbulo y, en los animales testigos, resultan cromófobas con las coloraciones efectuadas. Por acción de la metopirona sufren considerable hipertrofia e hiperplasia y aparecen en su citoplasma gránulos gruesos y relativamente escasos que presentan moderada afinidad hacia la hematoxilina férrica.

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The Course of Lymphocytic Choriomeningitis Virus Infection in Mice Treated by Phytohaemagglutinin

The effect of phytohaemagglutinin on the antibody response and the homograft rejection has been investigated by several authors[1]. The experimental data and the conclusions of the authors concerning the effect of phytohaemagglutin treatment on the immunological responses seem to be contradictory. Considering that the consequences of lymphocytic choriomeningitis (LCM) virus infection depend on the immune status of the mice and that the fatal choriomeningitis fails to develop in animals depressed immunologically[2-8], intracerebral infection with LCM virus was chosen as the experimental method for studying the effect of phytohaemagglutinin on immunological reactivity.

Materials and methods. 5-week-old, inbred mice of strain 'A', weighing on the average 15 g, were used in the experiments. The total quantity of phytohaemagglutinin applied in the treatment, as well as the time of the virus infection varied per experiment. In each experiment 30 mice were injected i.p. with 0.4 ml (0.4 mg) of Phytoclin (Wellcome Research Laboratories), while an equal number of controls received 0.4 ml of physiological NaCl solution. Each time half of the mice in every experiment were injected intracerebrally with the pre-titrated 100 LD_{50} dose of LCM virus. Each experiment thus covered 4 groups, as specified in the Table. Absolute lymphocyte counts were taken at intervals and also the weight was checked several times of the mice of PHA and control groups. In the mice of PHA + LCM and C + LCM groups, the typical neurological symptoms of the infection with subsequent death of the animals were observed. The brain of the dead animals was studied histologically using hematoxylin and eosin staining.

Results and discussion. The experimental data are summarized in the Figure. In experiments 1 and 2 the animals were injected with 3 times 0.4 mg and 6 times 0.4 mg of phytohaemagglutinin respectively. The LCM virus was inoculated on the last day of the phytohaemagglutinin treatment. In experiments 3 and 4 the treatment with phytohaemagglutinin was started on the 3rd and 4th day after the LCM virus infection, injecting 3 times 0.4 mg and 4 times 0.4 mg of phytohaemagglutinin, respectively.

As regards the effect of the phytohaemagglutinin treatment on the course of the LCM virus infection, the result was the same in each of the experiments. The

Groups	PHA	PHA + LCM	Control	C + LCM
Treatment	PHA i.p.	PHA i.p.	Phys. NaCl i.p.	Phys. NaCl i.p.
Infection with LCM virus	–	100 LD_{50} i.cer.	–	100 LD_{50} i.cer.

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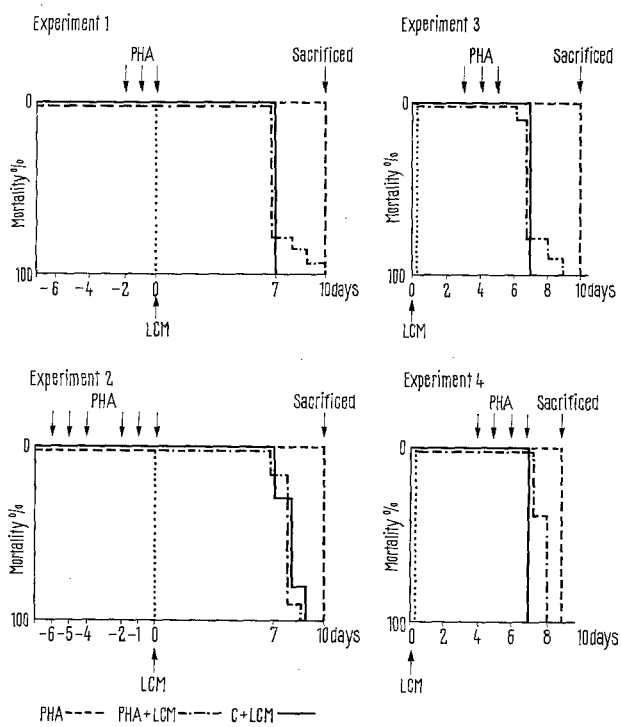
typical neurological symptoms of the virus infection were apparent after the usual incubation period in the mice of the PHA + LCM group also, and the animals died at the same rate as those of the infected control (C + LCM) group, on the 7th–9th days. Histological analysis showed in their brain the typical symptoms of lymphocytic choriomeningitis. In the non-infected groups treated with PHA none of the animals were lost, they were apparently healthy and no loss of weight could be observed as compared with the controls. The phytohaemagglutinin treatment did not cause any appreciable change of the number of circulating lymphocytes. Having lost the animals infected with the LCM virus, the mice of the PHA and control groups were sacrificed. *Splenomegaly* was observed in the mice which were killed 2 days after the administration of phytohaemagglutinin (experiment 4). *Splenomegaly* could not be shown in the animals which were sacrificed on the 5th–10th days after the astl phytohaemagglutinin injection (experiments 1, 2 and 3). This observation is consistent with the earlier findings which showed *Splenomegaly* to be the most pronounced on the 3rd day after the phytohaemagglutinin injection[9,10].



Mortality rate of mice inoculated with LCM virus and treated with phytohaemagglutinin.

It turned out from the present experiments that the phytohaemagglutinin treatment did not affect the development of intracerebral LCM virus infection, the animals died after exhibiting the typical neurological symptoms and in the cerebral tissues the histological characteristics of lymphocytic choriomeningitis could be identified. Our earlier observations as well as those of other authors have shown that there exists a relationship between the neurological symptoms of the LCM virus infection with subsequent development of the fatal hypersensitivity reaction and the number of circulating lymphocytes[6,8,11]. Since the phytohaemagglutinin treatment does not induce any permanent decrease in the number of circulating lymphocytes, this may be the reason why the development of fatal lymphocytic choriomeningitis could not be suppressed by the phytohaemagglutinin treatment.

The results of the present investigation, which showed that the phytohaemagglutinin treatment does not suppress the immunological reactions of mice to intracerebral LCM virus infection, confirm the opinion of those who do not believe in the immunosuppressing effect of phytohaemagglutinin.

Zusammenfassung. Es wurde festgestellt, dass die Virusinfektion in mit LCM-Virus intrazerebral infizierten Mäusen bei Phytohämagglutinin-Behandlung gleich verläuft wie in unbehandelten Kontrolltieren. Dies stützt die Annahme, Phytohämagglutinisierung vermindere die immunologische Reaktivität des Organismus nicht.

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Differences Between Plasma and Serum Mediated Chemotaxis of Leukocytes

We have recently shown in experiments in vitro that sera from normal rabbits contain chemotactic activity for both macrophages and neutrophils[1]. The specific activity in normal sera for macrophages was due to a fraction with the approximate molecular weight of 200,000, whereas the distinct main activity for neutrophils was located in a fraction with a molecular weight between 5000 and 35,000. Such low molecular weight neutrophil cytotaxins have also been observed by other workers and were found to be split products of complement components[2–4].

So far no explanation has been put forward for the variable presence of chemotactic activity in normal serum. The question arises whether these cytotaxins are already present in plasma or whether they are formed during the blood clotting process. In the latter case it is likely that a link between chemotaxis, blood coagulation and complement activation exists.

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