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EFFECT OF CORTICOSTEROIDS ON PASSIVE AVOIDANCE BEHAVIOUR OF RATS

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The effect of corticosteroids on passive avoidance behaviour of the rat has been investigated in two experimental situations based upon light-avoidance and thirst drive. Administration of a single dose of cortisone led to a suppression of both immediate and long-term retention of passive avoidance elicited by single or repeated shock trials in a light-avoidance situation. The effect of cortisone was a function of shock intensity eliciting the avoidance response. The higher the shock intensity, the more cortisone was necessary to suppress passive avoidance.

Passive avoidance reflected in the shock trial in a spine discriminative conditioned situation motivated by thirst was also suppressed by a single dose of dexamethasone. The reaction between the shocked and non-shocked side was, however, enhanced by dexamethasone.

A number of behavioural and electrophysiological experiments has demonstrated that the central nervous influence of corticosteroids is exerted through the enhancement of internal inhibitory processes of Pavlovian terminology (Enődözi and Lissák 1962; Bohus and Endődözi 1965; Bohus and Kókáni 1969; Endődözi 1969). It has also been shown that this effect of steroids is exerted directly on the forebrain structures (Bohus 1970) related to the elaboration of internal inhibitory influences (Lissák and Endődözi 1967). However, some observations cast doubt on the view that an enhancement of internal inhibition is the sole mechanism of the central nervous action of cortico-steroids. Thus, both behavioural (Bohus 1968, 1970; van Wimersma Greidanus and de Wied 1969) and electrophysiological (Slesher et al. 1966; Endődözi 1969) observations suggest the involvement of the reticular activating system in the action of cortico-steroids.

The present experiments were aimed at investigating the influence of corticosteroids on passive avoidance behaviour of rats in order to determine if any other mechanism than an enhancement of internal inhibition may be involved in the behavioural influence of corticosteroids.

Methods

Adult male albino rats of an inbred strain were used. They were fed on rat chow and allowed to drink ad libitum unless otherwise indicated.

Passive, one-trial shock avoidance behaviour of rats was studied in two different motivated situations. Inhibition of an inherent light-avoidance response was investigated in a conditioning apparatus which consisted of two plywood compartments connected by a 10 × 10 cm
TOVÁBBI VIZSGÁLATOK A CORTICOSTEROIDOKNAK
A PATKÁNYOK PASSZÍV ELHÁRÍTÓ MAGATARTÁSÁRA
KIFEJEZTETT HATÁSÁVAL KAPCSOLATBAN

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Corticosteron, 11-desoxycorticosteron (DOC) és a szintetikus 
6dehydro-16methylen-hydrocortison (6dh-16m-hydrocortison) hatá-
sát vizsgáltuk a patkányok szomjazási hajtóerőre épített passzív 
elhárító magatartására.

Corticosteron és 6dh-16m-hydrocortison szisztémás adása csök-
kentette az elektromos áramütés által létrehozott passzív elhárító 
magatartás latencia idejét közvetlenül a shockolás után és 24 óra 
múlva. Hasonló hatás volt megfigyelhető corticosteron agyi impla-
tantciója után is, ha a steroidot az elülső hypothalamusba, rostra-
lis septumba, dorsalis hippocampusba, vagy a középvenali thalami-
cus magvakba juttattuk, 6dh-16m-hydrocortison és DOC implantá-
tum ezen strukturák többségében hatástalan volt.

Megfigyeléseink arra utalnának, hogy a corticosteroidok maga-
tartási hatásának kialakulásában a hormonok közvetlen központi 
idérgendszeri hatása jelentősebb, mint az ACTH szekréció csök-
kentésén keresztül érvényesülő effektusuk. A hatás létrejöttéért az 
előágyi strukturaikon kívül a középvenali thalamikus magvak is 
felelősek.
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In the last years numerous investigators have studied the influence of gonadotrophic and gonadal hormones on the electrical activity of the central nervous system (CNS) and on behavioural processes (Kobayashi et al. 1962, Barrington and Cross 1963, Ramírez et al. 1967, Endröcz et al. 1968, Endröcz et al. 1969a, b, Újvári and Michael 1970, Branchey et al. 1971, Pfaff et al. 1971 et al.). Nevertheless, in this respect relatively few data are known about the effect of human chorionic gonadotrophin (HCG). In 1969 Kawakami and Sawter described the HCG-induced electrical and behavioural after-reactions in rabbits, namely the sleep pattern and the hippocampal hyperactivity of EEG and the changes of feeding behaviour. According to Umeda (1969) HCG plays an important role in the changes of the brain activity level; depending upon the level of progesterone or oestrogen in the animal, it can lower or increase the excitatory or the inhibitory level of the CNS. Telegdy and Rózsahegyi (1971) and Telegdy et al. (1971) have found that chronic HCG administration accelerates the extinction of a conditioned avoidance response in castrated female rats, and decreases the exploratory activity of the animals. In an earlier paper we have reported certain electrophysiological and behavioural changes in ovariectomized rats after intraperitoneal or intravenous HCG administration in acute and chronic experiments, namely the augmentation of amplitude of hypothalamic and hippocampal evoked potentials elicited by electrical stimulation of the vagina, the facilitation of stimulus-induced EEG after-synchronization, the increase of self-stimulation rate briefly after the HCG injection and its decrease following chronic hormone treatment (Hartmann et al. 1971). Although in these experiments the direct effects of HCG on the CNS have been clear, it remained to be solved whether the whole brain or only certain parts of the brain are responsible for the HCG-induced changes. In order to clarify this problem, in the present investigation the effect of the intra-cerebral administration of HCG has been studied on the brain electrical activity.

**Methods**

The experiments were performed on ovariectomized R-Amsterdam rats weighing 200—220 g. The animals were ovariectomized three months prior to the experiments. In the electrophysiological study the animals were anaesthetized by ether during the electrical implantation and the introduction of the tracheal cannula. After the surgical procedure the animal was immobilized by tubocurarine and respirated artificially.

For recording the electrical activity, stainless steel electrodes were implanted into the antero-lateral hypothalamus and hippocampus and silver-void electrodes were placed on the parietal cortex bilaterally. Bipolar stainless steel electrodes were

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