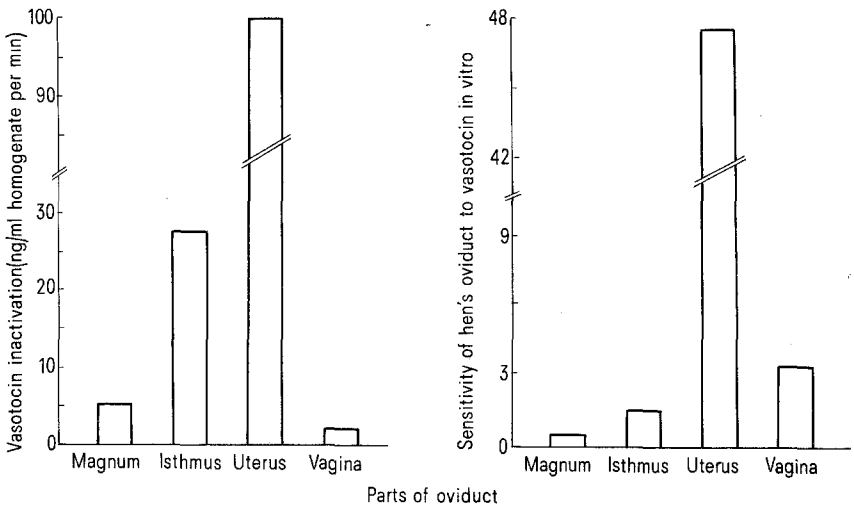

Comparison between the degree of vasotocin inactivation by hen's oviduct homogenates and sensitivity of the oviduct to vasotocin. Sensitivity of uterus taken as 100 (calculated by RZĄSA[13]).

hormone. Of the 4 parts of oviduct examined, the uterus showed the greatest sensitivity to vasotocin and simultaneously possessed the highest ability to inactivate this hormone.

The total protein content in tissue homogenates ranged from 6.65 (isthmus) to 40.92 mg/ml (magnum) (Table II). No relationship between the protein concentration and the vasotocin-inactivating activity was observed. Tissue inactivating ability expressed in ng of inactivated vasotocin/mg of protein per 1 min was 3 times greater in the case of uterus than in the liver. However, the rate of vasotocin inactivation by these tissues expressed in half-life was similar (Table I).

Ability of the hen's liver and kidney tissues to inactivate neurohypophysial hormones resembles that found in mammals. On the other hand, the striking phenomenon observed was the high vasotocin-inactivating activity of uterine portion of hen's oviduct. In mammals, uterus inactivates oxytocin approximately 10 times less than the liver and kidney[14,15]. It is probable that the high ability of hen's uterus to inactivate vasotocin is associated with a great sensitivity to vasotocin, as found in vitro and in vivo studies[4,8,13]. This point of view might be in general agreement with studies on the other hormones which shows that the tissues which are metabolically sensitive to the hormone possess simultaneously a high degree of inactivating activity to this hormone[16].

As found in our previous work, the inactivation of oxytocin in hen's, by contrast to mammals, occurs also in serum, irrespectively of the sex[10]. The enzyme of L-cystine aminopeptidase activity identified in hen's serum could be responsible for this inactivation[17]. In relation to vasotocin, HASAN and HELLER[9] did not observe an ability of hen's plasma to inactivate this hormone.

The results reported give no information about the mechanisms by which the biological activity of the vasotocin dissappeared from the circulation of the hen. It may be, that in the process of its inactivation both the aminopeptidases and disulfide reductases take part. It cannot be excluded also that bird tissues contain an enzyme of the endopeptidase type, similar to that found in rat[18] and kidneys[2] and possessing ability to release of glycinamide from the molecule of neurohormone[19].

Zusammenfassung. In vitro wurde die Inaktivation von Vasotocin in einigen Gewebehomogenaten (Leber, Niere, Eileitersegmenten, Magnum, Isthmus Uterus, Vagina) festgestellt. Die grösste Inaktivation wurde bei den Homogenaten von Leber und Uterus gefunden.

E. BRZEZIŃSKA-ŚLEBODZIŃSKA and J. RZĄSA

Agricultural University, Institute of Applied Physiology of Animals, Krakow (Poland), 17. May 1972.

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Effect of *Bordetella pertussis* Vaccine on Plasma Corticosterone Level and on ACTH-Induced Corticosterone Secretion in Rats

Both adrenalectomy and *Bordetella pertussis* vaccine (BPV) increase the sensitivity of mice and rats to anaphylactic shock, shock-mediators, various toxic and stress effects[1–6]. It was earlier suggested by KIND[7] that BPV exerts its effect by injuring the adrenal cortex. However, this supposition has not been confirmed in mice[2,8]. Our recent investigations regarding the histamine metabolism of BPV-treated rats again raised the possibility that reversible adrenal insufficiency develops following BPV sensitization[9]. The purpose of the present study was to examine whether BPV influences plasma corticosterone level and ACTH-induced corticostrone secretion in rats.

Materials and methods. Female Wistar rats (150–200 g) were maintained on standard diet and drinking water ad

libitum. BPV was administered i.p. in a dose of 3×10^{10} organisms. In the first experiments animals were killed and plasma corticosterone concentration was determined before and at various intervals after BPV inoculation. In the second experiments rats were divided into 4 groups. The animals of group 1 and 2 received 6 U/100 g ACTH (cortrophine, Organon) subcutaneously. The animals of group 2 had been inoculated with BPV 4 days earlier. Blood samples were taken 2 h after ACTH injection.

Groups 3 and 4 were treated orally with 80 µg dexamethasone (Organon) 18 h prior to corticosterone determination, and drinking water containing 20 µg/ml dexamethasone was given during the last 18 h. These animals were injected with 4 mU/100 g of ACTH i.v. and blood was taken 18 min later. Rats of group 4 were pretreated with BPV 4 days before ACTH administration.

In all experiments rats were killed between 10.00 and 11.00 h and blood was collected from the cervical vessels. Plasma corticosterone level was measured according to the fluorimetric method of ZENKER and BERNSTEIN[10] as modified by PURVES and SIRETT[11]. Each experimental result presented in the figures indicates the average of 10 animals and the corresponding standard deviations. The results were statistically evaluated by Student's *t*-test.

Results and discussion. As shown in Figure 1 plasma corticosterone level significantly decreased 4 and 8 days after BPV treatment and returned to the original value on the 18th day.

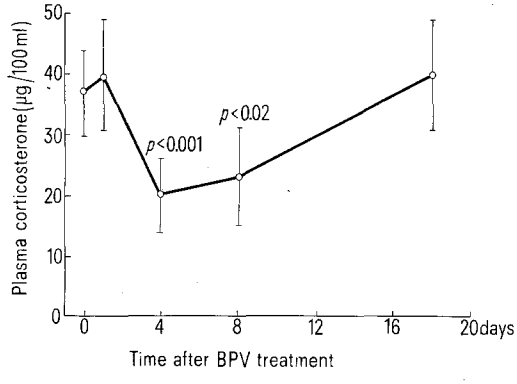

Fig. 1. Effect of BPV treatment on plasma corticosterone level in rats. Significance was related to untreated animals.

6 U/100 g of ACTH induced an elevation in plasma corticosterone level both in normal and in BPV-treated animals (Figure 2). The rate of increase was similar in both groups. Dexamethasone, by means of its feed-back effect on endogenous ACTH secretion, suppressed plasma corticosterone level. I.v. injection of exogenous ACTH increased corticosterone secretion both in dexamethasone and in BPV + dexamethasone-treated rats similarly.

As it was already mentioned, both adrenalectomy and BPV increased the sensitivity of rats and mice to various damaging effects. Our earlier experiments showed that the changes of tissue histamine[9] and 5-hydroxytryptamine (5-HT)[12] levels were similar in adrenalectomized and BPV-treated rats. Furthermore, in adrenalectomized rats BPV did not influence tissue histamine metabolism and cortisone inhibited changes caused by BPV[9]. Our present findings demonstrate a decrease in plasma corticosterone level following BPV inoculation. This decrease may explain – at least in part – certain similarities between the effect of adrenalectomy and BPV. Thus, it may be one of the factors causing increased sensitivity of BPV treated rats. However, some other effects of BPV, e.g. its influence on insulin secretion[13] and anaphylactic protease production[14,15], could hardly be explained by a reversible decrease of blood glucocorticoid level.

The reason of the decrease of corticosterone level is not known. This effect of BPV is presumably not due to an inhibition of corticosterone synthesis as the response of adrenal cortex to ACTH remained intact. As possible reasons, a disturbance in ACTH secretion, increased corticosterone catabolism and other possibilities could be taken into account. Obviously, further experiments will have to be carried out to clarify this problem.

Zusammenfassung. Der Corticosteron-Gehalt des Rattenplasmas wurde durch BPV-Behandlung vorübergehend reduziert, obwohl die ACTH-Wirkung auf den Corticosteron-Spiegel im Plasma unverändert blieb.

B. CSABA and L. MUSZBEK

Department of Pathophysiology, University Medical School, Debrecen 12 (Hungary),
25 June 1972.

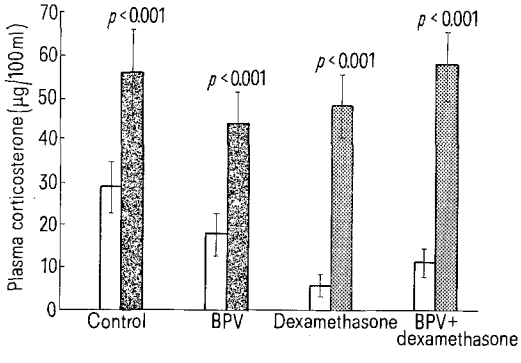

Fig. 2. Effect of BPV pretreatment on ACTH-induced corticosterone secretion in rats. □, without ACTH; ▨, 2 h after 6 U/100 g ACTH/ s.c./; ▦, 18 min after 4 mU/100 g of ACTH/ i.v./. Significance was related to animals without ACTH.

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