

*Brief Communication*

## URINARY OESTROGENS IN HEIFERS TREATED WITH PREDNISOLONE DURING THE OESTROUS CYCLE

Heifers treated with a glucocorticoid during the oestrous cycle, displayed increased follicular activity and small corpora lutea compared to untreated controls (*Tomasgard 1976a*), but showed no signs of oestrogen production during the treatment.

Three heifers were treated with 30 mg prednisolone per 100 kg body weight from day 2 after oestrus (i.e. the day after ovulation) until slaughter. One heifer was slaughtered on day 13, and two others on day 26 after last oestrus. Two untreated heifers served as controls.

For quantitative collection of urine the animals were kept in stalls equipped with rubber mats. In one of the prednisolone treated heifers urine was collected only from day 18 to day 21 after last oestrus. Samples of the 24 hrs.' urine were stored at  $-20^{\circ}\text{C}$  until assayed by the method of *Lunaas et al. (1974)* for oestrone and oestradiol-17 $\alpha$ .

Referring to Table 1 the urinary excretion of oestrogens was found to be relatively low in the prednisolone treated heifers.

Table 1. Urinary oestrogen excretion ( $\mu\text{g}$  per 24 hrs.) in two normal heifers and in three heifers treated daily with 30 mg prednisolone per 100 kg body weight. The treatment was started on day 2 after oestrus. One of the treated heifers was slaughtered on day 13 after last oestrus, and in one of the treated heifers urine was collected only from day 18 to day 21 after oestrus.

Days after oestrus		Untreated			Prednisolone treated			P*
		mean	n	range	mean	n	range	
2—7	E <sub>1</sub>	0.75	10	0.3—2.5	0.24	10	0.08—0.6	< 0.01
	E <sub>2</sub>	2.10	10	0.6—4.2	0.64	10	0.40—1.2	< 0.01
	E <sub>1</sub> + E <sub>2</sub>	2.85	10	0.9—4.5	0.88	10	0.48—1.5	< 0.01
8—17	E <sub>1</sub>	1.64	20	0.6—6.0	0.44	14	0.01—1.3	< 0.01
	E <sub>2</sub>	1.81	20	1.0—3.2	0.55	14	0.05—1.3	< 0.01
	E <sub>1</sub> + E <sub>2</sub>	3.45	20	2.1—8.0	0.99	14	0.25—2.0	< 0.01
18—19	E <sub>1</sub>	1.70	4	1.2—2.4	0.55	4	0.01—1.3	< 0.01
	E <sub>2</sub>	7.00	4	3.2—12.8	0.53	4	0.01—0.8	< 0.015
	E <sub>1</sub> + E <sub>2</sub>	8.70	4	4.6—10.6	1.08	4	0.02—1.9	< 0.015
20—21	E <sub>1</sub>	2.47	4	1.2—4.5	0.12	4	0.01—0.4	< 0.015
	E <sub>2</sub>	9.83	4	4.2—13.5	0.60	4	0.01—1.4	< 0.015
	E <sub>1</sub> + E <sub>2</sub>	12.30	4	6.6—18.0	0.72	4	0.02—1.4	< 0.015

E<sub>1</sub> = oestrone. E<sub>2</sub> = oestradiol-17 $\alpha$ . \* Wilcoxon two-sample test.

Under normal conditions the excretion of oestrogens is somewhat increased from day 4 after oestrus (Fig. 1). This increase is thought to be due to oestrogens produced in the follicles before the corpus luteum attains maximal function (*Lunaas 1973*).

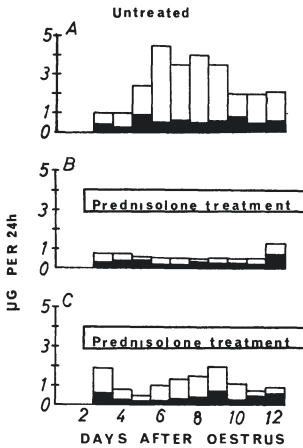


Figure 1. Urinary excretion of oestrone (solid bars) and oestradiol-17α (open bars) from day 3 to day 12 after oestrus in a representative, normal heifer, and in two prednisolone treated heifers. The heifers were treated daily with 30 mg prednisolone per 100 kg body weight from day 2 after oestrus.

The nature of the change in ovarian function which apparently results in a reduced ability to produce oestrogens during treatment with the glucocorticoid is unknown. In rats, dexamethasone treatment has been shown to prevent LH release (*Baldwin & Sawyer 1974*) which is presumably necessary for the production of oestrogens in the follicles. However, short-term prednisolone treatment of heifers during pro-oestrus and oestrus did not prevent the increase in oestrogen excretion at oestrus or inhibit ovulation (*Tomasgard 1976b*), and probably thus did not prevent LH release.

In primates there is a considerable production of oestrogens in the corpus luteum by cells originated from the theca (*Baird et al. 1975*). The fact that the normal increase in oestrogen excretion in late metoestrus in the bovine coincides with the growth of thecal cells in the corpus luteum (*Donaldson & Hansel 1965*) suggests that these cells could temporarily produce oestrogens.

Accepting this possibility, the apparent failure of ovarian oestrogen production found during late metoestrus (Fig. 1) might thus be explained by an interference of the glucocorticoid with the development and/or function of the thecal elements of the corpus luteum.

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