epinephrine produces an additional beta adrenergic blockade[9]. Research now in progress shows that in laparotomized rats injected with epinephrine and phentolamine (another alpha adrenergic blocker), plasma fibrinogen levels increase to values similar to those observed in the LEp group.

The absence of alteration in the fibrinogen level in normal intact rats injected with propranolol would indicate that an eventual decrease of epinephrine binding to hepatic cell receptors does not influence the plasma fibrinogen levels when tissue injury is not performed. On the contrary, it confirms that epinephrine effects on fibrinogen are only evidenced in rats submitted to tissue injury.

The beta adrenergic effect of epinephrine may be indirect, through its action on the synthesis of TSH or insulin hormones[18-20]. These hormones contribute to the increase of plasma fibrinogen in tissue injury[3,4]. In conclusion, according to our results, epinephrine would be responsible for the increase of plasma fibrinogen level in laparotomized rats.

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Stimulation of the incorporation of ^{3}H-leucine into proteins by oestradiol in the foetal uterus of the guinea-pig

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Summary. Oestradiol treatment increases the incorporation of ^{3}H-leucine into acid insoluble proteins in the foetal guinea-pig uterus (50–65 days of gestation) 10 times above control values by 8 h and 20 times by 24 h after administration of oestradiol to the mother.

The foetal guinea-pig uterus has been shown to respond to oestrogen treatment by an increase in wet weight[1] and a great stimulation of the progesterone receptor protein[2]. Moreover, these responses can be correlated with the translocation and nuclear retention of oestrogen receptor in the foetal uterus[3]. The present study now demonstrates that oestradiol also has an effect on the in vivo incorporation of ^{3}H-leucine into acid-insoluble proteins in the foetal uterus.

Pregnant guinea-pigs of the Hartley albino strain were obtained from a commercial breeder (Centre d'Elevage R. Janvier, Le Genest, France) and varied from 50 to 65 days of gestation. These animals were injected s.c. with 1 mg oestradiol/kg b.wt in 50% ethanol-saline (controls were given vehicle alone). 8 h or 24 h later, the animals were anesthetized with ether, the foetuses were exposed by laparotomy and each female foetus was injected with 60 µCi (1.6 µg or 0.012 µmoles) of ^{3}H-leucine (sp.act. 5 Ci/mmole, New England Nuclear, Dreieich, FRG) in 0.25 ml 0.1 N HCl. The foetuses were replaced in the abdomen and 30 min later the animals were sacrificed and the foetal uteri excised. Each uterus was homogenized in 1 ml cold distilled water and 1 ml of 1 N perchloric acid (PCA) was added. After 15 min, the homogenate was centrifuged at 900 × g for 10 min and the acid-insoluble precipitate was washed 4 times with 2 ml cold 0.2 N PCA. The washed precipitate was resuspended in 2 ml 0.5 N PCA, hydrolyzed at 90 °C for 30 min and centrifuged to obtain a supernatant which was assayed for DNA by the method of Burton[4] and a final precipitate which was completely dissolved in 2 ml of 0.1 N NaOH by heating at 50 °C for 30 min. Protein concentration was measured as described by Lowry et al.[5] and an aliquot was counted (46% efficiency) in Ready-Solv HP (Beckman Instruments, Gagny, France).

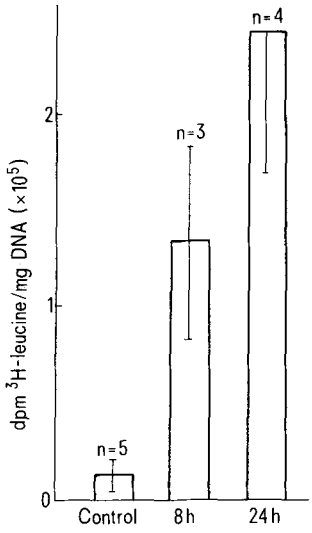
The figure shows that oestradiol treatment increases the in vivo incorporation of ^{3}H-leucine into acid-insoluble protein 10 times above control values in the non-treated animals by 8 h after treatment and 20 times by 24 h ($p < 0.001$ between the 8-h and 24-h values). Thus, the foetal guinea-pig uterus

Effect of oestradiol treatment on total acid-insoluble protein and total DNA in foetal guinea-pig uterus

	mg protein/ g tissue	mg DNA/ g tissue	mg protein/ mg DNA
Untreated control	75.22 ± 5.11	5.47 ± 0.83	15.11 ± 1.80
8-h oestradiol	61.21 ± 2.00	4.89 ± 0.75	13.47 ± 3.00
24-h oestradiol	66.05 ± 4.59	4.45 ± 0.50	15.97 ± 3.20

Protein and DNA were determined in the samples described in the figure. The values represent the means ± SEM.

also exhibits this parameter of oestrogenic response which can be demonstrated in vitro only from 15 days postnatally in the rat[6] and which then increases to 25 days. However, even at 25 days, the rate of amino acid incorporation only increases 1.8 times in the rat. It is interesting to note that increased progesterone receptor concentrations can first be detected in the foetal guinea-pig uterus by 6 h after oestradiol treatment and are maximal by 24 h[3]. Nevertheless, although increased amino acid incorporation could be demonstrated, there was no concomitant net increase in total protein concentration, as shown in the table, and at the same time there was no increase in total DNA. Whether this increased ^{3}H-leucine incorporation reflects increased protein synthesis or a mere variation of the intracellular amino acid uptake or pool remains to be elucidated. However, the observation that oestradiol can stimulate amino acid incorporation into proteins before significantly increasing the total protein concentration has also been made in the studies of the postnatal development of uterine responsiveness to oestradiol in the rat[7]. Moreover, the effects of oestradiol on the incorporation of ^{3}H-thymidine into DNA and on the total uterine DNA content are also dissociated temporally in the rat. Thus, the foetal uterus of the guinea-pig also responds to oestradiol treatment by an increased incorporation of ^{3}H-leucine into total uterine proteins without any net increase in the total concentration of uterine protein, and this now represents another parameter of oestrogenic action which can be provoked in the foetus.



Effect of oestradiol treatment on the incorporation of ^{3}H-leucine into acid-insoluble proteins of the foetal guinea-pig uterus. Pregnant guinea-pigs were injected with 1 mg oestradiol/kg b.wt and after 8 h and 24 h (controls received vehicle alone) the female foetuses were injected in vivo and in situ with 60 µCi of ^{3}H-leucine. After 30 min, the animals were sacrificed and the foetal uteri were excised. A PCA precipitate of the uterine proteins was prepared and the radioactivity counted. The columns represent the means ± SEM of n determinations.

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Changes in gastric parietal cells during prolonged intermittent corticosteroid treatment

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Summary. Immature A/J mice were treated for up to 7 weeks with intermittent doses of triamcinolone hexacetonide and were thereafter allowed to recover for 7 weeks. Structural examinations and morphological measurements were performed on the parietal cells in the gastric mucosa. By the 3rd injection a significant decrease was noted in the number of the above cells - a feature that lasted throughout the experimental period. In contrast, the diameter of the parietal cells increased. However, following recovery, the latter returned to their normal size.

Gastrointestinal complications that have been associated with corticosteroid therapy include peptic ulcer[2]. Possible mechanisms whereby corticosteroids may cause gastric ulceration have been investigated. Treatment of immature rats with high doses of glucocorticoid hormones has been shown to retard significantly the proliferative activity of cells along the gastric mucosa[3]. Reduction in the quantity of gastric mucus and changes in its quality have been described[4] along with a reduction in the turnover of gastric epithelial cells[5,6]. Among the various strains of mice, the A/J strain exhibits an exceptionally high degree of sensitivity to glucocorticoids due to the high level of glucocorticoid receptors in their cells[7]. The purpose of the present study was to examine: a) the effects of prolonged intermittent corticoid administration on the gastric mucosa, and b) the potential of recovery of mucosal cells following longterm hypercorticoidism. To the best of our knowledge, this study is the first one to use the highly sensitive A/J strain for such investigations.

Material and methods. 7-week-old A/J mice, fed Purina laboratory chow and drinking water ad libitum, were given intermittent (every 4 days) i.m. injections of 4 mg/kg b.wt of triamcinolone hexacetonide (Aristospan, Cyanamid Co.). Nontreated animals served as controls. Test and control animals were sacrificed after 3, 7 and 14 consecutive injections, respectively. Additional groups of treated and control animals were allowed to recover for 7 weeks following the cessation of the hormonal treatment. On sacrifice, the stomachs were removed intact and were immediately fixed in a mixture of 4% formaldehyde and 2%