highest doses. The reduced inhibition of the highest $PGF_{1\alpha}$ dose may result from failure of PGE receptors to discriminate at high $PGF_{1\alpha}$ concentrations.

The E and F prostaglandins exert antagonistic effects on aldosterone production at long time intervals. However the E prostaglandins, particularly PGE_2, mimic the inhibitory effects of $PGF_{1\alpha}$ and $PGF_{2\alpha}$ at the lower doses and earlier time intervals examined. Further PGE_1 and PGE_2, although equipotent in aldosterone release at the longest time interval, produce divergent effects at the earlier intervals studied. Finally, E prostaglandin stimulation of aldosterone synthesis and release may be separate events.

Dexamethasone suppression of ovulation in PMS-treated immature rats

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Summary. A single injection of 2.0 mg/kg dexamethasone (DXM) administered at 51 h after pregnant mare serum gonadatropin (PMS) treatment inhibited both ovulation and luteinization. S.c. injection of human chorionic gonadotropin (HGG) caused ovulation ond luteinization in DXM-PMS-treated rats, whereas treatment with ACTH failed to overcome the DXM inhibitory effect. These findings are interpreted to indicate that DXM inhibits ovulation through a mechanism which might involve the central nervous system.

Dexamethasone administration in the rat usually results in inhibition of ovulation. This inhibition was suggested as a direct effect of the lack of a physiologically functioning adrenal cortex, thought to produce preovulatory progesterone which facilitates ovulation[1]. There is some supporting evidence for this: a single injection of deoxycorticosterone or corticosterone facilitates ovulation in PMS-treated immature rats[2], ovulation rate in PMS-treated rats was reduced when animals were adrenalectomized[3]. However, there are several anomalies to the hypothesis. For example, PMS-induced ovulation in the immature rat could be inhibited by the concurrent administration of ACTH[3]. Similar results were obtained in the adult rat[1]. The present experiments were designed to study further the site of dexamethasone action inhibition ovulation in PMS-treated immature rats.

Materials and methods. Sprague Dawley 22-day-old rats were housed 14:10 light-dark cycle (6.00 a.m. to 8.00 p.m. eastern time). They were injected s.c. with 25 IU of pregnant mare's serum gonadotropin (PMS, Sigma) on the 24th day of life. Dexamethasone (1,9-fluoro-16, methyl cortisol, DXM, Sigma) was given i.p. at 12.00 noon on the 26th day, 51 h following PMS administration. In 1 group of PMS-treated rats, a single injection of DXM was given. 2 mg/kg were used in 0.1 ml of a mixture of equal ratio of propylene glycol and saline. Another group of rats was treated with both human chorionic gonadotropin (10 IU HCG, Sigma) and a single injection of DXM. Corticotropic hormone (ACTH and 10 IU, Sigma) was injected in another group, i.p. along with DXM, 51 h after PMS injection. An additional control received only a s.c. injection of saline and i.p. injection of DXM vehicle.

Autopsies were performed on the 27th day of age in all animals. The occurrence of ovulation was determined by microscopic examination of the oviduct for ova with no attempt to count the ova. Ovaries were weighed prior to fixation in Bouin's Solution for histological examinations. Analysis of variance was conducted using F-test and Duncan's multiple range test.

Results. PMS injection in 24-day-old immature rats caused follicular development, ovulation and luteinization. A single injection of DXM given at 51 h after PMS-inhibited luteinization. In this group of animals, ovarian follicles, as examined microscopically, were larger than those of immature rats which did not receive PMS. These follicles contained ova at autopsy. Ovaries from PMS-treated rats which received no DXM were larger than those from animals which were injected with PMS and DXM but the difference was not significant ($p < 0.05$) (table). The presence of many corpora lutea in the ovaries of rats injected with PMS alone indicated that ovulation had occurred. The group of immature rats that received PMS, DXM and HCG showed a high ovulation rate with high ovarian weight (table). In the PMS-DXM-treated group, ACTH administration did not affect the ovulation rate nor ovarian weight when compared to the PMS-DXM group ($p < 0.05$).

Effect of dexamethasone on PMS-induced ovulation rate

Treatment	Body weight****	Ovarian weight (mg)	Uterine weight (mg)	Ovulation rate (%)
Control	52.2 ± 2.5[a]	34.8 ± 8.3[a]	27.7 ± 8.9[a]	0
PMS*	57.5 ± 3.5[a]	115.3 ± 12.3[bc]	96.8 ± 5.2[b]	82.5
PMS + DXM**	55.5 ± 4.2[a]	93.3 ± 13.5[c]	89.1 ± 5.5[b]	12.5
PMS + DXM + HCG***	56.5 ± 1.5[a]	124.9 ± 7.5[b]	98.6 ± 4.6[b]	82.5
PMS + DXM + ACTH***	48.7 ± 3.6[a]	109.1 ± 14.9[bc]	88.1 ± 19.4[b]	12.5

* PMS 25 IU was injected s.c. 72 h before sacrificing the animal.

** Dexamethasone 2 mg/kg was injected 51 h after PMS treatment.

*** HCG (10 IU) and ACTH (10 IU) were injected i.p. 51 h after PMS injection.

**** Data represent the mean ± S. E. of 8 animals in each group. Values having the same superscript are not significantly different ($p < 0.05$).

The uteri of rats in every group that received an injection of PMS, irrespective of DXM administration, increased in weight significantly ($p < 0.05$) as compared to vehicle-injected control rats (table).

Discussion. Ovaries from PMS-treated rats which were given dexamethasone, had matured follicles or follicles approaching maturity, but ovulation had been almost entirely prevented since ova could be found in these follicles and corpora lutea were rarely present. Ovaries from PMS-treated control rats contained many well developed corpora lutea.

The results indicate that luteinization should have occurred unless dexamethasone had inhibited either the hypophysis or centers in the central nervous system which influence the function of the hypophysis and thereby had blocked the release of endogenous LH. To further test these proposals, a group of rats was given PMS and DXM, in addition, HCG, which has LH-like activity, was administered. Dexamethasone did not suppress exogenous LH activity on the ovary in this group of rats.

DXM suppression of ovulation might be due to its central effect rather than its blocking effect on ACTH release, since ACTH administration did not overcome the inhibiting effect of DXM on ovulation. It is possible that DXM effect on ovulation was brought about by its direct effect on the brain and the hypothalamic biogenic amines. An inverse relationship has been indicated between circulating glucocorticoid levels and brain serotonin levels in the hypothalamus[4], striatum, cortex and amygdala[5].

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PRO EXPERIMENTIS

A turbidimetric method for the screening of amylase-producing mutants of Aspergillus niger

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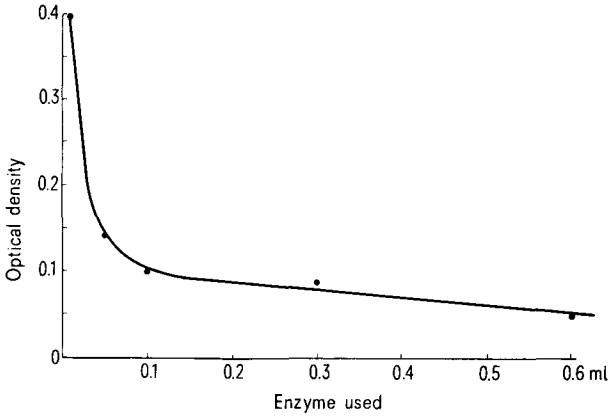
Summary. The method is based on the assumption that extracellular amylase, which is produced by strains of *Aspergillus niger* in liquid culture, hydrolyses the starch in the media and brings about a corresponding decrease in the turbidity of the media. Mutant strains which produced different quantities of amylase exhibited different degrees of decrease in turbidity of the media. The results showed that a greater degree of decrease in turbidity was observed for a higher quantity of amylase produced.

According to a recent FAO estimate[1], about 95 million tons of starch from cassava were produced in 1970. It has been reported that the fungus, *Aspergillus niger*, produces amylases which hydrolyse starch[2]. It is the particular object of the present research to obtain mutant strains of *Aspergillus niger* with high amylolytic activity.

In this communication, we report a convenient turbidimetric method for the screening of amylase producing mutants of *Aspergillus niger*. A turbidimetric method for the determination of α-amylase has already been described by Peralta and Reinhold[3]. The present method is based on the assumption that extracellular amylase produced by the organism in culture in a liquid suspension containing starch hydrolyses the starch and this brings about a decrease in the turbidity of the culture media. The degree of turbidity of the suspension is related to the quantity of extracellular amylases produced by the particular strain in culture.

Tapioca flour liquid media used to cultivate *A. niger* contained 2 g $NaNO_3$, 2 g tapioca flour and 100 ml water in 250 ml Erlenmeyer flasks. One loopful of conidiospores of each of 20 strains tested was inoculated into the media and incubated for 4 days at 34 °C. The mycelial mat which formed on the surface of the media was removed and the resultant growth media were subjected to turbidity measurement at 550 nm. The blank in this case was water and the control was tapioca flour liquid medium prior to inoculation with fungal spores. 4 replicates were used in each case.



Measurement of turbidity of tapioca flour media containing specific quantities of α-amylase. Optical density at 550 nm.

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