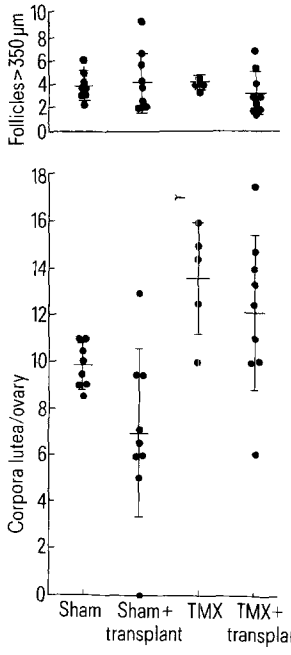

Number of corpora lutea per ovary and number of follicles greater than 350 µm diameter in 90-day-old Fischer 344 rats sham-thymectomized (sham) or thymectomized (TMX) and transplanted with a syngeneic testis at 5 days of age.

plant. Differences were seen however, when the numbers of corpora lutea were examined. Sham-operated rats receiving the transplant (sham+transplant) had significantly decreased numbers of corpora lutea when compared to sham-operated rats alone (sham), and thymectomized (TMX) rats had significantly increased numbers of corpora lutea when compared to sham-operated rats. However, the numbers of corpora lutea in TMX rats receiving the transplant (TMX+transplant) were not significantly decreased from TMX rats, and were increased when compared to sham rats. The difference between sham+transplant and TMX+transplant groups was also statistically significant.

A role for the thymus in reproductive function has been suggested not only by the experiments of Pierpaoli et al.[2], but also by Nishizuka and Sakakura[6] who reported the development of ovarian dysgenesis in certain strains of mice following thymectomy at 3-6 days of age, and by Lintern-Moore[7] who reported accelerated atresia of follicles and dysgenesis in the ovaries of neonatally thymectomized Wistar rats. Their data, along with ours, tends to support the concept of an involvement of the thymus in neuroprogramming. The detailed mechanism of this involvement is presently unknown. However, other published reports suggest a role for the thymus and/or thymocytes in steroid metabolism. These include the work of Kincl[8] who showed that injection of thymocytes prevented steroid-induced sterility in rats. Additionally, Weinstein et al.[10] have reported that the thymus and probably thymocytes metabolize progesterone, and there is other biochemical evidence of steroid biosynthesis in the thymus[10]. Our data showing a differential effect of testis transplantation on normal and thymectomized rats suggests the possibility that the thymus may influence neuroprogramming during the neonatal critical periods via its potential for production, metabolism, or conversion of steroid hormones. This hypothesis merits further detailed study.

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Effect of p-coumaric acid on immature estrogen treated and cyclic female mice

Anita Pakrashi[1], P.L. Pakrasi and S.N. Kabir

Reproductive Biology Section, Indian Institute of Experimental Medicine, 4, Raja Subodh Chandra Mullick Road, Jadavpur, Calcutta-700032 (India), 10 August 1978

Summary. Oral administration of p-coumaric acid to estrogen-primed immature female mice exerts neither estrogenic nor anti-estrogenic activity, but when it is administered to cyclic female mice in single dose at proestrus or in repeated doses, induces alteration in estrus cycle, ovarian and uterine weight and structure.

p-Coumaric acid [2 propionic acid, 3-(4 hydroxy phenyl)], a pure phenolic acid compound-active constituent of the alcoholic extract of the root of plant *Aristolochia indica* Linn has already been tested for antifertility and other biological properties in mice[2,3]. The present communication deals with the further follow-up studies of the compound on estrogen treated immature and mature cyclic female mice.

Materials and methods. Colony-bred Swiss albino mice-immature females, weighing 7-9 g and mature cyclic females weighing 22-24 g, were taken for study. Animals were maintained in controlled temperature (24-25 °C) and light regimen of 14 h light and darkness of 10 h.

Study 1. Immature animals were treated with estradiol dibenzoate (Schering), s.c. for 3 days. Grouping of animals, doses of the compound and hormone are summarized in table 1. 24 h after the last injection the vaginal opening and vaginal cornification were recorded, and then laparotomy was performed under light ether anesthesia. Uteri were dissected out and weighed in a semi-micro balance after pressing out the fluid in a blotting paper.

Study 2. Cyclic mature females were allocated to 2 groups. One group was treated with the drug, orally, in a single dose – the dose responsible for cent percent interceptive activity[2] – at their proestrus stages and another group was treated with repeated lower dose (table 2) for 15 consecutive days. Vaginal smears were recorded daily until the last day of drug administration; 24 h following the last dose, the animals were sacrificed and both ovaries and uteri were weighed. Tissues were fixed in Bouin's fluid and prepared for histological examination. Control animals were maintained in parallel with each of the 2 studies.

Table 1. Effect of p-coumaric acid on estrogen induced uterotropic action in immature mice

No. of mice used	p-Coumaric acid	Estrogen	Uterine weight (mg)*
5	Vehicle only	Oil only	6.98±0.89
10	–	0.5 µg/day for 3 days	41.44±4.46
10	50 mg/kg/day for 3 days	0.5 µg/day for 3 days	40.08±2.19
5	50 mg/kg/day for 3 days	–	6.44±0.38

* Values are mean ± SE.

Table 2. Effect of p-coumaric acid on cyclic mice

No. of mice used	p-Coumaric acid	Ovarian weight (mg)*	Uterine weight (mg)*
10	50 mg/kg/day for 1 day at proestrus	5.6 ±0.39**	85.03±0.21***
10	40 mg/kg/day for 15 consecutive days	6.38±0.65**	90.02±0.32***
5	Vehicle only (control)	5.5 ±0.49	61.71±8.34

* Values are mean ± SE, ** $p > 0.5$, *** $p > 0.01$.

Results. In the case of study 1, the day of vaginal opening and cornification remained the same in both the experimental and control groups. The results, as shown in table 1, indicated that the compound had no effect on exogenous estrogen. But when the drug was treated to cyclic females, both in a single and repeated doses, it prolonged the estrus period. In the case of single dose, the estrus phase was continued for 2–3 days; but in the case of successive days drug-treated females, the cycle remained normal up to day 9, and then it became gradually estrogenic with slow increase in the number of cornified cells in the vaginal smear. On 15th day vaginal smear of all the animals showed only cornified cells. Both the ovarian and uterine weights were increased though the change in ovarian weight was not significant (table 2). Histological picture of the ovary and uterus in the experimental group, showed excess number of follicular growth and highly proliferated endometrium with stratified epithelium and glandular development respectively.

Discussion. Our results indicate that the compound does not exert any direct uterotropic effect. It neither inhibits nor facilitates the action of exogenous estrogen. Prolongation of estrus, change in normal cyclic pattern by slow increase in the number of cornified cells, and increase in follicular growth reflects FSH predominancy which may be due to antiprolactin nature of the compound. In our previous work[2], we observed that PCA administered in early post-implantation stage of mouse and hamster terminates pregnancy and converts leukocytic vaginal smear to cornified one. These actions can be prevented by exogenous supplementation of only prolactin, but not LH (unpublished). These findings, along with our previous reports on crude extract[4] of the plant and the pure compound (PCA)[5], reflected possible antiprolactin nature of the compound. Dickerman et al.[6] reported that in rat haloperidol directly stimulate prolactin, and as a result there occurs inhibition of FSH. Seki et al.[7] reported that CB 154 – a known antiprolactin – suppressed prolactin secretion and thereby caused stimulation of FSH and LH secretion in women with amenorrhea – galactorrhea syndrome. These observations lead us to speculate that there exists an inverse relationship between FSH and prolactin and tempts us to suggest that in our study PCA – due to its antiprolactin nature – somehow interferes with prolactin secretion/function with resultant increase in FSH level which ultimately gives rise to the aforementioned manifestations.

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Plasma steroid and protein hormone concentrations in patients with benign prostatic hypertrophy and in normal men

E. Ortega, E. Ruiz, M.C. Mendoza, A. Martin-Andres and C. Osorio

Departamento de Fisiologia y Bioquimica Facultad de Medicina de Granada, Granada (España), 25 September 1978

Summary. Serum levels of testosterone, FSH, LH and prolactin were measured by RIA in patients with BPH (benign prostatic hypertrophy) and in control men. The testosterone concentrations in the BPH group were significantly lower than that of the control group. Serum concentrations of FSH, LH, prolactin and estradiol, did not differ significantly in the 2 groups.

In most men over 60 years of age benign prostatic hypertrophy (BPH) is a common occurrence. At the same time, several hormonal changes take place. It is known that testosterone metabolism in BPH patients is modified, since the ratio between testosterone and dihydrotestosterone in plasma is lower than in younger adults[1].

Serum levels of FSH and LH are elevated after 60 years of age[2]. Pituitary hormones may influence prostatic growth