prolonged exposure of AFP-estrogen complexes to charcoal, we believe that the techniques reported here (Millipore system) allowed a more accurate determination of k_{-1} for AFP.

Calculations of the k_{+1} and k_{-1} values for the AFP-estrogen complex allowed independent determinations of the equilibrium association constants. Ratios of the rate constants yielded K_a values for estradiol and estrone that were statistically identical to the K_a values obtained for each estrogen from Scatchard analysis. Therefore, based on the results of Scatchard analysis, k_{+1} and k_{-1} determinations, and independent K_a calculations, it can be concluded that AFP has binding affinities and capacities that are identical for estradiol and estrone.

1 The abbreviations used are: AFP, α-fetoprotein; FF, fetal fluid; K_a, equilibrium association constant; B_{max}, number of binding sites: k_{+1}, association rate constant; k_{-1}, dissociation rate constant.

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Decidual cell reaction in ovariectomized-adrenalectomized rats

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Summary. Enhancement of decidual cell reaction (DCR) following adrenalectomy was reversed by corticosterone as well as indomethacin. The results suggest the adrenal involvement in DCR through uterine prostaglandin production.

Decidual cell reaction (DCR) can be induced by endometrial traumatization or intrauterine oil instillation in pseudopregnant as well as suitably hormone-primed rodents[3]. Previous studies have suggested the involvement of prostaglandins (PGs) in the induction of DCR[4-7]. Glucocorticoids, known to inhibit synthesis and/or release of PGs in a cell culture system[8,9], prevent estradiol-induced implantation in hypophysectomized pregnant rats[10]. However, possible in vivo effects of corticosteroids on decidualization have not yet been adequately studied. The present experiments reveal the involvement of the adrenals in DCR following instillation of oil into the uterine lumen in adequately sensitized ovariectomized adult rats.

Five groups of female rats of the T strain used in the present study were maintained in a temperature- and light-controlled room (lights on from 05.00 to 19.00 h). The rats were ovariectomized on the day of the first vaginal estrus occurring after day 60 of age. 4 of the 5 groups were adrenalectomized at the time of ovariectomy (AX-OX rats), while the adrenals were left intact in the remaining one (OX rats). All the rats were given s.c. injections of 3 mg progesterone (P) in 0.1 ml sesame oil for 7 consecutive days commencing on the day after the operation. A single s.c. injection of 0.1 μg estradiol-17β (E_2) in 0.05 ml oil was given between 18.00 and 19.00 h on the 3rd day of the P injection period. 16 h after the E_2 injection, 0.1 ml sesame oil was instilled into the right uterine horn from its tubal end as a deciduogenic stimulus. 2 of the 4 AX-OX groups were given s.c. injections of 500 μg corticosterone or 50 μg aldosterone, each dissolved in 0.1 ml oil, twice daily (08.00 and 16.00 h) during the P injection period. The remaining 2 groups receiving no corticoid were provided with 0.9% NaCl as drinking water, instead of tap water. 1 of these 2 groups were given s.c. injections of 1 mg indomethacin in 0.2 ml oil, one 2 h before and the other 6 h after oil instillation. On the day following the last P injection, the animals were sacrificed. The weight of the stimulated horn bearing deciduomata was used to estimate the size of DCR. Data were analyzed by Student's t-test and Fisher's exact probability test.

As shown in the table, both OX and AX-OX rats invariably formed deciduomata. In the AX-OX rats, however, the treated horn bore massive deciduomata along its entire length, 2–3 times heavier than in the OX rats with their adrenals intact ($p < 0.001$). 9 of 10 AX-OX rats formed deciduomata not only in the treated horns but also in the contralateral untreated horns. Thus, the absence of adrenals resulted in a marked increase in uterine responsiveness to the oil instillation stimulus. Under the conditions of the present study, administration of 500 μg corticosterone twice daily during the P injection period significantly reduced the size of DCR in AX-OX rats ($p < 0.001$). There was no difference in mean weight of the treated horns between OX rats with their adrenals and AX-OX rats given corticosterone ($0.2 > p > 0.1$). However, the reduction in number of rats with positive DCR in the untreated horns in latter group was not significant ($p = 0.151$). By contrast, all AX-OX rats given a similar treatment with 50 μg aldosterone produced deciduomata in both treated and untreated horns, incidence and size of DCR being approximately the same as in AX-OX rats receiving no corticoid (size of response: treated horn, $0.6 > p > 0.4$, untreated horn, $0.2 > p > 0.1$; incidence in untreated horns: $p = 0.588$). These results suggest that the increase in DCR following adrenalec-

tomy is attributable to the shortage of circulating glucocorticoids rather than mineralocorticoids.

Evidence has accumulated suggesting the importance of PG synthesis in initiation of DCR. Deciduogenic stimulus increases the uterine content of PGs[6,7]. Administration of indomethacin, an inhibitor of PG synthesis[11], interferes with implantation and DCR[4,5]. In the present study, if 2 injections of 1 mg indomethacin were given, one 2 h before and the other 6 h after intraluminal oil instillation, DCR was almost similar in magnitude to that in OX rats with their adrenals intact ($0.2 > p > 0.1$). None of the rats of this group formed deciduomata in the untreated horns. Therefore, it is not inconceivable that the increase in DCR in AX-OX rats as compared to OX rats was due to an increased uterine PG production in the former. Consistent with this view is the finding that glucocorticoids suppress the synthesis and/or release of PGs in a cell culture system, mineralocorticoids being less potent in this respect[8,9]. Anyhow, the present findings suggest the involvement of adrenal secretions in the regulation of DCR. Occasional DCR occurring in the untreated uterine horns contralateral to the treated ones has already been noted[12–14]. In the case of the present experiments, transfer of instilled oil, possibly together with PGs, from the treated horns to the contralateral ones via the uterine cervix or diffusion of secondary effectors, possibly PGs, released from treated horns might be involved in the induction of DCR in the untreated horns. Further studies are needed in order to draw a final conclusion.

Deciduoma formation in rats ovariectomized and ovariectomized-adrenalectomized as adults

Group	Positive response		Mean weight (mg ± SE) of	
	Treated horns	Untreated horns	Treated horns	Untreated horns
OX	9/ 9	0/ 9	305.8 ± 34.6	72.2 ± 1.7
AX-OX	10/10	9/10**	749.7 ± 40.4**	71 (460.9 ± 68.1)[a]
AX-OX + 500 µg CT[b]	10/10	6/10*	436.8 ± 64.4(**)	82.8 ± 8.2 (385.8 ± 110.4)
AX-OX + 50 µg AD[b]	7/ 7	7/ 7**	792.0 ± 36.6**	(626.1 ± 65.3)
AX-OX + 1 mg IN[c]	7/ 7	0/ 7**	229.4 ± 30.4(**)	65.1 ± 5.1

AD, aldosterone; AX, adrenalectomy; CT, corticosterone; IN, indomethacin; OX, ovariectomy. [a] Mean weight of untreated horns bearing deciduomata is given in parenthesis. [b] Given twice daily for 7 days after operation. [c] Given 2 h before and 6 h after deciduogenic stimulus. * $p < 0.01$ and ** $p < 0.001$, significance of difference in DCR incidence (Fisher's exact probability test) and in mean weight of horns (Student's t-test) from OX group, (**) $p < 0.001$, from AX-OX group.

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Facilitation or inhibition of memory by morphine: a question of experimental parameters[1]

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Summary. The effects of morphine on memory are highly controversial. According to some investigators post-trial injections of morphine facilitate memory. Others, however, have reported impairment of memory after morphine injections. To investigate the extent to which this may be due to different experimental parameters, foot-shock intensity and dosage of morphine were systematically varied in a passive-avoidance task. It was found that post-trial administration of medium and relatively high doses of morphine facilitate retention performance following moderate levels of foot-shock. Under other conditions of dose and shock intensity, the drug was not effective or even impaired retention.

The discovery of the endogenous opiates[2,3] has rekindled interest in the effects of morphine on the central nervous system. Many studies have been made of the influence of morphine on learning and memory, but so far the results obtained have been divergent.

Some authors have shown that post-trial treatment with opiate agonists improves retention. Belluzzi and Stein[4], Mondadori and Waser[5], and Stäubli and Huston[6] have all reported that morphine, when given in relatively high doses, improves retention of a one-trial passive-avoidance situation. It also has been demonstrated that an injection of morphine given after a one-trial appetitive task facilitated its memorization.

Other investigators have found that retention is impaired by morphine. Retention of a step-through passive-avoidance task[7,8] or of a shuttle-box avoidance task[9] has been shown to be diminished by low post-trial doses of morphine. Similarly, post-trial administration of Leu-enkephalin or beta-endorphin resulted in poorer retention of a shuttle-avoidance task and inhibited the habituation of rearing in response to an acoustic stimulus[10–12].

Contradictory results are not uncommon in the pharmacology of memory[13]. It has been shown that the effects of drugs on the memory often depend on the nature of the task set[14–16] and the dose used[17,18]. Amongst others, Gold and van Buskirk[19] and