

spent in each stimulus chamber ($F = 5.20$; $df = 5, 36$; $p < .005$) and number of entries into each chamber ($F = 8.13$; $df = 5, 36$; $p < .001$).

Table 1 shows that (i) the animals in group 1 spent 600 percent more time with the human than with the monkey; (ii) monkeys in group 6 (who also had early handling, with no early peer contact) spent the second greatest amount of time with the human, although they spent more overall time with the monkey; and (iii) all other groups spent at least six times more time with the monkey than with the human. The same general result was obtained for the number of entries. The consistency of these results is shown by the proportion of subjects in each group making the monkey compartment their first choice, and the proportion spending more time with the monkey than with man.

A further point is the amount of time spent in the "neutral" central chamber. Both the 1-year and the 6-month early isolate animals spent almost two-thirds of the time in the center, while all other groups spent most of their time with either human or monkey. However, when these two isolate groups did choose a stimulus, they picked the monkey. In interpreting this result it was assumed that introducing a monkey into a completely novel environment without prior adaptation produces at least mild fear; choice of either monkey or human is therefore thought to be a response to the most fear-reducing stimulus available. Time spent in the center chamber would thus indicate the relative degree to which both choice stimuli failed to reduce fear.

These data lead to the conclusion, not well established in the primate literature, that the very early experience of a monkey with a specific type of stimulus can have lasting effects upon later preferential choice concerning that stimulus. Thus, a phenomenon perhaps related to imprinting may be operative in primate behavior. However, unlike the theoretically irreversible effects of imprinting in birds, learning in the first month by monkeys is apparently overcome by experience with other stimuli after the 1st month; this is shown by the strong preference by animals in group 2 for a monkey. Thus, effects on the monkey of early learning may be reversible, or the duration of an imprinting-like process leading to the formation of long-lasting "social" stimulus preferences may be much longer in monkeys than in birds.

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Feminine Behavior in Neonatally Castrated and Estrogen-Treated Male Rats

Abstract. *Male rats castrated within 4 days after birth are behaviorally feminized. On the other hand, intact or castrated males given estrogen neonatally show little estrous behavior in adulthood. Thus, feminization is induced by lack of neonatal androgen rather than by the presence of estrogen. Estrogen administered to newborn rats suppresses feminization.*

Male rats injected with estrogen on the 4th day of postnatal life show reduced male sexual behavior as adults (1); such estrogen-treated males are also said to display some female behavior in adulthood. It was suggested that these animals might have been feminized, perhaps by a chemical cas-

tration attributable to the injected estrogen. The work described here was done, in part, to determine whether estrogen actually does induce behavioral feminization when injected into newborn male rats.

The presence of androgen during the period of differentiation of the neural

tissues mediating sexual behavior prevents the full display of female sexual behavior in adulthood. This period of neural differentiation occurs prenatally in guinea pigs (2) and during the 1st week of postnatal life in rats (3). Females treated with androgen during these periods fail to show normal feminine behavior as adults (2, 3). On the other hand, male rats castrated no later than 1 week after birth, and thereby deprived of testicular androgen during neural differentiation, are capable of displaying female behavior in adulthood (4). The question was asked whether rats castrated during the 1st week would be further feminized by injecting estrogen during this time. Thus, our second aim was to assess the relative roles of absence of androgen and presence of estrogen in the feminization of sexual behavior in the rat (5).

In experiment A the following four groups were used: group 1A, rats castrated 16 to 32 hours after birth; group 2A, rats castrated 16 to 30 hours after birth and given 100 μ g of estradiol dipropionate subcutaneously on day 5; group 3A, rats sham-operated 16 to 30 hours after birth and given 100 μ g of estradiol dipropionate on day 5; and group 4A, rats sham-operated 16 to 30 hours after birth. Animals in groups 3A and 4A were castrated when 60 days old. Tests were begun when the animals were 80 to 95 days of age. To induce estrous behavior each rat was injected with 4 μ g of estradiol dipropionate and 40 hours later with 0.5 mg of progesterone. One hour after the progesterone injection, each animal was "fingered" for the lordosis response by lightly stroking the back between the iliac crests. Each subject was then placed with three stimulus males for 3 minutes or until it had been mounted 10 times. This process was repeated every 2 hours until 13 hours had elapsed from the time of progesterone injection. This entire procedure constituted one test. Animals underwent three such tests at 2-week intervals. Frequency of lordosis, ear-wiggling, and crouching (all indications of receptivity) were recorded. Frequency of mounting by stimulus males was also used to measure the stimulation value of the subjects.

Four groups of rats were also used in experiment B: group 1B, rats castrated 96 hours after birth; group 2B, rats castrated 96 hours after birth and then given 200 μ g of estradiol benzoate subcutaneously; group 3B, rats given

Table 1. Female behavior of male rats castrated (1A), castrated and given estradiol dipropionate (2A), given estradiol dipropionate (3A), or not treated (4A) when newborn. Males in groups 1A and 2A were castrated 16 to 32 hours after birth.

Group	N	Mean latency to lordosis induced by males (hr)	Tests positive for crouching, ear-wiggling (%)	Lordoses Mounts $\times 100$ (%)
1A	8	6.1*	70.8*	40.0*
2A	9	11.2	0	6.4
3A	9	9.9	0	14.0
4A	8	9.6	8.3	6.6

* Significantly different from all other groups by Mann-Whitney U-test ($p < .05$). Groups 2A, 3A, and 4A did not differ significantly from one another.

200 μ g of estradiol benzoate 96 hours after birth; and group 4B, rats given 0.2 ml of peanut oil 96 hours after birth. At 180 days males in groups 3B and 4B were castrated. Starting at 200 days, three weekly tests were given. To induce estrous behavior, 10 μ g of estradiol benzoate was given on each of two successive days, followed by 0.5 mg of progesterone the next day. Each subject was paired for 10 minutes with a stimulus male 3½ hours after the progesterone was injected. The frequency of mounting by a stimulus male and frequency of lordosis were recorded.

Results from experiments A and B show that the intact male rats treated with estrogen during the 1st week of life (groups 3A and 3B) did not display more female behavior than corresponding males of groups 4A and 4B (Tables 1 and 2). These findings indicate that estrogen injected into newborn intact males does not induce feminization.

On the other hand, the males in group 1A (castrated 16 to 32 hours after birth) and the males in group 1B (castrated 96 hours after birth) displayed significantly more feminine behavior than all the other groups in their respective experiments (Tables 1 and 2). Thus, it is confirmed that absence

of androgen during neural differentiation induces feminization. It is evident that the injection of estrogen to the neonatally castrated males (groups 2A and 2B) not only failed to induce further feminization, but actually suppressed female behavior (Tables 1 and 2).

Table 1 indicates that the males in group 1A displayed lordosis in response to mounting at a mean time of 6.1 hours after the injection of progesterone. The lordosis response was also elicited by fingering these males. The latency was 7.5 hours from the time of progesterone injection. Animals in groups 2A, 3A, and 4A never responded to fingering. Although not quantified, castrated males of group 1A displayed a deeper and more prolonged arching of the back when they responded to mounting or fingering. All other groups in experiment A exhibited weaker and shorter lordoses.

Animals in group 1A were mounted twice as frequently (33.4 times per test) as the next highest group 4A (16.9 times per test). This difference was significant [$p < .002$, Mann-Whitney U-test (6)], and indicates that stimulus males preferred the animals in group 1A to all other animals as sex partners. These results were not duplicated in experiment B. In experiment B, there were no significant differences between groups in the number of times the animals were mounted by stimulus males (Table 2). This discrepancy probably reflects differences in testing procedures in the two experiments.

We predicted that the males in group 1A (castrated 16 to 32 hours after birth) would show more female behavior than the males in group 1B (castrated 96 hours after birth), because of the already demonstrated rapidity with which neural differentiation occurs after birth (7). This prediction appears confirmed (Tables 1 and 2) but the differences in testing procedures between experiments A and B should be considered.

Group 1A males showed significantly more female sexual behavior (in all measures) than animals of groups 2A, 3A, and 4A. These last three groups did not differ significantly from one another in any of the measures shown in Table 1. In experiment B, males in group 1B exhibited the lordosis response on significantly more occasions than animals of the other three groups (Table 2).

Thus, estrogen injected into newborn male rats does not induce female behavior. In fact, such treatment suppresses its display. Inasmuch as early treatment with estrogen prevents receptivity in adult female rats, these results are not surprising (8). We conclude that it is the absence of androgen during the period of neural differentiation rather than the presence of estrogen which induces female sexual behavior.

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5. While this experiment was in progress, H. H. Feder learned that R. E. Whalen was attempting to answer this same question. A comparison of notes revealed that although procedural details differed, results were generally confirmatory. We therefore decided to present our work jointly. Experiment A (H.H.F.) was supported by grant NIH 08634 from the USPHS. Experiment B (R.E.W.) was supported by research grant HD-00893-02 from the National Institute of Child Health and Human Development of the USPHS.
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9. Estradiol dipropionate (Ovoclyn) was supplied through the courtesy of Ciba Inc. Estradiol benzoate (Progyon) was generously supplied by Schering Corp., Bloomfield, New Jersey. We thank William C. Young for his many valuable criticisms of the manuscript.

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Albinism and Water Escape Performance in Mice

The interpretation given by Winston and Lindzey (1) of the interesting behavioral differences they found between certain albino and pigmented strains of mice does not do justice to differences reported elsewhere. We have shown, for example (2), that at least one albino strain (CF/1) demonstrates routinely short escape times from a water maze and that its overall performance is very much like that of two of the pigmented strains used

Table 2. Frequency of lordosis in male rats castrated (1B), castrated and given estradiol benzoate (2B), given estradiol benzoate (3B), or injected with oil (4B) when newborn. Rats in groups 1B and 2B were castrated 96 hours after birth.

Group	N	Total times mounted	Lordoses Mounts $\times 100$ (%)
1B	6	174	19.5*
2B	5	145	0.0
3B	9	210	0.0
4B	8	168	6.5

* Significantly different from all other groups by the F-test ($p < .01$). Groups 2B, 3B, and 4B did not differ significantly in the number of lordoses shown.