at approximately 0.8-km intervals along a semicircle formed by the Arima-Blanchisseuse Road at the head of the Arima Valley (Fig. 2). The point of release was on a hill south of the semicircle and distances to the traps varied from 0.4 to 1.2 km. Recaptures were made at all six traps. The results of this experiment were similar to the first: mimics and control moths, contrary to prediction, were recaptured to nearly the same extent (Table 1).

The failure of these two attempts to demonstrate mimetic advantage is possibly explained by the fact that the artificial mimic and control moths are both relatively inconspicuous and consequently attract such a small amount of predation that differential recapture could not be detected within the magnitude of the experiments.

Therefore, a third experiment was carried out in the same area as experiment 2, with the prediction that a highly conspicuous color pattern that is nonmimetic would be less frequently recaptured than its control. Moths were painted a unique pattern with extremely bright and contrasting colors, but were not painted to resemble any species of Trinidad lepidopteron (Fig. 1*C*). The frequency of recapture of the painted controls exceeded that of the uniquely painted experimentals by a factor of 2 to 1. This difference is significant at the .05 level (Table 1). The experiment has therefore established that the color pattern of a diurnal insect can influence its survival in the natural environment.

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Estrogen Administered Neonatally Affects Adult Sexual Behavior in Male and Female Rats

Abstract. *The injection of 100 μg of estradiol benzoate into female rats 96 hours after birth abolished sexual receptivity in adulthood, even with estrogen and progesterone replacement after ovariectomy. The administration of testosterone propionate to these animals in adulthood elicited the full pattern of male sexual behavior. The same dose of estrogen given to male rats 96 hours after birth produced adults which were unable to achieve intromission, although they mounted as frequently as normal animals. Testosterone replacement after castration in adulthood reproduced this abnormal behavior.*

There is evidence (*1*) that secretions of the fetal and neonatal gonads influence sexual differentiation. It has been reported that neonatal female rats receiving a single injection of testosterone propionate in doses of 50 μg or more exhibit constant vaginal cornification and possess atrophic ovaries containing ripe follicles and no corpora lutea. It has also been found that neonatal males given a single injection of estradiol benzoate in doses of 50 μg and above show marked atrophy of the testes.

Although the major interest in the effects of neonatal sex steroids has been on the subsequent reproductive capacity of the adult organism, there has recently been considerable attention (*2*) directed toward the effects of these neonatal hormone treatments on patterns of adult sexual behavior. Female rats treated neonatally with testosterone propionate in doses of 50 μg or more are sexually unreceptive in adulthood, even when castrated and given estrogen and progesterone replacement. Administration of testosterone propionate to these adult anovulatory females does, however, elicit male sexual behavior patterns in the presence of a receptive female. Recently Whalen and Nadler (*3*) reported a study in which neonatal female rats treated with 200 μg of estradiol benzoate are also sexually unreceptive when castrated and given estrogen and progesterone replacement. These data are in contrast to the findings of Harris and Levine (*4*) that neonatal female rats treated with 50 μg of estradiol benzoate showed sexual receptivity when given estrogen and progesterone replacement and exhibited persistent vaginal cornification with the same ovarian histology as the testosterone-treated females mentioned above. The one obvious difference between these studies is the dose of estradiol benzoate administered to the neonate. It has been shown recently that the dose of the hormone given to the neonatal female is critical with regard to sexual behavior. Barraclough and Gorski (*5*) have reported that doses of testosterone propionate in the range of 10 μg do not abolish sexual receptivity but, in fact, produce an animal that is constantly sexually receptive.

In order to attempt to reconcile the differences between the data reported by Whalen and Nadler and those found by Harris and Levine, neonatal female rats ($N = 13$) derived from a parent Long-Evans strain were injected with 100 μg of estradiol benzoate in 0.1 ml of oil at 4 days of age. Control animals ($N = 11$) were injected with 0.1 ml of the oil only (sesame oil). All animals were weaned at 21 days of age and weighed once weekly until 105 days of age. When the rats were 65 days old, vaginal smears were taken on all animals for 18 days. At 105 days, the females were tested with sexually vigorous adult males. Approximately 2 weeks after this, all the females were ovariectomized, and at 170 days they were tested with proven males, with no replacement therapy. At 200 days, the animals were given estrogen and progesterone replacement and again tested with proven males, and finally, at 230 days of age, the females were given testosterone replacement and tested for male sexual behavior with a receptive female. With the exception of the final testing for male sexual behavior, all of the test sessions were for a single 15-minute period. The regime used for estrogen and progesterone replacement consisted of injections of 200 μg of estradiol benzoate 72 and 24 hours prior to the test, and an injection of

Table 1. Effect of neonatally injected estrogen on sexual behavior of female rats that were ovariectomized at about 119 days of age and given estrogen and progesterone replacement and tested with male rats at 200 days of age.

Neonatal injection	*N*	Mean frequency of mounts by male	Mean frequency of lordosis	Lordosis/ mount ratio
100 μg estrogen	13	38.38	1.23	0.03
Oil (controls)	11	22.73	20.45	0.90

1 mg of progesterone 6 hours before the test. This regime successfully produces sexual receptivity in 100 percent of normal spayed females. To test for male sexual behavior, 100 μg of testosterone propionate was injected daily for 9 days. On each of the last 7 days of injections, the animals were tested for 15 minutes with a receptive female. All testing took place in a cylindrical glass observation cage. During the tests for sexual receptivity, frequencies of mounts made by the male and lordotic responses made by the female were recorded, as well as the presence or absence of back-kicking. During the tests for male sexual behavior, the frequencies of mounts, mounts with intromissions, and ejaculation patterns were recorded.

The results obtained in this study with regard to the sexual receptivity of the neonatally estrogen-treated females are consistent with those reported by Whalen and Nadler, with a few exceptions. The females in this study, given 100 μg of estradiol benzoate as neonates, were unreceptive in adulthood, both when intact and when castrated and given estrogen and progesterone replacement (Table 1). One difference is that the females reported by Whalen and Nadler showed 100 percent kicking responses, while 75 percent of the females in our study were extremely passive during mounting, either crouching or wandering and sniffing around the cage.

Although no sexual receptivity was found with these neonatally estrogenized females, some of them did show male sexual behavior when castrated and when given testosterone. The averages given in Table 2 are based upon only those animals which made two or more mounts per 15-minute test session. Although the number of females exhibiting complete male sexual behavior (including ejaculation patterns) was small, it is striking that this was shown at all. Not only were all of the components of the ejaculation pattern exhibited (long mount, final thrust, and slow raising of the front paws), but also the post-ejaculation latency before the next mount. (A normal male shows little activity immediately after ejaculation, and then gradually regains interest in the female. After the first ejaculation, this period lasts about 5 minutes.) In one female, a second ejaculation pattern was observed in one session, and this came after fewer intromission patterns than did the first one, just as occurs with normal males.

There also were a number of interesting physiological observations on these estrogenized females. First, full vaginal opening occurred at 18 days of age, in comparison to the normal time of 35 days for this strain. Since the estrogen was administered at 4 days of age, it is difficult to attribute this effect to a direct action of estrogen on the tissues involved. As mentioned above, these animals showed atrophic ovaries with ripe follicles but no corpora lutea. (Mean ovarian weight for estrogenized females was 39.28 mg and for normal females it was 48.27 mg, with $p < .001$, 22 *df*.) Also, after 9 days of testosterone replacement in adulthood, these females showed extreme masculinization of the genitalia which persisted for at least 30 days after the end of testosterone administration. This is in contrast to the less extreme effects seen in the control animals. The masculinization of the genitalia of the controls was not evident unless the prepuce was pulled back, but the genitalia of some of the estrogenized females protruded constantly.

Also in this study, neonatal males were injected at 4 days of age with 100 μg of estradiol benzoate ($N = 13$) and a control group was injected with the oil vehicle ($N = 12$). The animals were weaned and weighed according to the schedule described above for the females. The males were tested intact at 115 days of age, and castrated at 130 days. At 175 days of age they were tested with no replacement, and then were put on the same testosterone replacement schedule as that used with the females (9 days of 100 μg of testosterone propionate daily, with 15-minute tests with a receptive female being conducted on each of the last 7 days).

The adult sexual behavior of these neonatally estrogenized males revealed some unusual components. As can be seen from Table 3, the estrogenized males did not make a significantly higher number of mounts per 15-minute session than did the control animals, when tested before castration, but they did make a significantly lower number of intromissions. Only one of the estrogenized males ejaculated. The nature of the mounts made by these males was indeed bizarre, for they were generally from the head, the side, or high up on the back of the receptive female. When castrated, these males again showed fewer intromissions than the controls, and with testosterone replacement, the original pattern of behavior reappeared, the intromission/mount ratio being almost the same as in

Table 2. Average number of mounts, intromission patterns (I.P.), and ejaculation patterns (E.P.) shown by female rats (i) after ovariectomy and (ii) after ovariectomy and testosterone treatment. Of the 13 experimental and 11 control animals that were used in this portion of the study, only those that made two or more mounts in any session are included in this table. The figures in this table have been rounded to the nearest whole number.

Neonatal treatment	*N*	Mounts	I.P.	E.P.
Ovariectomized				
100 μg estrogen	3	16	2	0
Oil injected	0	0	0	0
Average of 7 days with testosterone				
100 μg estrogen	6	22	3	2
Oil injected	4	14	2	0

Table 3. Average number of mounts and intromissions by male rats (i) when intact, (ii) after castration, and (iii) after castration and testosterone treatment. Of the 13 experimental and 12 control animals that were used in this portion of the study, only those that made two or more mounts in any session are included in this table. The figures in this table have been rounded to the nearest whole number. Int., intromissions; Ejac., number ejaculating.

Neonatal treatment	*N*	Mounts	Int.	Ejac.
Intact				
100 μg estrogen	10	23	3	1
Oil injected	11	17	12	9
Castrated				
100 μg estrogen	6	23	2	0
Oil injected	8	22	6	2
Average of 7 days with testosterone				
100 μg estrogen	11	34	4	1
Oil injected	9	25	15	6

the intact animals. In contrast, the oil-treated controls showed very high intromission/mount ratios both when intact and with testosterone replacement after castration. The possibility cannot be excluded that the failure of the estrogenized males to achieve normal numbers of intromissions is due to lack of development of the accessory sexual apparatus, particularly the os penis, as has been found by Beach in neonatally castrated males (*6*). If this is so, it will be necessary to identify the mechanism through which the neonatal estrogen injection can cause this block in development. The testes of the estrogenized males ($\bar{x}$=1.95 g) were significantly smaller ($p < .001$, 25 *df*) than those of the controls ($\bar{x}$=3.47 g). Previous research (*2*) has shown that the accessory sexual organs, such as the seminal vesicles, are also small and undeveloped in males given this dose of estrogen neonatally.

These data are difficult to reconcile at this time with the hypothesis previously proposed by Harris and Levine concerning the effect of neonatal hormones on the sexual differentiation of the central nervous system. The evidence thus far suggests that the newborn rat of either sex possesses a common, undifferentiated regulatory mechanism for the cyclic release of gonadotrophin from the anterior pituitary. During development in the neonatal female rat, this mechanism becomes fixed in its original cyclic pattern. During the critical period of the first few days of life in the neonatal male (*7*), this mechanism appears to be organized and changed to one of acyclic gonadotrophin release by the secretions of the infantile testes. If the newborn male is castrated or the normal secretion of the testes blocked in some other way, the original cyclic mechanism is retained throughout adult life. Conversely, if the neonatal female receives androgens during the critical period (*8*), the original cyclic mechanism is differentiated therefore into an acyclic pattern. Thus, the nature of the hypothesis implicates testosterone as the active hormone organizing the sexual control system, both in terms of reproductive cycles and sexual behavior.

Therefore, the effects of estrogen on the male may be accounted for by the direct action of large doses of estrogen on the neonatal testes (*9*), producing a male that is functionally partially castrated. However, the estrogenized males in this study actually showed a great deal of sexual vigor, although their orientation to the female in mounting was strikingly different from normal.

The more difficult finding to integrate with the hypothesis, however, is the action of estrogen in the neonatal female. If the main organizing hormone is that secreted by the neonatal testes, why does neonatal administration of estrogen produce effects that are in many ways similar to those produced by neonatal administration of testosterone? In the male, the effects of neonatal hormones seem to show some degree of specificity. Testosterone administered to the infant male rat in large doses produces little or no effect on sexual behavior and reproductive capacity (*2*). In contrast, the neonatal female rat shows a greater degree of nonspecificity to the action of the sex steroids. Whether other steroids will produce similar effects has not been determined as yet. Although these effects initially appear to be somewhat paradoxical, the influence of gonadal hormones administered in infancy upon adult patterns of both sexual behavior and gonadotrophin secretion appears now to be a well-established phenomenon.

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Synaptic "Learning" Due to Electroosmosis: A Theory

Abstract. *Since the interstitial water of the central nervous system is in narrow channels between cells that have surface charge, electroosmotic flow of water should accompany current flow. Current density is high close to an excitatory postsynaptic membrane and the electroosmotic effect must be significant. Simultaneity relationships between action potentials in pre- and postsynaptic cells affect postsynaptic membrane current density and modulate the effect in a way which should produce "learning."*

When electric current flows in a liquid which is surrounded by a substance with a surface charge, the liquid will move. This is the phenomenon of electroosmosis. It can be shown that electroosmotic water flow is probably a powerful factor by which electrical activity can cause morphological change in the central nervous system. Furthermore, the effect is sensitive to whether or not pre- and postsynaptic cells fire simultaneously, and therefore will exert an integrative influence on synaptic relations.

A simplified basis of electroosmosis is described as follows. If the wall of a channel containing water has a negatively charged surface, the water will have an equal and opposite net positive charge. The layer of positive charge in the water is at the surface, and, for mammalian ionic strength, is about 10 Å thick. When an additional voltage is applied along the channel, the net positive charge on the surface of the water will move and carry water with it, as illustrated in Fig. 1. For negatively charged surfaces, the water will move in the same direction as electric current. There will be no pressure gradient with this water flow (*1, 2*).

At physiological *p*H and ionic concentrations, zeta potentials (a measure of surface charge) of −10 to −30 mv are uniformly obtained (*3*). While I am not aware of any studies having been made on neurons or neuroglia, it is reasonable to assume that these cells would be like all others. Therefore, a zeta potential of −15 mv will be assumed for the plasma membranes of neurons and neuroglia. (The surface charge is unrelated to transmembrane potential.)