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# Neonatal Alcohol Exposure Alters Suckling Behavior in Neonatal Rat Pups

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BARRON, S., S. J. KELLY AND E. P. RILEY. *Neonatal alcohol exposure alters suckling behavior in neonatal rat pups.* PHARMACOL BIOCHEM BEHAV **39**(2) 423–427, 1991.—Prenatal alcohol exposure has been associated with a variety of suckling deficits in both humans and animals. In this study, the effect of neonatal alcohol exposure on suckling performance was examined in 15-day-old rat pups. Neonatal alcohol exposure has been used as a model to study the effects of alcohol exposure during a period equivalent to the human third trimester with respect to brain growth. Subjects were Long-Evans rats which had been artificially reared (AR) and fed through gastrostomy tubes from postnatal day (PN) 4–PN 12. The AR groups included two groups given ethanol doses of 6 g/kg/day or 4 g/kg/day and an isocaloric maltose-dextrin control group. A suckled control group raised by their natural mothers was also included to control for artificial rearing. Fifteen-day-old pups were individually placed with an anesthetized dam for a 1-h videotaped test session. Pups in the 6 g/kg alcohol group took longer to attach to the nipple and spent less time suckling than pups from all other treatment groups. Nipple-shifting behavior was disrupted in all artificially reared groups, but it was most severely affected in the 6 g/kg group. These findings suggest that neonatal alcohol exposure interferes with suckling performance and these altered behaviors may contribute to the postnatal growth deficits that have been reported following alcohol exposure in utero.

Suckling deficits Neonatal alcohol exposure Fetal alcohol effects

PRENATAL alcohol exposure has been associated with a variety of feeding and suckling deficits in humans (16, 18, 32, 33). Alcohol-exposed infants took longer to attach and initiate suckling on a nonnutritive nipple. Additionally, the amount of pressure exerted on the nipple was weaker among alcohol-exposed infants relative to controls (16). These feeding difficulties may persist throughout infancy. For example, in one case study, three infants diagnosed with FAS required naso- and/or intragastric feeding at various periods during their first year of life. These infants showed abnormal sucking patterns, increased fatigue and increased distractibility while suckling. Although no long-term follow-up of these infants has been reported, it was noted that two of the children continued to require a special feeding program during their second year of life (33).

Similar types of deficits have been reported in rat pups following prenatal alcohol exposure. At 3 and 6 days of age, rat pups exposed to a 35% ethanol-derived calorie (EDC) liquid diet during prenatal development took longer to attach to the nipple of an anesthetized dam than controls (3). These deficits in attachment disappeared by 13 days of age (3) and were not the result of postnatal suckling experience since similar deficits were found in newborn pups tested within 30 min of birth prior to any suckling experience (23). Prenatal alcohol exposure also reduced suckling pressure at 6–7 and 9–10 days of age (25), although once again, these deficits disappeared by 15 days of age (22).

Nipple shifting has also been examined following prenatal alcohol exposure. When rat pups younger than 2 weeks of age attach to the nipple of a nonlactating anesthetized dam, they remain attached to the nipple although no milk is delivered. Starting at approximately 2 weeks of age, however, pups will begin to shift from one nipple to another when milk delivery is blocked. This nipple shifting is an integral component in the development of early feeding behaviors and has been suggested to be a measure of reactivity to the environment (4). Following prenatal alcohol exposure, 15-day-old male offspring displayed more nipple shifting and were more likely to leave the anesthetized dam than controls (22).

The present study was designed to examine the effects of neonatal alcohol exposure on suckling behavior in rat pups. The rationale for neonatal alcohol administration stems from temporal differences across species in central nervous system (CNS) development. Although the pattern of CNS development is relatively similar across species, the timing at which birth occurs relative to CNS development differs greatly. In rats, brain development in early postnatal life coincides with the third trimester of human development, a period noted for substantial CNS growth and proliferation (6,7). Therefore, administration of alcohol to neonatal rats has been used to assess the effects of alcohol during a period of CNS development that is equivalent to the third trimester "brain growth spurt" in human pregnancy (6,7).

## METHOD

### *Subjects*

Parent animals were Long-Evans rats obtained from Blue

Spruce Farms, Inc., Altamont, NY. Females were individually housed with males in the late afternoon and examined the following morning for the presence of a seminal plug which was taken as an indication that copulation had occurred. The morning the plug was detected was noted as Day 0 of pregnancy. Females were weighed and individually housed in breeding cages with ad lib rat chow (Wayne Rodent-Blox) and water in a temperature and humidity controlled nursery with a 12-h light/dark cycle.

Twenty-four hours following parturition, litters were weighed and culled to 5 males and 5 females. On PN 4, individual pups from each litter were assigned to one of four treatment groups; artificially reared (AR) receiving a 6 g/kg dose of ethanol, AR receiving a 4 g/kg dose of ethanol, AR receiving an isocaloric maltose-dextrin control diet, or sham surgery animals that remained with their dam as suckled controls. Within each litter, 1 male and 1 female pup were routinely assigned to each of the 4 possible groups.

*Neonatal Surgery Procedure*

The surgical procedure has been detailed in numerous reports and the reader is referred to these studies for additional details (5, 19, 20, 27). Briefly, on PN 4 (gestation day 26), pups in the artificially reared groups (AR) were anesthetized with halothane and implanted with an intragastric cannula made of polyethylene tubing (Clay Adams PE-10). A sham surgery group was also included. This sham control group underwent the same surgical procedure as the artificially reared pups except the gastrostomy tube was not implanted. Upon recovery from the anesthesia, the sham surgery pups were returned to their home cage and reared normally by their dams.

*Neonatal Rearing and Maintenance*

The AR pups were individually housed and maintained in plastic cups lined with hardwood chips and a piece of artificial fur, which was used to minimize the behavioral depression that may occur with maternal deprivation associated with AR (30). The cups floated in a stainless steel tank filled with aerated water maintained at 38°C.

The intragastric tubing was attached to a tygon tubing lead which connected to a syringe containing a formula that mimics rat milk [see (27)]. The syringes were mounted on a multi-syringe infusion pump (Harvard Apparatus Model #2265) which was operated via a timer and programmed to administer milk for 20 min every 2 h resulting in 12 daily feeds. Each morning, the pups were weighed, cleaned and their bladders voided. The amount of milk infused each day was equivalent to 33% of their average daily body weight. Alcohol exposure was confined to the 4 feeding periods that occurred between 1000 h and 1600 h in order to represent a condensed or "binge drinking" exposure model (12,20). During the remaining 8 feeding periods each day, all AR subjects received the stock milk solution. Maltose-dextrin was added to both the 4 g/kg ethanol and maltose-dextrin diets to make the formulas isocaloric with the 6 g/kg ethanol group. The AR animals were maintained under these conditions from PN 4–10. On PN 11 and PN 12, all AR subjects were maintained on the stock milk formula in order to allow the alcohol-exposed animals to recover from any acute effects of ethanol and ethanol withdrawal, which occasionally were observed.

From PN 4 through PN 12, the sham surgery control pups were also weighed daily. These pups were maintained with their natural dam. An additional six surrogate pups were placed with each dam in the home cage, in order to maintain the lactational performance of the dam until the AR pups were returned to their litter.

On PN 13, the six surrogate pups were removed from the litters and replaced with six AR pups. Prior to returning the AR pups to the nest, the cannulas were removed from the nape of the neck and cut close to the abdominal wall and the pups were ear-punched for later identification. Both the AR and sham-operated controls were bathed in a slurry of feces and water from the mother's home cage and then returned to the dam. With this procedure, there was a very low incidence of pup mortality.

*Blood Alcohol Measures*

Blood alcohol concentrations were measured in all AR subjects 30 min after the last alcohol feed on PN 6. This day was routinely used to sample blood alcohol levels since the peak of the "brain growth spurt" occurs between PN 6 and PN 8 (7). To obtain blood samples, a 1 mm cut was made at the tip of each pup's tail and a 10 μl sample of blood was taken. The sample was analyzed using an enzymatic assay (10).

*Experimental Procedure*

Pups were removed from their home cage on PN 14 for a 15-h deprivation period (n's = 15, 16, 17 and 17 for 6 g/kg, 4 g/kg, maltose-dextrin and sham, respectively). During this time, they were kept with conspecifics in a breeding cage on a warm heating pad. Testing for suckling behavior was conducted on PN 15. Dams were anesthetized with pentobarbital (80 mg/kg), which blocks milk letdown, and placed in a supine position in the center of a heated breeding cage (19″ × 10″ × 8″). Each pup was weighed and individually placed in the lower right corner of the test cage for a 1-h videotaped test session. At the conclusion of the test, each pup was weighed again and inspected to determine if its eyes were open. The pre- and posttest weighings were conducted to ensure that milk letdown had been blocked.

The videotape was scored by an experimenter blind to treatment condition with a real-time event recorder. The dependent measures included the latency to attach to the nipple, the total time spent suckling and the latency and number of nipple shifts (leaving one nipple and immediately attaching to another nipple).

*Statistical Analyses*

Parametric data were analyzed with analysis of variance (ANOVA). Significant interactions were broken down with simple main effect analyses and group differences discerned by Newman-Keul's post hoc comparisons. Chi square analyses were calculated for nonparametric percentage data. A probability value of 0.05 or less for all statistical analyses was considered significant unless otherwise stated.

## RESULTS

Nipple attachment latencies are presented in Fig. 1. The ANOVA yielded a significant effect of neonatal treatment, $F(3,57) = 12.72$, $p < 0.0001$. The 6 g/kg/day ethanol group displayed longer latencies than all other treatment groups ($ps < 0.01$) which did not differ from each other. No sex differences were observed for any of the dependent measures.

All of the pups in the 4 g/kg, maltose-dextrin and sham control groups attached to the nipple during the test session; however, 33% of the 6 g/kg pups (two males and three females) never attached during the 1-h test session. This resulted in a significant effect, $\chi^2(3) = 18.11$, $p < 0.01$. A chi square analysis

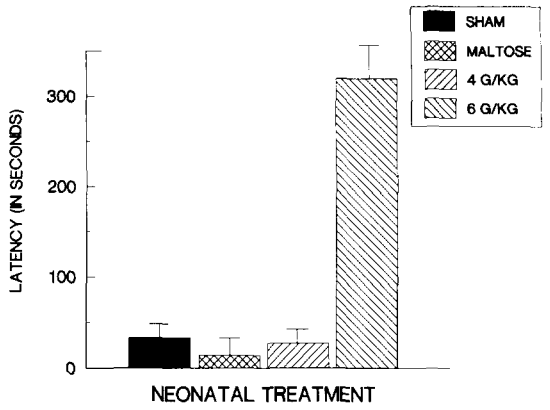


FIG. 1. Average nipple attachment latency ± SEM as a function of neonatal treatment.

was also conducted on the number of pups in each treatment group with both eyes open. Significantly fewer 6 g/kg pups had both eyes open at the time of testing relative to all other groups (42% for the 6 g/kg group vs. 100%, 93% and 90% for the 4 g/kg, maltose and sham groups respectively), $\chi^2(3) = 15.63$, $p<0.01$.

The amount of time spent suckling during the 60-min test session was also examined. Since there were group differences in nipple attachment latencies, a ratio was calculated to examine the proportion of time spent suckling after initial attachment. This proportion was calculated by the following formula: (seconds spent suckling)/(test session length − latency to attach). These data are presented in Fig. 2. Deficits appeared to be specific to the 6 g/kg female pups. Female 6 g/kg pups that attached to the nipple spent less of the remaining test session suckling relative to 6 g/kg males and all other groups [group × sex interaction, $F(3,52) = 3.69$, $p<0.02$]. The 6 g/kg males did not differ from the 4 g/kg, maltose-dextrin or sham control groups.

The percentage of pups displaying nipple shifting is presented in Table 1. The incidence of nipple shifting in the 6 g/kg group was very low (only 1 subject displayed this behavior). Both the 4 g/kg and maltose groups were intermediate and the sham surgery group had the greatest number of pups displaying nipple shifting, $\chi^2(3) = 21.69$, $p<0.01$.

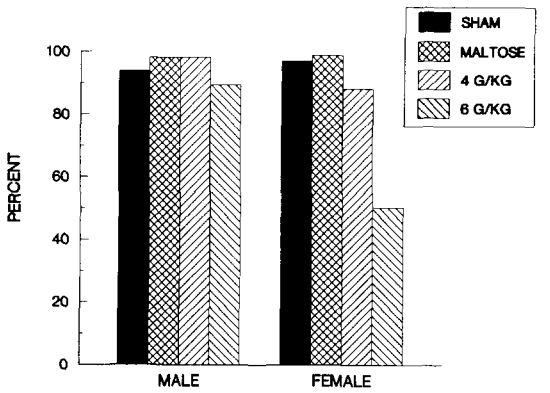


FIG. 2. Ratio of amount of time spent suckling after attachment as a function of neonatal treatment.

TABLE 1

PERCENT OF SUBJECTS DISPLAYING NIPPLE-SHIFTING

| Treatment Group | Percent |
|---|---|
| 6 g/kg | 6.6 |
| 4 g/kg | 44 |
| Maltose | 47 |
| Sham | 82 |

Frequency of Nipple-Shifting (mean ± S.E.M.)

| Treatment Group | N | Frequency |
|---|---|---|
| 6 g/kg | 1 | 3 |
| 4 g/kg | 7 | 2.83 (± 0.94) |
| Maltose | 8 | 2.63 (± 0.63) |
| Sham | 14 | 9.15 (± 1.75) |

Latency to First Nipple-Shift

| Treatment Group | N | Latency (in s) |
|---|---|---|
| 6 g/kg | 1 | 1776 |
| 4 g/kg | 7 | 1299 (± 552) |
| Maltose | 8 | 1605 (± 440) |
| Sham | 14 | 880 (± 254) |

Two additional related measures were examined among the pups that displayed nipple shifting: latency to the first nipple shift and the number of shifts during the test session. The means ± S.E.M. are presented in Table 1. It was impossible to include the 6 g/kg group in the ANOVA since only one subject in this group displayed nipple shifting, although the data for this single subject is also presented in Table 1. The sham group displayed significantly more episodes of nipple shifting than the 4 g/kg or maltose treatment groups, $F(1,26) = 6.32$, $p<0.01$. The latency to the first nipple-shifting episode did not differ across neonatal treatment groups.

Body weights on PN 13 and 15 are presented in Table 2. On PN 13, there were no significant differences in body weights among the AR pups although the AR groups weighed significantly less than the sham controls, $F(3,57) = 5.28$. On PN 15, the 6 g/kg group weighed significantly less than all other groups, the 4 g/kg and maltose groups were intermediate, and the sham group weighed the most, $F(3,57) = 16.10$, $p<0.001$. Blood alcohol levels on PN 6 averaged 367 ± 24 mg/dl (S.E.M.) for the 6 g/kg group and 265 ± 21.8 for the 4 g/kg group.

TABLE 2

MEAN BODY WEIGHT (IN g) ± S.E.M.

| Treatment Group | PN 13 | PN 15 |
|---|---|---|
| 6 g/kg | 25.57 ± 1.73 | 21.58 ± 1.20* |
| 4 g/kg | 27.43 ± 1.49 | 27.75 ± 0.83† |
| Maltose | 28.04 ± 0.898 | 25.60 ± 0.62† |
| Sham | 30.69 ± 1.42* | 31.33 ± 1.32* |

*Differs from all other same-age treatment groups, $p<0.05$.

†Do not differ from each other but differ from all other same-age groups, $p<0.05$.

## DISCUSSION

Neonatal alcohol exposure was associated with a variety of suckling deficits. Exposure to 6 g/kg ethanol resulted in an increased latency to attach to the nipple relative to all other treatment groups. In addition, fewer 6 g/kg ethanol pups displayed nipple attachment during the test session relative to the other neonatal treatment groups. A sex-specific effect was also observed in that the 6 g/kg female pups that attached to the dam's nipple spent less time suckling relative to all other neonatal treatment groups while this variable was not affected in the 6 g/kg males. These deficits in suckling behavior appear to be dose related since pups exposed to the lower ethanol dose (4 g/kg) did not show these deficits. Additional findings from this study suggest that while artificial rearing interfered with nipple shifting behavior, the 6 g/kg group was particularly affected on this measure. It should be noted that these suckling deficits were observed 5 days after the last alcohol exposure, suggesting that the effects were not due to the depressive actions of alcohol. Furthermore, these alterations in suckling behavior cannot be explained by the artificial rearing procedure since the suckling deficits appeared specific to the 6 g/kg artificially reared group.

One obvious question from these data concerns what the 6 g/kg pups were doing if they were not engaged in suckling behavior. The pups did not appear to be rooting or attempting to suckle unsuccessfully. Some pups merely sat near the dam, while others were very active, running around the test chamber. Neonatal alcohol exposure has been associated with overactivity in 16–20-day-old pups (12) which may be one explanation for the effects reported in the present study. In addition, neonatal alcohol exposure results in delays in a number of developmental milestones, particularly those that require a motor component such as head elevation, hindlimb elevation (17), and mid-air righting (11,27). Since suckling behavior requires certain motor capabilities in the pup, alcohol-related motor deficits may also contribute to the suckling deficits observed in this study. Furthermore, a significantly greater number of pups in the 6 g/kg group were delayed in eye opening. While the neonatal pup typically relies on other sensory modalities, i.e., olfaction, for nipple attachment and maintaining contact with the dam, this delay in eye opening may have also played a role in the suckling deficits observed in this study.

While suckling deficits have previously been reported in rat pups following prenatal alcohol exposure, most of the suckling deficits were not observed in older pups (3, 22, 25). For example, following prenatal alcohol exposure, normal attachment latencies were observed at 12–13 days of age. In the present study, 6 g/kg pups displayed longer attachment latencies at 15 days of age. Therefore, it appears that prenatal and neonatal alcohol exposure do not exert identical effects and that neonatal alcohol exposure may have a more prolonged effect on suckling behaviors than prenatal alcohol exposure. Alternatively, it should be noted that it is possible that alcohol's effects on suckling may be more temporally mediated, i.e., contingent on the amount of time between alcohol exposure and subsequent suckling behavior.

The sex differences in sensitivity to neonatal alcohol exposure on some suckling measures reported in this study are particularly interesting. We have previously reported two studies in which females appeared more sensitive to neonatal alcohol exposure. Deficits in passive avoidance learning and 24-h retention were observed in 23-day-old female rats exposed to alcohol neonatally although males did not differ from controls (1). Similarly, when tested in a Morris water maze, adult female rats exposed to alcohol neonatally showed performance deficits while males appeared unaffected (13). The data from the present study provides further support for a differential sensitivity between males and females to the effects of neonatal alcohol exposure. Furthermore, these data may help explain some of the long-term sex differences reported following neonatal alcohol exposure if female pups are not suckling as well and therefore, not obtaining adequate nutrition prior to weaning.

Body weights at PN 13 and PN 15 provide support for these suckling deficits. When pups were artificially reared, the amount of milk formula consumed was controlled by the experimenter. During this time, there were no weight differences between the AR groups. After pups were returned to the nest, however, the pups were required to suckle and the 6 g/kg group gained less weight than the other AR groups. It should be noted that it is also possible that the lower body weight of the 6 g/kg offspring may have contributed to some of the deficits in suckling behavior observed in this study.

One obvious behavioral difference between the artificially reared groups and the normally reared sham control group was the incidence of nipple shifting, with the AR groups displaying less nipple shifting than the sham surgery group. Since nipple shifting appears to be a distinctive stage in the maturation of early suckling and feeding behaviors among rat pups (4), it is not surprising that the artificially reared groups displayed either developmental delays or permanent changes in the onset of this behavior, since they had been deprived of normal suckling experience for a number of days. It is important to note that although AR pups were less likely to display nipple shifting than sham controls, the 6 g/kg group was still the most affected group. This may be due, in part, to the fact that the 6 g/kg pups spent less time suckling and did not have as much opportunity to display nipple-shifting. Alternatively, artificial rearing in combination with exposure to the high dose of alcohol may have produced a greater developmental delay in the emergence of nipple-shifting behavior.

While the CNS structures that mediate suckling behavior are not well understood, there is a considerable amount of data regarding the neurotransmitters involved. Serotonin is involved in many of the components of suckling behavior. While administration of serotonergic antagonists to 3- to 8-day old pups increased attachment latencies and decreased suckling time, it had either no effect or an opposite effect in weanling aged pups (14, 15, 24, 34). These data suggest that the serotonergic system undergoes critical developmental changes during the first 2–3 weeks of life. There is also some evidence suggesting cholinergic involvement in suckling behavior since scopolamine, a cholinergic agonist, reduced suckling in younger pups (24). Prenatal alcohol exposure has been reported to affect these neurotransmitter systems. For example, decreased serotonin uptake in cerebral cortex, decreased synthesis of 5-hydroxytryptophan, and a reduction in 5-$HT_1$ but not 5-$HT_2$ binding sites [see (8, 9, 28, 29)] have been reported following prenatal alcohol exposure and one study has reported alcohol-related deficits in the cholinergic system following pre- and early postnatal alcohol exposure (21). Since both of these systems undergo substantial postnatal development (2,31), it is possible that this may be another mechanism by which third trimester alcohol exposure affects suckling.

These findings may have important clinical implications because they emphasize the potential significance of "third trimester alcohol exposure" in the suckling deficits displayed by human infants with prenatal alcohol histories. There are clinical data that suggest that women who abuse alcohol throughout pregnancy are more likely to have an infant with growth retardation and congenital malformations than alcoholic women who reduce or abstain from drinking before the third trimester (26).

The severe suckling deficits associated with "third trimester" alcohol exposure suggest a possible explanation for the improved postnatal growth in infants of women who reduce their drinking prior to the third trimester.

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