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Effects of Lactation on Fetal Survival and Development in Rabbit Does Mated Shortly After Parturition[1]

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ABSTRACT: Primiparous rabbit does were mated within 12 h after parturition (d 0). They were immediately weaned (Group W; n = 31) or allowed to suckle 10 young rabbits (Group L; n = 32). Blood samples were collected at d 0, 7, 17, and 28. Feed intake and live weight were measured weekly and pregnant does (Group W, n = 24; Group L = 25) were slaughtered at d 28. Feed intake was 78% higher in L than in W females throughout gestation ($P < .001$). However, L females lost weight during the second half of gestation (−243 ± 25 g) compared with W females, which gained weight (246 ± 20 g). The weights of carcass, skin, and adipose tissues were severely reduced at d 28 in the L group ($P < .01$). Ovulation rate (11.0 ± .3 corpora lutea) and early embryonic mortality (< d 15) were similar in both groups (12.3 ± 2%), whereas late embryonic mortality (≥ d 15) was higher in L than in W does (13.9 ± 3 vs 3.9 ± 1%; $P < .01$). Fetal weight was reduced by nearly 20% in L compared with W females ($P < .01$). Plasma concentrations of progesterone were lower in L than in W females at d 7 and 17 ($P < .001$), whereas concentrations of estradiol were similar in both groups throughout gestation. These results indicate that fetal survival and development can be impaired in lactating females.

Key Words: Lactation, Gestation, Fetal Death, Fetal Growth, Progesterone, Rabbits

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Introduction

In rabbit does, ovulation is induced by coitus. The high receptivity immediately after parturition allows concurrent gestation and lactation (Hammond and Marshall, 1925). When a female is pregnant during lactation she must supply the nutrients necessary for growth of both fetuses and young, and a situation of competition can occur. This can be particularly crucial in the rabbit species, because energetic requirements for fetal growth and milk production are very high (Chilliard, 1986).

Previous workers have shown that occurrence of ovulation (Foxcroft and Hasnain, 1973), ovulation rate (Harned and Casida, 1969; Lamb et al., 1991), and fertilization of oocytes (Torres et al., 1977) are lower in lactating than in nonlactating does. Effects of lactation on embryonic and fetal mortality are controversial. A higher mortality was observed in some experiments (Selme and Prud'hon, 1977; Garcia et al., 1983; Garcia and Perez, 1989), whereas no effect was observed in others (Harned and Casida, 1969; Torres et al., 1977; Partridge et al., 1984; Lamb et al., 1988). Endocrine mechanisms underlying such effects have been poorly investigated.

The aim of this experiment was to compare reproductive performance and steroid patterns in rabbit does mated shortly after parturition and in which the young continued to nurse or were weaned.

Materials and Methods

Sixty-three primiparous crossbred does (22 ± .2 wk of age) from the INRA lines A1066 and A1077 were mated within 12 h after parturition (d 0). Does were assigned randomly at parturition to one of two experimental groups: does in Group W were weaned immediately after parturition (n = 31), whereas does in Group L were allowed to lactate (n = 32). Mean litter size was 9.7 ± .3 total rabbits born and 9.0 ± .3 born alive. Litters were equalized at 10 young rabbits after crossfostering.

Does had ad libitum access to a diet containing 17.5% CP and 2,400 kcal/kg of DE. Feed intake was determined weekly from mating to d 28 of gestation. Young rabbits from the L group had free access to

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their dam's diet. Litters and does were weighed weekly.

Blood samples (4 mL) were collected in heparinized tubes on d 0, 7, and 17 by puncture of an auricular artery. Rabbit does were slaughtered on d 28, and a blood sample was collected at that time. Samples were immediately centrifuged and plasma was stored at −20°C until it was assayed for steroid concentrations.

Plasma progesterone and estradiol were quantified by RIA as previously described (Thibier and Saumande, 1975). For progesterone, the sensitivity of the assay was .2 ng/mL, and intra- and interassay CV were 9 and 12%, respectively. For estradiol, the sensitivity of the assay was 5 pg/mL, and intra- and interassay CV were 10 and 25%, respectively. Estradiol data on d 7 are missing because of an error in the RIA and sufficient plasma was not available for a reanalysis.

Immediately after slaughter on d 28, the reproductive tract was removed and dissected. Placentas and fetuses were counted and weighed. Three classes of fetuses were defined: 1) live (**L**), when fetuses were well developed and already moving or breathing; 2) resorbed (**R**), when the fetus was not recognizable and only the placenta was present (this mortality may have occurred between d 15 and 20 of gestation); and 3) dead (**D**), when a fetus was recognizable, but unmoving and showing marked developmental delay (this mortality may have occurred between d 20 and 27 of gestation).

Uterine horns and ovaries were weighed and ovulation rate was determined by counting the number of corpora lutea (**CL**). Embryonic and fetal mortality (percentage) were defined as follows: total mortality (TM) = [CL − L]*100/CL; early mortality (EM) = [CL − (L + R + D)]*100/CL; and late mortality (LM) = [(R + D)*100]/[L + R + D].

Early mortality included all CL that were not represented by a fetus (live, resorbed, or dead) at slaughter. It may have occurred before d 15, including fertilization failures. Late mortality included resorbed or dead fetuses, and it may have occurred after d 15.

Does were dissected and carcass (muscles, bones, heart, lung, and liver), skin, full digestive tract, and adipose tissues (perirenal and interscapular tissues) were weighed.

Data from pregnant does were analyzed by analysis of variance using the GLM procedures (SAS, 1987). For reproductive performance and characteristics at slaughter, the main effect was treatment. Analysis of mortality was made with the actual numbers (e.g., CL − L values for total mortality). Hormone concentrations, live weight, and feed intake were analyzed according to a split-plot design including the effects of treatment, the effect of rabbit doe within treatment (error to test the treatment effect), the effect of stage of gestation, and the interaction of treatment × stage of gestation. When the interaction was significant, comparisons between treatments were made for each stage of gestation by a Student's *t*-test.

Table 1. Traits of body development in does that were lactating (L) or not lactating (W) during gestation

Item	Treatment groups W	L	SEM
No. of pregnant does	24	25	—
Live wt, g	4,264	3,851**	53
Carcass wt, g[a]	2,445	2,100**	39
Skin wt, g	628	524**	12
Digestive tract wt, g	412	557**	13
Adipose tissues wt, g[b]	131	39**	8
Uterine wt, g	54	42**	2

[a]Muscles + bones + heart + lung + liver.
[b]Perirenal + interscapular tissues.
**Different between treatment groups, $P < .001$.

Results

All does were receptive to the male and were mated within 12 h after parturition. The percentage of pregnant does was similar (77%) in the W (24 of 31) and L (25 of 32) groups.

The interaction of treatment × stage of gestation was significant for feed intake and live weight of does; therefore, comparisons between treatments were made for each stage of gestation. Feed intake of does in Group L was 78% greater than in Group W throughout gestation (354 ± 7 g/d vs 199 ± 8 g/d; $P < .001$). Live weight of does was similar in the two groups from mating (3,566 ± 44 g) until d 14 (4,056 ± 47 g). Live weight was 5% lower in L than in W does at d 21 (3,967 ± 49 vs 4,169 ± 78 g) and 10% lower at slaughter (3,821 ± 50 vs 4,264 ± 74 g). The L does lost weight, whereas W does gained weight during the second half of gestation. Overall weight gain during pregnancy was higher among does from Group W than from Group L (722 vs 260 g). Similarly at d 28, the weights of carcass, skin, and adipose tissues were lower in the L group (Table 1; $P < .01$); however, weight of the digestive tract was higher in this group ($P < .01$). In Group L, mortality of the young during lactation was 2.4% and BW of live young at d 28 was 481 ± 18 g.

Ovulation rate (CL = 11.0 ± .3) and ovarian weight (.45 ± .01 g each) were similar between two groups (Table 2). Total mortality tended to be higher in the L group than in the W group (Table 2; $P = .1$). This difference was due to late mortality that was nearly four times greater in the L group ($P < .001$), whereas early mortality was similar between groups (Table 2). The increase in late mortality was mainly related to the number of resorbed fetuses ($P < .01$). However, the same tendency was observed for dead fetuses (Table 2). Individual live fetus weight was approximately 20% lower in the L than in the W group ($P < .01$). The weights of uterine horns and placentas were lower in the L group ($P < .01$; Table 2); however, the difference in uterine horn weight between the two

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Table 2. Reproductive characteristics of does that were lactating (L) or not lactating (W) during gestation

Item[a]	Treatment group W	L	SEM
No. of pregnant does	24	25	—
No. of corpora lutea	11.1	10.9	.28
Ovarian wt, g	.45	.44	.02
No. of fetuses			
Live	9.4	8.2	.36
Resorbed	.25	.92*	.13
Dead	.13	.40	.10
TM, %	15.3	24.8	3.64
EM, %	11.9	12.7	2.45
LM, %	3.9	13.9**	2.95
Fetal wt, g	40.2	32.3*	.78
Placenta wt, g	7.8	7.1*	.14

[a]TM = total mortality; EM = early mortality; and LM = late mortality. See text for calculations.
*Different between treatment groups, $P < .01$.
**Different between treatment groups, $P < .001$.

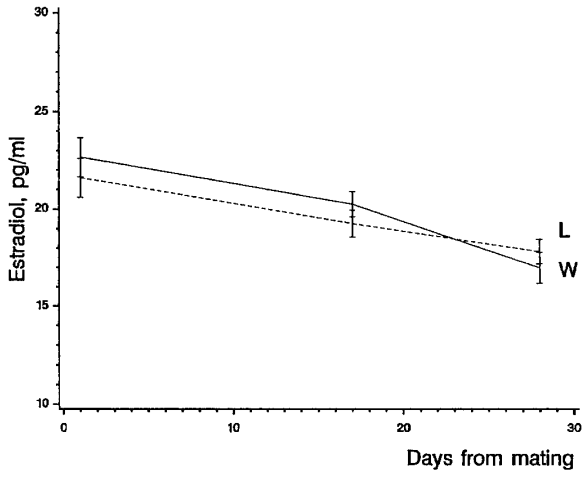

Figure 1. Concentrations of estradiol throughout gestation in does that were lactating (L) or not lactating (W).

groups was not significant when the number of fetuses was included as a covariate in the statistical model.

Concentrations of plasma estradiol decreased through gestation ($P < .01$), but there was no difference between treatment groups, and the interaction of treatment × stage of gestation was not significant (Figure 1). In contrast, the interaction of treatment × stage of gestation was significant for plasma progesterone. In the W group, progesterone did not vary through gestation, whereas in the L group, progesterone decreased between d 1 and 7. The concentration of plasma progesterone was lower in the L group than in the W group on d 7 and 17 of pregnancy ($P < .001$) but not on d 1 and 28 (Figure 2).

Discussion

These results indicate that fetal survival and development can be impaired when does are simultaneously pregnant and lactating. An increase in mortality seemed to occur after d 15 of gestation, because a greater number of resorbed fetuses were found at d 28 in lactating females. This period (d 15 to 20 of gestation) coincided with the period of maximal milk production. Two hypotheses, which are not exclusive, can explain this impairment of fetal survival and development: nutrient requirements of fetuses are not satisfied, and(or) lactation induces a hormonal environment that is unfavorable for fetal development.

It has been shown previously that the energetic balance of rabbit does is negative during the second half of gestation (Jean-Blain and Durix, 1985; Parigi-Bini et al., 1990a) and at the end of lactation (Parigi-Bini et al., 1990b). Thus, the energy deficit is certainly very high in does that are pregnant and lactating simultaneously. This phenomenon may be particularly important in the doe because the rabbit is a species with relatively low lipid reserves (Ouhayoun et al., 1986).

To limit their energy deficit, lactating does increased their feed intake. However, this increase was not sufficient and lactating does lost weight between d 14 and 28 of gestation. This weight loss corresponded

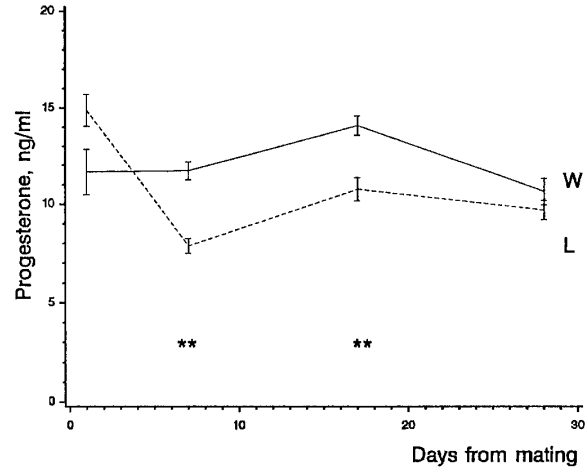

Figure 2. Concentrations of progesterone throughout gestation in does that were lactating (L) or not lactating (W). Differences between treatment occurred at d 7 and 17 (**$P < .001$).

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to protein and lipid mobilization, because the weights of carcass, skin, and adipose tissues were lower at slaughter in L than in W does.

The higher weight of the digestive tract in Group L can be at least partly explained by the higher feed intake observed in this group, because digestive contents were not removed. Milk production of lactating does seemed not to be affected, because growth rate of suckling young was in a normal range, compared with other results (Lebas, 1969). Similarly, Mendez et al. (1986) did not observe any effects of gestation on milk production of suckling pregnant does during the first 20 d of lactation.

The hypothesis of a nutritional deficiency is plausible because the peak of lactation occurs at approximately d 15, when fetal death probably occurred. Moreover, in rabbits, mammary glands and fetoplacental units use the same substrates such as glucose, long-chain fatty acids, and free fatty acids (Elphick and Hull, 1977; Jones and Parker, 1981; Jones and Rolph, 1985; Fraga et al., 1989; Stephenson et al., 1990). In addition, there is an antagonism between the increase in blood flow toward the uterus during gestation (Gilbert et al., 1984) and its redirection toward the mammary glands during lactation (Jones and David, 1988). In rabbits and guinea pigs, a positive correlation was found between placental blood flow and fetal weight (Bruce and Abdul-Karim, 1973; Myers et al., 1982). Consequently, lower fetal weight observed in lactating females could be explained by a lower blood flow to the uterus, which competes with the blood flow to the mammary glands.

Among the hormones controlling the uterine activity during gestation, prolactin, estradiol, and progesterone play important roles. Prolactin was not assayed, but it is well known that its secretion is increased during lactation (Durand and Djiane, 1979). This hormone has been shown to be detrimental to embryonic viability (Daniel et al., 1988) and to affect uterine function (Daniel et al., 1988; Young et al., 1989) and uterine secretory activity during pregnancy (Chilton and Daniel, 1987; Daniel and Juneja, 1989). Thus, higher prolactin secretion in lactating does cannot be excluded as a cause of increased embryonic death observed in the present experiment.

Our experiment shows that progesterone levels were lower on d 7 and 14 in lactating does than in does whose young were weaned. In our experiment, the difference in ovulation rate was not involved because it was similar in both groups of females. Estradiol has been shown to have an indispensable luteotrophic role in the pregnant rabbit (Gadsby et al., 1983). However, in our study, estradiol levels were similar in both weaned and lactating does. The origin of lower progesterone levels remains unknown but several hypotheses can be made: 1) the action of estrogen is regulated by a placental luteotrophic hormone (Gadsby and Keyes, 1984), placentas were lighter in the L group than in the W group; consequently, compromised secretion of placental luteotropin may contribute to decreased progesterone concentrations; 2) prolactin is required to sustain pregnancy in the rabbit (Hilliard, 1973), whereas high levels of prolactin decreased progesterone production in rabbit (Lin et al., 1987) and therefore progesterone production may be lowered during lactation; 3) progesterone production in the rabbit CL is dependent on lipoprotein-delivered cholesterol (Holt, 1989), the mammary gland is a site that uses lipoproteins for milk production (Guesnet and Demarne, 1987) and therefore the supply of lipoproteins to the ovary may not meet the requirements for optimal luteal steroidogenesis in lactating does; and 4) the high feed intake that occurs during lactation may alter the metabolic turnover rate of progesterone and influence its peripheral concentration (Symonds and Prime, 1989; Lamb et al., 1991). Whatever the origin, it can be proposed that lower progesterone levels lead to higher fetal mortality. The work of McCarthy et al. (1977) emphasized the need for adequate progesterone levels to maintain a normal pregnancy. Moreover, low peripheral progestin levels were associated with higher fetal death in aged rabbits (Spilman et al., 1972). The high progesterone concentration in plasma observed on d 1 may be related to the peak due to copulation (Spilman and Wilks, 1976).

Implications

Nature has accorded a high priority to the function of pregnancy and to milk secretion. This involves homeorhetic controls arising from the conceptus and the mammary gland, respectively. The present experiment demonstrates that fetal mortality is increased and fetal growth is reduced in lactating does mated immediately after parturition, despite a 78% increase in feed intake and the mobilization of maternal body reserves. A nutritional defect and(or) a hormonal disturbance may explain this phenomenon.

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