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# Evidence That a Novel Type of Progestational Phase Control Occurs in the Corn Mouse, A South American Murid Rodent[1]

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## ABSTRACT

**Most Muridae display a short luteal phase that becomes functional as a consequence of the prolactin release induced by the stimulation of copulation and/or lactation. The corn mouse also shows a short luteal phase, and we wanted to know whether copulation and/or lactation would release prolactin and maintain progesterone secretion in this species. Females in postpartum estrus were either allowed to copulate with an intact male or not, and either to lactate their young or not. Afterward, plasma progesterone was elevated over the baseline level only in females that had copulated and were bearing growing embryos (whether or not they were lactating), while prolactin was elevated only in lactating females.**

**In another experiment, endometrial scratching induced decidualization both in females that had copulated with a vasectomized male and in those that had not copulated; sham operations had no effect in either case. Progesterone levels were elevated in decidualized animals as compared with their sham-operated controls.**

**Results indicate that the initiation of the progestational phase in the corn mouse is not dependent on prolactin release. A short luteal phase during which nidation may occur has not yet been described in any other mammal.**

## INTRODUCTION

Conaway [1] has proposed that type III ovarian cycles (i.e., those characterized by a combination of spontaneous ovulation, short luteal phases in those females that have not copulated, and the ability to develop a fully functional progestational phase if copulation does occur) have evolved among murid rodents as an adaptation to high predator pressure. In fact, this combination increases the probability that a female will become pregnant by allowing her to return rapidly to estrus if copulation does not occur.

Type III cycles are not known to occur outside the family Muridae (sensu [2]). Their underlying physiological mechanisms have been extensively studied in the laboratory rat [3, 4], and there is evidence that similar adaptations may be present in other members of the subfamily Murinae [5, 6] as well as in other murid subfamilies: Arvicolinae [7], Gerbillinae [8, 9], Cricetinae [6, 10–12], and Sigmodontinae [6, 13].

At least in the laboratory rat, the ability to extend a short luteal phase (from 2–3 days to about 2 wk) is based on the existence of two physiological mechanisms: 1) an increase in prolactin secretion, occurring in response to copulation, that shows a unique temporal pattern of two daily surges (one at the end of the light period of the day and the other at the end of the dark period) [14]; and 2) the manifestation by prolactin of a marked luteotrophic activity that results in the rescue of the corpus luteum of ovulation [14]. Also, prolactin is released by suckling stimulation during lactation [15], resulting in the luteotrophic stimulation of the corpora lutea formed in postpartum ovulation and in a massive and prolonged secretion of progesterone [16].

According to Reig's view [17, 18] of the evolution of the family Muridae, Neotropical Sigmodontinae would have been an early sprout of the phylogenetic tree of the Muridae that arrived in South America during late Oligocene/early Miocene times and there evolved in isolation since. It seemed interesting, therefore, to investigate the extent to which progestational phase control processes of Neotropical Sigmodontinae may differ from those of other Muridae, particularly those found in Nearctic Sigmodontinae and in the well-known laboratory rat (*Rattus norvegicus*).

The present paper deals with the effect of copulation and lactation on prolactin and progesterone secretion in the corn mouse (*Calomys musculinus*, a Neotropical Sigmodontinae). These are small (15–20 g, 14-cm length) vesperal-nocturnal mice that live mostly above ground in partially overlapping home ranges. They can be found mostly in corn fields, other croplands, and pastures. Along the more arid borders of their range, they are most frequently found near water, in thick stands of grass or of relatively lush vegetation [19]. The corn mouse is the main reservoir of Junín virus, the causal agent of the Argentine hemorrhagic fever [20], and it may also be infected by *Trypanosoma cruzi*, the causal agent of Chagas-Mazza disease [21].

## MATERIALS AND METHODS

### Animals

Colony-bred animals, at least 3 mo old, were used. The colony originated from animals captured in two localities (La Pega and Ñacuñán) of the province of Mendoza (Argentina). It should be noted that *Calomys* taxonomy has been particularly unstable and that it may result in some confusion, with different studies utilizing different nomenclatures. In this study we have followed the nomenclature of Massoia et al. [22], which distinguishes *C. musculinus* from *C. laucha*. Sample specimens (the skull and skin of 5 adult females and 5 adult males) of our colony were deposited at the mammal collection of the Instituto Argentino de Investigaciones de Zonas Aridas, Mendoza, Argentina (the females were identified as #CM-03540 to #CM-03544, and the males as #CM-03545 to #CM-03549).

Each female was caged with a male, in a shoe box-type plastic cage (12 × 20 × 30 cm), under 14L:10D at 24°C, with free access to a commercial rodent diet and water. In all cases, the males were removed from the cage 2–3 days before the expected day of parturition (normal length of

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gestation is 20–21 days) to make sure that copulation would not occur at postpartum estrus without being observed.

### *Experiment I*

The experimental groups resulted from combining the effect of copulation (C+) or no copulation (C−), and of lactation (L+) or no lactation (L−). Animals from the four groups (i.e., C+/L+, C+/L−, C−/L+, and C−/L−) were killed on Days 3, 6, 11, and 16 after postpartum ovulation (the latter was arbitrarily assumed to occur at the middle of the dark period after parturition, and the following day was taken as Day 1). In all L− groups, and in an additional control group that was killed on Day 1, the litter was removed immediately after the pups were observed. In the groups that were allowed to copulate (C+ groups), the pair were placed together again at the beginning of the dark period [23]; copulation occurred under visual inspection and until a satiety criterion of 30 min without penile intromission was met.

### *Experiment II*

The experimental groups resulted from combining the effect of copulation with a vasectomized male (V+) or no copulation (V−) with the surgical induction of decidualization (D+) or sham operations (D−). Decidualization was induced by endometrial scratching on day 3 after postpartum ovulation (i.e., 1 day earlier than the optimal time in the rat [24], since there is evidence that implantation occurs earlier in this species [25]). All animals were killed on Day 8. In all groups, the litter was removed immediately after the pups were observed. In the groups that were allowed to copulate (V+ groups), each female was mated at the beginning of the dark period with a vasectomized male; copulation occurred under visual inspection until a satiety criterion of 30 min without penile intromissions was met.

### *Surgical Procedures*

Males with sexual experience were vasectomized under ether anesthesia. A 1-cm segment of each vas deferens was removed through a small scrotal incision, and the animals were allowed to recover for 2 h in a warm environment. They were used for experimentation at least 3 wk after surgery; lack of sperm in the ejaculate was confirmed in the female's vaginal smear after copulation.

Decidualization was induced in postpartum females under ether anesthesia at the end of the light period. The uterus was exposed through a ventral approach, and a small knife [24] was used to stimulate the antimesometrial endometrium of both horns. Sham-operated animals were similarly treated except that the knife was not passed into the uterus. All females were allowed to recover for 2 h in a warm environment.

### *Killing of Animals, Plasma Sampling, and RIAs*

Animals were killed by decapitation during the last 2 h of the light period of each day. Trunk blood was collected in heparinized tubes and centrifuged; the plasma obtained was kept frozen (−30°C) until the hormone assays were run. In experiment I, the number of implanted embryos was also recorded at the time the animal was killed. In experiment II, the uteri were excised out and weighed in order to quantify the decidual reaction, which was also confirmed histologically (Bouin's fixation, paraffin embedding, and hematoxylin-eosin staining).

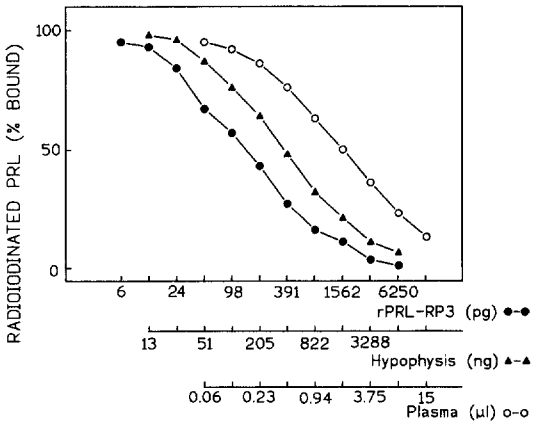


FIG. 1. Displacement curves of radioiodinated prolactin (NIDDK-rPRL-I-5) bound to anti-prolactin serum AFP-131078, by either a rat prolactin standard (NIDDK-rPRL-RP3), an extract of corn mice hypophyses, or blood plasma of castrated male corn mice, treated with estradiol benzoate (100 µg/kg, 72 h before animals were killed) and sulpiride (17 mg/kg, 20 min before animals were killed).

Prolactin was measured in plasma (15-µl duplicate aliquots) with a heterologous assay that used an antiserum against *Mus musculus* prolactin (AFP-131078) and purified rat prolactin for iodination (rPRL-I-5; NIDDK, Baltimore, MD). An extract obtained from corn mice anterior hypophyses (i.e., the supernatant obtained after centrifugation of a homogenate of glands in 0.1% Triton X-100, 0.32 M sucrose, 0.01 M phosphate buffer) was used to displace radioiodinated prolactin in standard curves.

Possible proteinase contamination of this extract was controlled for by incubation of radioiodinated purified rat prolactin (200 000 cpm, in PBS-BSA/50% glycerol) with a 1:6000 dilution of the hypophyseal *Calomys* extract in 0.01 M phosphate buffer-0.9% ClNa with 1% BSA at a final volume of 200 µl during 24 h at room temperature. Control amounts of radioiodinated prolactin were incubated without the extract under similar conditions. After incubation, an aliquot of 2.5 µl of each sample was separated by SDS-PAGE electrophoresis (15%) according to Laemmli [26]. An empty lane was used as background-noise control. Intensity of the radioactive bands was determined by densitometry of a radioautograph of the electrophoretic run. Radiolabeled prolactin incubated with and without the extracts appeared as a single peak in the radioautograph. Densitometry of the lower-molecular-weight part of the lanes was undistinguishable from background-noise level.

The equivalence of this corn mouse standard in terms of a widely used reference preparation (NIDDK-rPRL-RP3) was determined by using the same RIA and was found to be 27 ng/mg of hypophysis. The range of concentrations of the corn mouse standard used in all assays was equivalent to 0.8–416.6 ng/ml of NIDDK-rPRL-RP3. Linear regression of the displacement of the antiserum-bound, radioiodinated prolactin (between 20% and 80% cpm bound) by either the rPRL-RP3 standard, the hypophyseal extract, or a pool of blood plasma obtained from corn mice (castrated, estrogen-treated males, treated with sulpiride to increase prolactin secretion [27]) yielded parallel curves, as indicated by similar slopes and different y intercepts (analysis of covariance; $p > 0.05$ and $p < 0.0001$, respectively [28]) (Fig. 1). The sensitivity of the assay was defined by adding known amounts of rPRL to plasma obtained from females

TABLE 1. Embryo implantation in females that had copulated during postpartum estrus (i.e., C+ groups) and that were either lactating or not.

| Days after ovulation | Lactation | Number of cases | Females with implantations (%) | Implantations/ female* (mean ± SEM) |
|---|---|---|---|---|
| 3 | no | 7 | 0 | — |
| | yes | 8 | 0 | — |
| 6 | no | 8 | 87 | 5.6 ± 1.0 |
| | yes | 10 | 50 | 6.2 ± 1.5 |
| 11 | no | 8 | 100 | 5.3 ± 0.7 |
| | yes | 9 | 78 | 6.6 ± 0.9 |
| 16 | no | 6 | 100 | 6.8 ± 1.1 |
| | yes | 9 | 89 | 8.5 ± 0.5 |

* Only those females that bore at least one implantation site were included.

that were separated from their litters after birth and were treated with bromocriptine mesylate (6.7 mg/kg BW, administered 12 h before animals were killed on Day 6 postpartum); the smallest amount of plasma-diluted prolactin that caused a significant displacement of the antiserum-bound labeled prolactin was found to be 1.6 ng NIDDK-rPRL-RP-3/ml of plasma (or 24 pg/tube). The intraassay coefficient of variation was 8.9%. The values reported in this paper were obtained in two assays (the interassay coefficient of variation of 8 assays, including the ones reported here, was 13.9%).

Progesterone determinations by RIA were made on 10-μl plasma aliquots. The labeled hormone was [1,2,6,7-$^3$H]progesterone (New England Nuclear, Boston, MA; spec. act. 90–115 Ci/nmol). The antiserum against progesterone was raised in rabbits in this laboratory and used in a 1:15 000 dilution, to obtain about a 40% binding of labeled progesterone. Cross-reactivity of this antiserum (progesterone 100%) was determined for 20α-hydroxyprogesterone, 17α-hydroxyprogesterone, corticosterone, pregnenolone, testosterone, and estradiol-17β and was found to be less than 1%. The sensitivity of the assay was 4.97 nM (0.156 ng/ml or 1.56 pg/tube), determined as the smallest amount of progesterone added to charcoal-treated plasma that caused a significant displacement of the antiserum-bound labeled hormone. Other data on the validation of this assay have been published [29].

### *Statistics*

Comparisons of the percentage of animals showing either implanted embryos or decidualization were made by Fisher's Exact Probability test [30]. Multigroup comparisons of hormone levels and tissue weights were made by either one-way or two-way analysis of variance (ANOVA I or II), followed by the Tukey test as post hoc analysis [28]. Significance level was fixed at $p < 0.05$.

## RESULTS

### *Experiment I*

Embryo implantation occurred between Day 3, when no uterine nodules were apparent, and Day 6, when large implantation sites were seen in at least half of the animals that had copulated (Table 1). Lactating animals tended to show a lower incidence of implantation than their nonlactating counterparts; this tendency was more marked on Day 6 than on Days 11 and 16, although it did not reach statistical significance, either when the lactating and the nonlactating groups were compared on each day ($p > 0.05$; Fisher's Exact Probability test) or when females from all these days were grouped as either lactating or nonlactating (20 of 28 lactating and 21 of 22 nonlactating females; $p = 0.0596$, Fisher's Exact Probability test [30]).

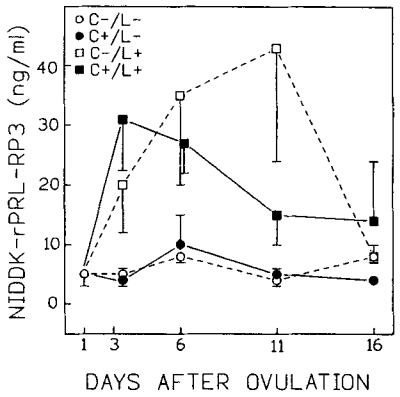


FIG. 2. Plasma prolactin levels (NIDDK-rPRL-RP3 ng/ml) in groups of postpartum females that had been either observed to copulate (C+) or not (C−), and were either lactating (L+) or not (L−). Day 1 was defined as the day after parturition. Symbols indicate mean values, and the vertical lines are standard errors of the mean. The number of cases in each group was 7–10. The levels were significantly increased in lactating female corn mice as compared with their nonlactating control groups (C+/L+ and C−/L+ groups vs. C+/L− and C−/L− groups; $p < 0.05$, ANOVA II, Tukey test). There were no significant interactions by days, or by days and groups (same test).

The changes in plasma prolactin concentration are presented in Figure 2. The levels were significantly increased in lactating female corn mice as compared with their nonlactating control groups ($p < 0.05$, C+/L+ and C−/L+ groups vs. C+/L− and C−/L− groups; ANOVA II, Tukey test); no other significant differences by groups were found. There were no significant interactions by days, or by days and groups (same test).

The changes in plasma progesterone levels in groups of postpartum females are presented in Figure 3. The initial

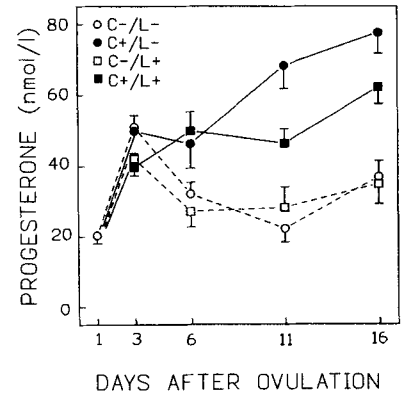


FIG. 3. Plasma progesterone levels (nM) in groups of postpartum females that had been either observed to copulate (C+) or not (C−) and that were either lactating (L+) or not (L−). Symbols indicate mean values, and the vertical lines are standard errors of the mean. Progesterone levels in C+ females from Days 6 to 16 that did not show implanted embryos were not included in the means (see text). Day 1 was the day after parturition. The number of cases in each group was 7–10. After implantation occurred, the levels were significantly increased in C+ females as compared with their C− controls (C+/L+ and C+/L− groups vs. C−/L+ and C−/L− groups; $p < 0.05$, ANOVA II, Tukey test). There were also significant interactions by days and groups (on Day 11, between both C− groups and the C+/L− group, and on Day 16, between both C− groups and both C+ groups, same test).

TABLE 2. Decidualization and plasma progesterone after either endometrial scratching (D+) or sham operations (D−), in females that had either copulated (V+) or not (V−) with vasectomized males.*

| Group | Number of cases | Females with decidual reactions (%) | Uterine weight (mg; mean ± SEM) | Plasma progesterone (nM; mean ± SEM) |
|---|---|---|---|---|
| V+/D+ | 10 | 90 | 288.5 ± 51.4 | 61.1 ± 4.8 |
| V+/D− | 8 | 0 | 84.9 ± 9.4 | 17.2 ± 3.9 |
| V−/D+ | 10 | 100 | 330.1 ± 30.3 | 65.9 ± 4.7 |
| V−/D− | 10 | 0 | 81.5 ± 11.5 | 13.4 ± 1.3 |

* Animals were killed and plasma was sampled on Day 8 (Day 1 = day after parturition).

elevation on Day 3 occurred in all groups. After implantation occurred, progesterone levels in groups of C+ females that bore implanted embryos were higher than in C− females (significant interaction by groups, $p < 0.05$, between both C+ groups and both C− groups; ANOVA II, Tukey test). There were also significant interactions by days and groups (on Day 11, between both C− groups and the C+/L− group, and on Day 16, between both C− groups and both C+ groups, same test).

Progesterone levels in those C+ females that were killed from Days 6 to 16, and that did not show implanted embryos, were not included in the mean values presented in Figure 3. These females tended to have progesterone levels that were lower than those of pregnant females in the same group and were within the range (7.9–79.2 nM) for nonpregnant females that had not copulated: on Day 6, five C+/L+ females that bore no implantation sites had progesterone levels of 33.4 ± 6.7 nM (range: 13.2–50.1 nM), and three C+/L+ females on Days 11–16 had progesterone values that ranged from 6.4 to 24.2 nM.

### *Experiment II*

Decidualization (as confirmed histologically on Day 8) was induced by endometrial scratching in 90% of females that had copulated during postpartum estrus and in 100% of females that had not copulated (Table 2). Sham operations were ineffective, whether the females had copulated or not. Mean uterine weight on Day 8 was significantly higher in both D+ groups as compared with both D− groups ($p < 0.05$, ANOVA I, Tukey test), even though the mean of the V+/D+ group in Table 2 includes the only nondecidualized uterus that weighed 59 mg.

Plasma progesterone levels in these animals are shown in Table 2. They were also significantly higher in the D+ than in the D− groups ($p < 0.05$, ANOVA I, Tukey test) whether the females had copulated or not. The only V+/D+ female that did not show a decidual reaction had 61.1 nM of plasma progesterone, a value that was equal to the mean of the group.

## DISCUSSION

Embryo implantation occurred between Days 3 and 6 in females that had copulated on postpartum estrus. Although our experimental design precluded a more precise definition of the time of implantation, it is clear that it occurred much earlier than in the laboratory rat, since large implantation nodules were found on Day 6 (in the rat, blastocysts are merely attached to the endometrium on Day 6 of pregnancy in nonlactating females, and similar nodules can be found only 2–3 days later [31]). An early time of implantation in corn mice, as compared with the rat, is consistent with previously reported findings [25].

The percentage of mated females that did not show implantation sites on postpartum Day 6 was greater in the group that was also lactating; this may suggest a lactational delay of implantation. However, this possibility could not be demonstrated, since the difference was not statistically significant. Nevertheless, the occurrence of a lactational delay of implantation has been reported in another *Calomys* species [32].

The pattern of prolactin secretion during lactation that was observed in female corn mice is consistent with that observed in the rat and other species, in which prolactin secretion is stimulated by suckling at the beginning and in the middle of lactation and in which it declines thereafter (e.g., [15, 33]). The rather large standard errors found in lactating female corn mice in the present study are also a common observation in rats that remain continuously with their pups (e.g., [34]) and reflect spike-like variations in the rate of prolactin secretion in such conditions. However, contrary to what one would have expected from studies in other murid rodents (e.g., [11, 14, 35]), mated female corn mice did not show higher prolactin levels when compared with their unmated controls even though blood samples in this study were obtained at the time when the diurnal surge of prolactin occurs in those species.

The rise in plasma progesterone levels that occurred in all groups on postpartum Day 3 (i.e., after postpartum ovulation) is similar to that observed after ovulation in virgin females of this species [36], and it may reflect the "autonomous" progesterone secretion of the short luteal phase [37]. These autonomous levels did not differ whether the females had copulated or not during postpartum estrus or whether they were or were not lactating. Also, these levels, as well as those observed later during gestation, are definitely low as compared with those of other murid [37, 38] and nonmurid species [39], whose maximum levels during gestation are above 350 nM.

Plasma progesterone declined from Days 3 to 16 in those females that had not copulated. Unlike what it is known in other murid species (e.g. [16, 33]), this decline also occurred in lactating females, which indicates that the elevated prolactin levels found in lactating females do not have a luteotrophic effect in female corn mice. In other words, there seems to be no lactational pseudopregnancy in this species.

The sustained increase in progesterone secretion observed (Days 6, 11, and 16) in females that had copulated seems to be associated with the presence of implanted embryos and placentae on those days, and not with a neuroendocrine response to copulatory stimulation. The low prolactin levels of C+/L− females indicate that copulation-induced prolactin release does not occur at a time when the diurnal surge of prolactin is expected to occur in other murid species ([11, 14, 35]; Fig. 2). Also, both the ability of females that had not copulated to sustain decidual growth and the elevated progesterone levels found on Day 8 in

these nonmated, deciduoma-bearing females indicate that luteal activation in this species is not a neuroendocrine response to the mechanical stimulation of copulation. It should be established in the future whether the placentae themselves (the decidua? the trophoblast?) are secreting progesterone at these stages or whether instead they are producing a trophic hormone that stimulates progesterone secretion by the corpora lutea; evidence in the laboratory rat (e.g., [40]) suggests the likelihood of the latter possibility.

In a previous paper [36] we provisionally classified the ovarian cycle of the corn mouse as the type III of Conaway [1]. This was based on the occurrence of spontaneous ovulation and of a short luteal phase in this species. In other species that have been studied [1], these short luteal phases are nonfunctional in the sense that embryo implantation cannot occur during them. Nevertheless, the current findings indicate that the short luteal phase of the corn mouse is functional (i.e., decidual growth and embryo implantation can occur within it); they suggest that it may have the adaptive value of increasing the probability of a female's becoming pregnant and, in this sense, may be an adaptation to high predation pressure. However, the maintenance of progesterone secretion beyond Day 3 in the corn mouse is dependent upon embryo implantation or the presence of a conceptus, not on copulatory stimulation, prolactin, or suckling stimulation as in type III species (e.g., the laboratory rat and mouse). Moreover, it seems also that the role of prolactin, as seen in the ovarian cycle of the laboratory rat [14], is not necessarily representative of other murid rodents, even those such as the corn mouse, which exhibit a short-lived corpus luteum following spontaneous ovulation.

It would be interesting to know whether or not conditions similar to those of the corn mouse are restricted to the Neotropical division of the subfamily Sigmodontinae (sensu [2]), since this may give insight into the problem of the North/South dichotomy within this subfamily [18, 41, 42]. There is evidence [6, 13, 43] indicating that the Nearctic genus *Peromyscus* bears a type III cycle, i.e., similar to that found in most Old World Muridae, including the laboratory rat.

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## REFERENCES

1. Conaway CH. Ecological adaptation and mammalian reproduction. Biol Reprod 1971; 4:239–247.
2. Musser GG, Carleton MD. Family Muridae. In: Wilson DE, Reeder DM (eds.), Mammal Species of the World—A Taxonomic and Geographic Reference. Washington DC: Smithsonian Institution Press and the American Society of Mammalogists; 1993: 501–755.
3. Castro-Vázquez A, Luque EH, Carreño NB. The pseudopregnancy response: control by sensory inputs, ovarian steroids and luteinizing hormone-releasing hormone. In: Endröczi E (ed.), Neuropeptides, Neurotransmitters and Regulation of Endocrine Processes. Budapest: Akadémiai Kiadó; 1983: 349–360.
4. Gunnet JW, Freeman ME. The mating-induced release of prolactin: a unique neuroendocrine response. Endocr Rev 1983; 4:44–61.
5. Diamond M. Intromission pattern and species vaginal code in relation to induction of pseudopregnancy. Science 1970; 169:995–997.
6. Kenney AM, Lanier DQ, Dewsbury DA. Effects of vaginal-cervical stimulation in seven species of muroid rodents. J Reprod Fertil 1977; 49:305–309.
7. Milligan SR. Mating, ovulation and corpus luteum function in the vole, *Microtus agrestis*. J Reprod Fertil 1975; 42:35–44.
8. Dewsbury DA, Estep DQ, Oglesby JM. Copulatory behavior and the initiation of pregnancy in Israeli gerbils (*Meriones tristrami*). Biol Behav 1978; 3:243–257.
9. McLean BK, Nikitovitch-Winer MB. Effects of pituitary transplantation and hypothalamic lesions on function in the Mongolian gerbil. Neuroendocrinology 1974; 15:120–125.
10. Diamond M, Yanagimachi R. Induction of pseudopregnancy in the golden hamster. J Reprod Fertil 1968; 17:165–168.
11. Erb GE, Wynne-Edwards KE. Prolactin, follicle-stimulating hormone, and luteinizing hormone levels during preimplantation in the Djungarian hamster (*Phodopus campbelli*). Biol Reprod 1994; 50:1328–1333.
12. Erb GE, Wynne-Edwards KE. Preimplantation endocrinology in the Djungarian hamster (*Phodopus campbelli*): progesterone, estrogen, corpora lutea, and embryonic development. Biol Reprod 1993; 49: 822–830.
13. Dewsbury DA, Estep DQ. Pregnancy in cactus mice: effects of prolonged copulation. Science 1974; 187:552–553.
14. Smith MS, Freeman ME, Neill JD. The control of progesterone secretion during the estrous cycle and early pseudopregnancy in the rat: prolactin, gonadotropin and steroid levels associated with the rescue of the corpus luteum of pseudopregnancy. Endocrinology 1975; 96: 219–226.
15. Amenomori Y, Chen CL, Meites J. Serum prolactin levels in rats during different reproductive states. Endocrinology 1970; 86:506–510.
16. Pepe GJ, Rothchild I. A comparative study of serum progesterone levels in pregnancy and in various types of pseudopregnancy in the rat. Endocrinology 1974; 95:275–279.
17. Reig OA. Distribuiçao geográfica e história evolutiva dos roedores muroideos sudamericanos (Cricetidae; Sigmodontinae). Rev Bras Genet 1984; 2:333–365. (English abstract).
18. Smith M, Patton JL. The diversification of South American murid rodents: evidence for mitochondrial DNA sequence data for the akodontine tribe. Biol J Linn Soc 1993; 50:149–177.
19. Castro-Vázquez A, Carreño NB, Cutrera RA, Martínez AR, Koninckx A. The corn mouse (*Calomys musculinus*) as an experimental model for reproductive studies. In: Rothschild HA, Rosenkranz A, Moura Duarte FA (eds.), Laboratory Animal Studies in the Quest for Health and Knowledge. São Paulo: Sociedade Brasileira de Genética; 1987: 274–282.
20. Weissenbacher M, Damonte EB. Fiebre hemorragica argentina. Adel Microbiol Enferm Infecc 1983; 2:119–171.
21. Basso B, Eraso A, Moretti E, Albesa I, Kravetz FO. Infección natural de *Calomys musculinus* (Rodentia; Cricetidae) por *Trypanosoma cruzi*. Rev Asoc Argent Microbiol 1977; 7:13–16.
22. Massoia E, Fornés A, Wainberg RL, de Fronza T. Nuevos aportes al conocimiento de las especies bonaerenses del género *Calomys* (Rodentia-Cricetidae). Rev Invest Agropecu INTA Ser 1 Biol Reprod Anim 1968; 5:63–92. (English abstract).
23. Cutrera RA, Yunes RMF, Castro-Vázquez A. Postpartum sexual behavior of the corn mouse (*Calomys musculinus*): repertoire, measurements and effects of removal of pups. J Comp Psychol 1988; 102:83–89.
24. De Feo VJ. Temporal aspects of uterine sensitivity in the pseudopregnant or pregnant rat. Endocrinology 1963; 72:305–316.
25. Montoro LS, Quintans CJ, Tancredi AM, Donaldson MJ, Hodara V. *Calomys musculinus* (Rodentia; Cricetidae): its reproduction in captivity. In: Rothschild HA, Rosenkranz A, Moura Duarte FA (eds.), Laboratory Animal Studies in the Quest for Health and Knowledge. São Paulo: Sociedade Brasileira de Genética; 1987: 303–308.
26. Laemmli UK. Cleavage of structural proteins during the assembly of the head of bacteriophage T4. Nature (Lond) 1970; 227:680–685.
27. Debeljuk L, Rozados R, Daskal H, Velez CV, Mancini AM. Acute and chronic effects of sulpiride on serum prolactin and gonadotropin levels in castrated male rats. Proc Soc Exp Biol Med 1975; 148:550–552.
28. Zar JH. Biostatistical Analysis (2nd edition). New Jersey: Prentice-Hall International, Inc.; 1984.
29. Castro-Vázquez A, Carreño NB. A study of the sensory requirements for eliciting the pseudopregnancy response. Brain Res 1981; 230:205–220.

30. Motulsky H. Intuitive Biostatistics. New York: Oxford University Press; 1995.
31. Lobel BL, Levy E, Shelesnyak MC. Studies on the mechanism of nidation. XXXIV. Dynamics of cellular interactions during progestation and implantation in the rat. Acta Endocrinol (Kbh) 1967; 56(suppl 123):1–109.
32. Hodara V, Espinosa MB, Merani MS, Quintans C. *Calomys laucha* (Rodentia, Cricetidae): growth and breeding in laboratory conditions. Lab Anim 1989; 23:340–344.
33. Roy BN, Wynne-Edwards KE. Progesterone, estradiol, and prolactin involvement in lactation, including lactation following a postpartum mating, in the Djungarian hamster (*Phodopus campbelli*). Biol Reprod 1995; 52:855–863.
34. Lu KH, Chen HT, Huang HH, Grandison L, Marshall S, Meites J. Relation between prolactin and gonadotrophin secretion in post-partum lactating rats. J Endocrinol 1976; 68:241–250.
35. Barkley MS, Bradford GE, Geschwind II. The pattern of prolactin concentration during the first half of mouse gestation. Biol Reprod 1978; 19:291–296.
36. Cutrera RA, Carreño NB, Castro-Vazquez A. Correlative genital tract morphology and plasma progesterone levels during the ovarian cycle in female corn mice. Z Säugetierkunde 1992; 57:14–22.
37. Rothchild I. The regulation of the mammalian corpus luteum. Recent Prog Horm Res 1981; 37:183–298.
38. Barkley MS, Geschwind II, Bradford GE. The gestational pattern of estradiol, testosterone and progesterone secretion in selected strains of mice. Biol Reprod 1979; 20:733–738.
39. Challis JRG, Heap RB, Illingworth DV. Concentrations of oestrogen and progesterone in the plasma of nonpregnant, pregnant and lactating guinea-pigs. J Endocrinol 1971; 51:333–345.
40. Gibori G. The corpus luteum of pregnancy. In: Adashi EY, Leung PCK (eds.), The Ovary. New York: Raven Press; 1993: 261–317.
41. Hooper ET, Musser GG. The glans penis in Neotropical cricetines (family Muridae), with comments on the classification of muroid rodents. Misc Publ Mus Zool Univ Mich 1964; 123:1–57.
42. Carleton MD. Phylogenetic relationships in neotomine-peromyscine rodents (Muroidea) and a reappraisal of the dichomoty within New World Cricetinae. Misc Publ Mus Zool Univ Mich 1980; 157:1–146.
43. Dewsbury DA. Copulatory behavior of deer mice (*Peromyscus maniculatus*): III. Effects on pregnancy initiation. J Comp Physiol Psychol 1979; 93:178–188.