Occurrence of subnormal corpus luteum in superovulated Santa Inês sheep using protocols with or without LH administrated at the end of the FSH treatment

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Introduction

Corpora lutea (CL) that are small (<5mm), grossly pale, with little or no protrusion from the surface of the ovary are defined as subnormal and are associated to low circulating progesterone concentrations. The formation of a subnormal or short-lived CL has been widely reported in superovulated sheep (1). Two causes of subnormal CL formation have been proposed: i) inadequate follicle development preceding luteal gland formation (2), and ii) premature activation of the luteolytic mechanism (3). Based on the latter possibility in practice, luteolytic inhibitors such as Flunixine Meglumine are used. Thus, the objective of the present study was to evaluate the occurrence of subnormal CL in superovulated Santa Inês sheep using protocols with or without LH administrated at the end of the FSH treatment.

Materials and Methods

Twenty multiple ovulations were accomplished and the experiment was designed as a 2 by 2 factorial, with the main effects of replicate and treatment. The estrus was synchronized with a progesterone-releasing intravaginal device (CIDR™; Pfizer-New Zealand) inserted on Day 0, replaced by a new one that was maintained from Day 7 to Day 14. Two doses of the 37.5 µg of D-cloprostenol (Prolise™, Arsa - Argentina) were administered, IM, on Day 7 and 14. Then, 256 mg of FSHp (Folltropin™, Bioniche-Canada) were administrated in 8 decreasing doses, starting on Day 12. On Day 14, all females received 200 IU of eCG (Novormon™, Syntex-Argentina). On Day 15, the animals were homogeneously allocated in one of the two experimental groups: Control (GC) and treated (G-LH). Sheep in GC did not receive exogenous LH, while sheep in G-LH were treated with 7.5 mg of LH (Lutropin™, Bioniche-Canada), 24 h after device withdrawal (Day 15). On Day 16, the number of ovulatory follicles was verified by laparoscopy. On Day 17, 18 and 19, all females received 75 mg of Flunixine meglumine (Banamine™, Shering-Plough, Brazil). On Day 21, the ovarian structures were evaluated by laparoscopy. Number of normal CL, subnormal CL and anovulatory follicles were measured. Two-way analysis was run for the effects of replicate (2 replicates) and groups (GC and G-LH). Data were analyzed using by ANOVA using procedure GLM of SAS, and means (±SD) were compared using Kruskal-Wallis test ($P < 0.05$).

Results and Discussion

Ovulation rate tended to be increased in G-LH (85.44% *vs* 77.77%, $P = 0.08$). The number of CL (mean ± SD) was 10.5 ± 3.8 in GC and 13.5 ± 4.84 in G-LH; $P > 0.1$. There was not effect of replicate evaluating the number of CL in GC (12 ± 3.46 vs 9 ± 3.87); whereas in G-LH there was a negative effect (16.6 ± 2.97 vs 10.4 ± 4.45). The number of anovulatory follicles did not differ statistically between groups (GC: 3.0 ± 3.19; G-LH: 2.3 ± 1.63). Likewise, no effect of repeatability was shown for this variable. The anovulatory rate tended to decrease in G-LH (22.22% *vs* 14.55%, $P = 0.08$). Even using Flunixine Meglunine to prevent luteolysis, 20% of the ewes treated with GC and 40% of the ewes in G-LH presented at least one subnormal CL ($P > 0.05$). The numbers of subnormal CL were 3.0 ± 1.41 for GC and 1.25 ± 0.5 for G-LH ($P > 0.1$). Failure in formation of CLs was not punctual characteristic because it was found that a single female had normal and subnormal CL. These data does not corroborate with data published by Rubianes et al. (1996). The subnormal CLs were observed just in second replicate of each female. This fact suggests that the repeatability of treatment could have interfered in formation of CL, however, statistical difference was observed just for number of subnormal CL in G-LH. The greater number of subnormal CL in G-LH in the second reply might have been caused by the administration of LH, which can induce the ovulation of a follicle without appropriate capacity of formation of a CL. Indeed, a previous study showed that GnRH administration when follicles are not completely maturated induce the formation of CL without adequate function or short lived (4). The results showed that the formation of a subnormal or a short-lived CL can be more related to the incapacity of the ovulated follicle to form a normal CL than to the premature activation of luteolytic mechanism.

References

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