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Effect of hormonal environment at emergence on persistence of ovarian follicles in cattle

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INTRODUCTION

Reduced fertility often occurs when progestins are used to control the timing of oestrus in cattle and is especially evident when a corpus luteum (CL) is not present in the ovary (Kinder *et al.*, 1996). The lowered conception rates at the synchronised oestrus are associated with the development of abnormally large, "persistent" ovarian follicles as a result of increased secretion of LH. An understanding of the endocrine regulation of persistent follicles is essential for development of methods to prevent ovulation of this aberrant structure in progestin-based oestrous control systems. The influence of time of luteal regression relative to follicle emergence on persistence of dominant follicles was evaluated in progestin-treated cows.

MATERIALS AND METHODS

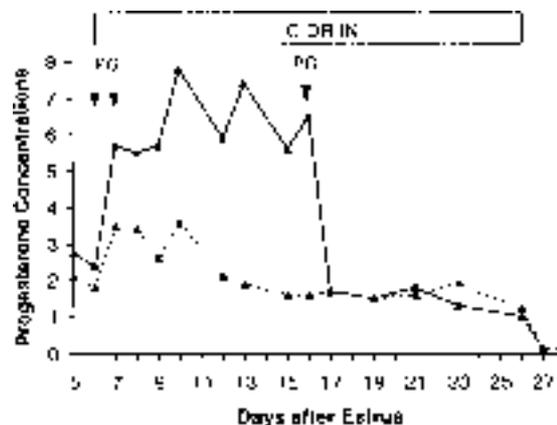
Oestrus was synchronised in 10 nonlactating Friesian cows (day of oestrus = Day 0). Each cow received a CIDR-B® (InterAg, NZ) device from Day 6 to 26 and also received prostaglandin F_{2α} on either Days 6 and 7 (d6PG; n=5), or Day 16 (d16PG; n=5). Ovaries were examined by ultrasonography, and blood samples collected via the coccygeal vein on a daily, or alternate day basis from Days 5 to 30 to monitor follicular growth, luteal regression, ovulation and plasma concentrations of progesterone. Cows were observed for oestrus from Days 27 to 30. Maximum diameter of the first-wave (DF1) and second-wave dominant follicles (DF2) and lifespan of DF1 (defined by the day of emergence of the second-wave dominant follicle) and DF2 (defined as number of days from emergence of DF2 to emergence of DF3 or oestrus) were compared between treatments by one way analyses of variance. Patterns of growth of DF1 and DF2, and concentrations of progesterone over time were compared among treatments using split-plot analyses of variance.

RESULTS

Concentrations of progesterone were similar among treatments on Days 5, 6 and 17 to 28 (Fig. 1).

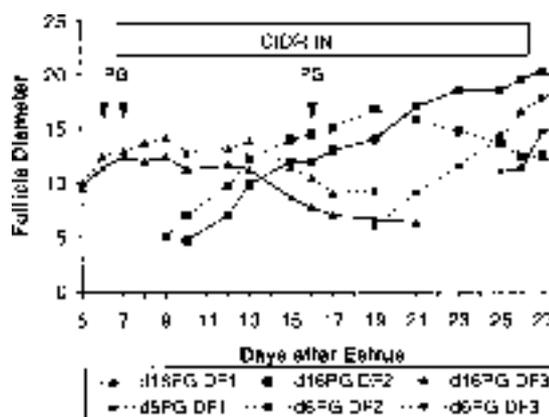
Concentrations of progesterone were greater ($P < .05$) in the d16PG than the d6PG treatment from Days 7 to 16 (treatment x day; $P < .05$). Lifespan of DF1 ($10.4 \pm .9$ and $10.8 \pm .5$, respectively) did not differ among the d6PG and d16PG treatments (Fig. 2). The maximum diameter of DF1 tended to be greater ($P = .06$) for the d6PG treatment. Pattern of growth of DF2 varied between treatments (treatment x Day; $P < .05$). In the d6PG treatment, DF2 grew more rapidly to Day 19 ($P < .05$) than in the d16PG treatment, but was replaced on day $20.5 \pm .96$ by a third-wave

FIGURE 1: Concentrations of progesterone (ng/ml).



dominant follicle (DF3). The DF3 ovulated in every d6PG cow on Day 29. In contrast, DF2 in the d16PG persisted for a longer time ($P < .05$) than in the d6PG treatment (14.4 ± 1.4 vs. $10.5 \pm .3$ days, respectively). In 3/5 cows in the d16PG treatment, DF2 was replaced by DF3 on day 23.3 ± 2.2 . In the remaining 2 cows, DF2 persisted to the day of oestrus (Days 28 or 29) and either ovulated or continued to persist following oestrus.

FIGURE 2: Diameter of dominant follicles (mm).



DISCUSSION

In cows administered CIDR devices and PG on day 6 of the oestrous cycle, the DF1 did not have an extended lifespan. Persistent follicles have been observed to develop in females treated with this protocol and a greater incidence of persistent follicles occurred in lactating cows than in yearling heifers in that study (Inskeep *et al.*, 1996). In the

d6PG treatment, the DF2 emerged in the absence of the CL, grew rapidly, and had a lifespan of 10.5 days. In contrast, if the CL was present for the first 5 days following emergence of DF2 (d16PG treatment) and then regressed with PG, the DF2 persisted for an additional 10 days giving a total lifespan of approximately 14.5 days. Regardless of treatment, it appeared that follicles became atretic after exposure to low progesterone concentrations

for 10 days. It is suggested that the lifespan of follicles exposed to a period of low progesterone is determined, in part, by the hormonal environment at emergence.

REFERENCES

- Kinder *et al.* (1996) *Journal of Animal Science* **74**:1424-1441.
Inskeep *et al.* (1996) *Journal of Animal Science* **74**:1943-1952.