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Calvin L. Ferrell and Thomas G. Jenkins[1]

Introduction

The mechanisms that control follicular development, oocyte maturation and ovulation, are complex and poorly understood in farm animals. Superovulation via gonadotrophin stimulation of the ovaries provides a model to study follicular development and ovulation and the endocrine interactions at the follicle level. This study focused on the importance of luteinizing hormone (LH) in follicular development, hormonal secretion, and ovulation. The objectives of this study were to describe differences in follicular development, hormonal secretion, and oocyte quality in superovulated heifers that exhibited a normal LH surge, no LH surge, and in which the LH surge was suppressed with a progestin implant.

Materials and Methods

Crossbred heifers (n = 137) were synchronized to estrus with prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) and superovulated with follicle stimulating hormone (FSH-P). Animals were divided into three treatment groups to consist of 1) animals that exhibited an LH surge (n = 86), 2) animals that had no LH surge (n = 23), and 3) animals in which the LH surge was suppressed with a progestin implant (Norgestomet, n = 28) inserted in the ear 12 hr prior to the initial prostaglandin injection. Animals were ovariectomized every 12 hr after the prostaglandin injection (n = 7-9/time, 12-108 hr post $PGF_{2\alpha}$). Animals implanted with progestin were ovariectomized at 72, 84, 96, and 108 hr post $PGF_{2\alpha}$. Post ovulatory follicular changes involving atresia were monitored by ovariectomizing animals at 192 and 240 hr post $PGF_{2\alpha}$ (n = 34). Follicular fluid was collected after follicles were measured for size. Oocytes were centrifuged from the fluid and evaluated for viability.

Results

In the heifers that exhibited an LH surge, follicular progesterone and estradiol were increased (Figs. 1 and 2; $p<.05$), particularly at the time of the LH surge (x = 45 hr). Follicular fluid glycosaminoglycans (GAG) were increased in animals not exhibiting an LH surge, primarily in the small- and medium-size follicles (Fig. 3). Follicular progesterone and estradiol concentrations increased with follicular size whereas glycosaminoglycans decreased in concentration as follicles increased in size (Figs. 1-3). Follicular progesterone concentrations were increased in animals that did not show an LH surge as compared to the treatment group that had the LH surge inhibited with progestin implants. Follicular estradiol and glycosaminoglycan concentrations were similar in the no LH surge group and the progestin-implanted group. Oocyte recovery was 77%. Oocyte quality was poorest in small-size follicles and best in the large-size follicles (Table 1). The LH surge treatment group had the highest quality of oocytes whereas the progestin-implanted animals had the poorest quality oocytes (22% viable). Oocyte quality from follicles into the next cycle (day 4, 6) was very low (16-30% viable) and presumably indicative of aspects of follicular atresia. Estrogen and progesterone concentrations remained low in these follicles but glycosaminoglycan concentrations increased, also indicative of atresia.

Discussion

Increases in progesterone alter the release of pituitary LH and subsequently inhibit both the steroidogenic function and ovulation as indicated in the progestin-implanted animals. Also, animals in which no LH surge was detected due to handling and blood sampling during the experiment (21%) had altered steroidogenesis (Figs. 1-2), no ovulation, and decreased oocyte viability (Table 1), but peripheral circulating concentrations of progesterone were not different from those in animals that exhibited an LH surge and later ovulation. The LH stimulation during the estrual period is also important for oocyte maturation. In animals in which the LH surge was suppressed (progestin implanted), oocyte quality was low. Animals in which no LH surge was detected had intermediate viability of oocytes, and thus probably received some LH stimulation but insufficient for complete development and ovulation. Glycosaminoglycans can be used as a biochemical marker for atresia of follicles, but also high concentrations of follicular glycosaminoglycans are related to low *in vitro* fertilization rates. High concentrations of glycosaminoglycans noted particularly in small-size follicles were related to quality (viability) of oocytes in all treatments. Further studies on the ovulatory events that related to follicular steroidogenesis, oocyte development and maturation, and ovulation will define the critical events associated with follicular development and help refine techniques that produce the maximum number of quality oocytes/embryos.

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Table 1—Percentage of viable oocytes

Treatment	Hours post prostaglandin injection										
	12	24	36	48	60	72	84	96	108	192 (4)[a]	240 (6)
<u>≤ 5 mm diameter (n = 716)</u>											
LH surge	75.5±11.6[b]	39.9±8.6	59.2±7.8	67.9±15.8	28.5± 9.7	29.1±12.3	42.1±12.0	66.3±11.2	77.9±20.2	35.0± 8.4	1.0± 3.3
No LH surge				85.7± 4.1	52.4±18.2	90.7±16.9[c]	7.3± 9.0c	31.5±18.7[c]	37.9±12.9[c]		
Norgestomet implanted						54.4± 5.8[c,d]	37.6± 5.7d	21.4± 5.6[c]	24.2± 6.3[c]		0.0
<u>5-8 mm diameter (n = 441)</u>											
LH surge	54.9± 6.6	65.7±8.2	29.3±8.7	67.8±12.5	84.6± 9.8	97.2±10.2	80.9±12.1	70.9±13.8	17.4±19.6	24.0±12.6	23.3± 9.1
No LH				28.6±16.6[c]	57.6±16.2	68.0±14.5	63.5±14.4[b]	69.8±18.4	65.7±11.9		30.0±24.3
Norgestomet implanted						36.7± 6.7[c,d]	29.2± 7.0[c,d]	22.5± 7.2[c,d]	20.8± 6.0[c,d]		
<u>> 8 mm diameter (n = 528)</u>											
LH surge	53.6± 7.1	57.7±5.5	82.6±8.3	67.8± 9.7	66.6± 5.4	96.6±13.8	98.7±20.3	70.5±22.9	17.4±38.0	5.6±29.0	11.3± 9.4
No LH				68.5±10.8	76.6± 9.7	72.2±14.3	22.9±13.3[c]	56.4± 9.0	18.5±26.0		100.0
Norgestomet implanted						16.7± 7.5[c,d]	19.3± 5.6[c]	19.7± 7.8[c,d]	19.5± 6.4		

[a] Day of subsequent estrous cycle ( ).

[b] Time 12-36 hr same for LH and no LH surge groups.

[c] LH vs no LH or Norgestomet implanted, p<.05.

[d] No LH vs Norgestomet implanted, p<.05.

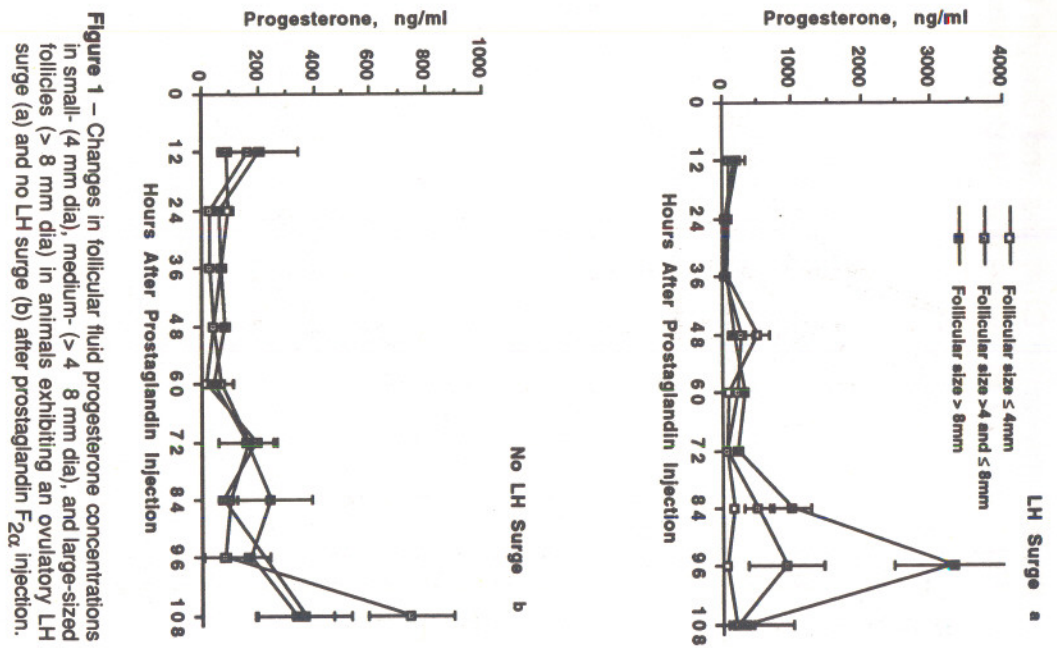

Figure 1 – Changes in follicular fluid progesterone concentrations in small- (4 mm dia), medium- (> 4 8 mm dia), and large-sized follicles (> 8 mm dia) in animals exhibiting an ovulatory LH surge (a) and no LH surge (b) after prostaglandin $F_{2\alpha}$ injection.

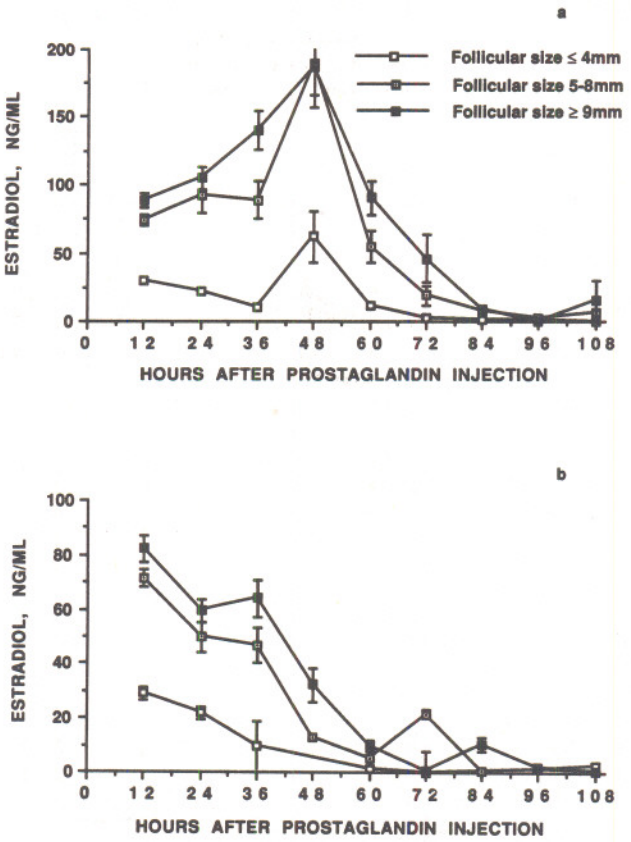

Figure 2 – Changes in follicular fluid estradiol concentrations in small- (4 mm dia), medium- (> 4 8 mm dia), and large-sized follicles (> 8 mm dia) in animals exhibiting an ovulatory LH surge (a) and no LH surge (b) after prostaglandin $F_{2\alpha}$ injection.

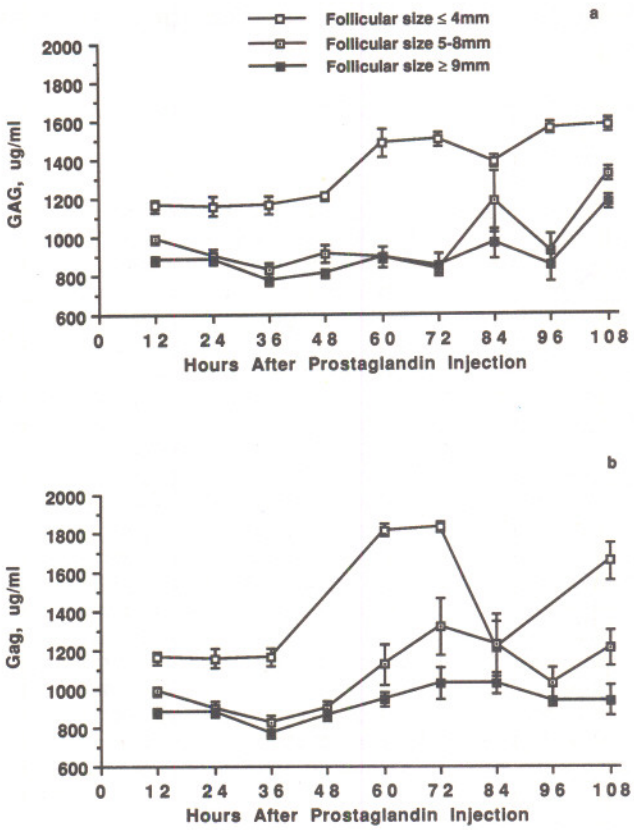

Figure 3 – Changes in follicular fluid glycosaminoglycans (GAG) concentrations in small- (4 mm dia), medium- (> 4 8 mm dia), and large-sized follicles (> 8 mm dia) in animals exhibiting an ovulatory LH surge (a) and no LH surge (b) after prostaglandin $F_{2\alpha}$ injection.