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Introduction

As the female cow matures, the majority of her follicles become atretic and are lost as a store house of the female gamete (oocyte). Emphasis on *in vitro* fertilization to maximize the utilization of superior animals for transplant of frozen embryos is considerably limited by the number of viable mature oocytes that can be collected. Understanding the biochemical environment required to produce maximum numbers of mature, fertilizable oocytes is a prime requirement to utilize this technology in increasing meat animal production and efficiency.

The goal of these studies was to characterize the endocrine and biochemical events associated with follicle and oocyte maturation to establish the best environment for follicle development and maximize numbers.

Procedure

Experiment 1 involved 60 crossbred heifers assigned to control (n = 30) or superovulatory regimen (n = 30) via follicle stimulating hormone (FSH-P). One-third of the animals from each group were ovariectomized 24, 48, and 72 hr after prostaglandin F2α synchronization (PGF2α). Follicular fluid was collected from individual follicles (n = 76, controls; n = 551, FSH = stimulated) and analyzed for progesterone, estradiol, prostaglandin E, prostaglandin F, oxytocin, sodium, and potassium content.

Experiment 2 involved 103 crossbred heifers which were estrous synchronized (PGF2α) and superovulated with FSH. Animals were ovariectomized every 12 hr after PGF2α injection, and follicles were harvested. Twenty-eight animals were implanted with Norgestomet implants 12 hr before the PGF injection, then ovariectomized 72, 84, 96, and 108 hr after PGF2α. Oocytes were evaluated from 2,470 follicles, classified as degenerate or viable, and compared to follicular endocrine parameters (progesterone, prolactin).

Results

Experiment 1. Of the follicular parameters monitored, no differences were noted between follicles developed under normal circumstances or with FSH stimulation to induce multiple follicles. By 72 hr after PGF2α administration, follicular estradiol was decreasing in concentration, and progesterone and oxytocin were increasing, thus signifying a change in the secretory role of the granulosa cells. At 48 hr after the PGF2α injection, sodium decreased and potassium increased, thus signifying a considerable physiological change in the follicle. Prostaglandin E and F increased ten-fold by 72 hr, and increased concentrations were generally found in estrogen-inactive follicles at 72 hr.

Experiment 2. Since 65% of the animals exhibited normal LH surge, the data was divided into three groups (animals exhibiting an LH surge, animals not exhibiting an LH surge, and animals in which the LH surge was suppressed with Norgestomet implants). Follicular fluid prolactin concentrations were similar in all treatment groups in that, from 12 to 48 hr after PGF2α, prolactin increased, then steeply decreased. Follicular progesterone concentrations in large- and medium-sized follicles increased after the LH surge. Animals in which no LH surge was detected or suppressed with Norgestomet implants had follicular progesterone concentrations that remained low in all follicular sizes.

Oocyte recovery was 77% from 2,470 follicles. Oocyte quality increased from 60 to 70% up to the LH surge and remained at 60% for the rest of the time analyzed (60-108 hr). Oocyte quality was considerably better (80%) in large-sized follicles (> 9 mm dia). In Norgestomet-implanted animals, oocyte quality was 36% good at 72 hr after PGF2α and 19% good at 108 hr. Prolactin concentrations in follicular fluid increased up to the period of the LH surge, then sharply declined. For animals in which no LH surge was detected, follicular fluid prolactin changes were similar to those noted in normally ovulating animals. Prolactin concentrations were increased in follicles producing high quality oocytes (before and during the LH surge). After the LH surge, prolactin decreased in all follicles, and, as an index of oocyte quality, is questionable.

Increased concentrations of progesterone and prolactin in human follicular fluid have been reported to be related to oocyte maturity and successful *in vitro* fertilization. Analysis of bovine oocytes and follicular fluid prolactin concentrations support the human data, but no relationships were detected between follicular fluid progesterone concentrations and oocyte quality. The characterization of the endocrine and biochemical events associated with follicle and oocyte maturation will eventually lead to the correct stimulating regimes to maximize oocyte quality and numbers.

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