ABSTRACTS

6th International Symposium on Canine and Feline Reproduction &

6th Biennial EVSSAR Congress

European Veterinary Society for Small Animal Reproduction

"Reproductive biology and medicine of domestic and exotic carnivores"

University of Veterinary Sciences
9th – 11th July 2008
Vienna, Austria

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ENDOCRINOLOGICAL CONTROL OF CANINE OVARIAN FUNCTION

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This review addresses the following conclusions and questions. The termination of anestrus, onset of proestrus, and recruitment of a dominant cohort of follicles appear due to a pre-proestrus increase in LH pulse rate, caused by increased GnRH pulsatility; modest concomitant increases in FSH may participate but appear not to be required. Unclear is the role or bioactivity of relatively elevated concentrations of immuno-reactive FSH throughout much of anestrus, the timing of any increased FSH-dependent aromatase activity, and the extent to which pre-proestrus and early proestrus increases in estradiol secretion are the result of increased aromatase synthesis and/or activity, or to LH-stimulated synthesis of precursor androgen for already available aromatase.

The mid and late proestrus follicular phase may be autonomous or semi-autonomous, with intra-follicular estradiol being folliculo-trophic and with the fate of dominant follicles being predetermined as either atresia or ovulation as the endogenous capacity to further increase estradiol secretion becomes limited. Nevertheless, GnRH agonist induced proestrus rarely proceeds to full estrus if agonist is discontinued before 8 days. Increasing intra-follicular estradiol likely causes decreases in the ER:PR ratio in follicle cells, as suggested in other species as well.

Both the LH surge and sex behavior appear to be stimulated to occur after the peak in dominant follicle development by a spontaneous decline in the E:P ratio; the further LH surge-promoted decline in E:P ratio in conjunction with any decease in ER:PR ratio also participates in the signals for initiation of oocyte maturation and changes in the pathways leading to the increased intra-follicle PG production required for ovulation. Unclear is the extent to which in vitro and in vivo studies can confirm that intra-follicular changes in dogs are similar to or the same as those reported for the development and ovulation of dominant follicles in other species; that gonadotrophin surges regardless of whether predominantly LH or FSH in activity can elicit ovulation; that ovulation has the potential to occur spontaneously at the end of the follicular phase, although almost always facilitated or precipitated by a ovarian-signalized, properly timed gonadotrophin surge.

Luteal function in bitches requires LH as a luteotrophic hormone throughout the luteal phase and is negatively impacted by GnRH antagonist treatments, or by persistent and LH-down-regulating GnRH-agonist treatment; prolactin quickly become a required luteotropin by day 25, after which pregnancy-specific increases in prolactin can increase total progesterone production and extend the functional life of luteal tissue to the end of gestation even in animals in which the luteal phase of the cycle would be shorter. Unclear are the extents to which to cause of the pregnancy specific rise in prolactin can be experimentally explained satisfactorily, and/or shown to be related to the simultaneous rise in relaxin; the extent to which in vitro studies can address the luteal cell populations responsive to in vivo LH and prolactin support or stimulation, or to luteolytic insults by exogenous or endogenous-prepartum PGF; and the extent to which low intra-ovarian concentrations of progesterone may impact the timing of proestrus onset beyond the central effects of peripheral progesterone on the hypothalamic-pituitary (HP) axis.

The combination of factors that regulate the timing and occurrence of the spontaneous termination of anestrus by means of increased GnRH pulsatility include the following. Cessation of luteal function and/or declines in peripheral progesterone to concentrations no longer suppressive to the HP-axis; alterations in hypothalamic serotonergic and/or dopaminergic activity that increase GnRH pulsatility either directly or as a result of effects on
prolactin secretion; increased GnRH pulastility elicited directly or indirectly by changes the in peripheral and central hormonal milieu that occur as part of an endogenous, typically non-photoperiod-entrained circannual cycle; and each of the latter as modified by exposure to environmental cues including bitch-bitch pheremonal stimulation. Unclear are the extents to which these factors can be experimentally elucidated using the following - study of dogs with short cycles following abbreviated luteal phases, as in some families of Alsatian dogs; examination of endogenous difference between animals that respond well vs. poorly to dopamine-agonist or GnRH- agonist treatments for proestrus induction; examination of hormonal effects of exposure of anestrus-bitches to bitches in proestrus/estrus as used to shorten intra-estrus interval in commercial breeding kennels; examination of circannual changes in reproductive and other hormones in neutral-photoperiod-exposed gonadectomized dogs. Interesting would be the examination of corresponding changes in dogs that appear to have photoperiod-entrained circannual reproductive cycles (e.g., Basenji) in Northern vs. Southern hemisphere locales.

The most unique aspects of canine ovarian cycles appear to be involved with their timing and rate of progression, with the underlying mechanisms being very similar to those shown to be involved in cycles of more extensively studied domestic animals and other mammalian animal models.