NUTRITIONAL REGULATION OF REPRODUCTION AND OVARIAN FUNCTION IN POSTPARTUM BEEF COWS

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CHAPTER I

INTRODUCTION

Reproductive potential of beef cows is strongly dependent on nutrient availability. Since cow-calf production systems use natural forage as the main source of feed, grazing cows often have quantitative or qualitative deficiencies of nutrient supply, which can compromise reproduction. Because length of gestation limits cows to one calf per year, the potential number of calves per year is not achieved due to prolonged anovulation and delayed establishment of pregnancy.

The mechanisms associated with the onset of first postpartum estrus and ovulation are not completely understood. Nutritional management can be a practical option to initiate the resumption of ovarian function before the onset of breeding. However, the sequence of events and metabolic signals responsible for induction of first ovulation must be elucidated so optimal feeding strategies can be developed.

Postpartum anestrus is a transition period when the hypothalamic-pituitary-ovarian axis recovers the cyclic activity that was disrupted during pregnancy. Several metabolic signals that can potentially affect the reproductive axis and resumption of ovulation have been studied (Bossis et al., 1999, 2000) using nutritionally induced anestrous heifers as a model. This approach allows endocrine and reproductive function of well-fed and underfed animals to be evaluated, but it does not consider the suckling-induced delay in resumption of pulsatile LH secretion. Therefore, metabolic signals that mediate nutritional modulation of reproductive function should be studied in suckled beef cows to determine endocrine and reproductive regulation in a production situation.

Under this scenario, two experiments were conducted to study the influence of nutrient intake on endocrine and reproductive function at the first postpartum estrus in suckled beef cows. The specific objectives were: 1) to determine the effects of BCS at calving and postpartum nutrient intake on estrous behavior, ovarian function, pregnancy rate at first estrus, and plasma concentrations of IGF-I, leptin, NEFA, glucose, insulin, and thyroxine in primiparous beef cows; 2) to explore potential relationships between postpartum endocrine and reproductive

functions, and 3) to determine the effect of nutrition on concentrations of steroids in follicular fluid aspirated at the first estrus and on luteal function during the subsequent estrous cycle.

CHAPTER II

REVIEW OF LITERATURE

Postpartum Reproduction in Cows

Characteristics of the postpartum anestrous period

Introduction. Mechanisms controlling postpartum anestrus and resumption of estrous cycles after parturition in beef cows have not been elucidated. Suckling, nutrition and the interaction are major factors controlling the length of the postpartum interval. Calving season, age at parturition, breed, presence of a bull, and ocurrence of dystocia are minor factors that can also influence the length of the postpartum anestrous period (Short et al., 1990; Yavas and Walton, 2000). These minor factors are beyond the scope of this study, and will not by included in this review.

Secretion of gonadotropin and steroid hormones from parturition to first postpartum estrus resemble those

ocurring during the transition from the prepubertal period to normal estrous cycles. Different anestrous conditions may share similar mechanisms to suppress ovarian function. A sequence of events occur during the transition from parturition to resumption of estrous cycles, including: 1) involution of the uterine tract after parturition, 2) reinitiation of follicular development and maturation, 3) increased secretion of GnRH, LH, and estradiol, 4) ovulation followed by a transient increase in progesterone secretion, and 5) estrous behavior followed by ovulation and normal luteal function. Uterine involution does not influence the length of the postpartum anestrous period (Kiracofe, 1980), so it will not be discussed in this review.

Gonadotropins. Mechanisms controlling LH secretion may be responsible for the initiation of ovarian function after parturition in cattle (Wettemann, 1980; Butler and Smith, 1989; Short et al., 1990). Pulsatile LH secretion is controlled by the hypothalamic GnRH pulse generator in ewes (Clarke and Cummins, 1982). Secretion of LH is temporally associated with GnRH release into the third-ventricle cerebrospinal fluid of cyclic (Yoshioka et al., 2001) and

anestrous (Gazal et al., 1998) cows. Increased frequency of exogenous GnRH pulses reduces LH pituitary concentrations, increases LH pulse frequency and serum concentration, as well as stimulates ovarian function of anovulatory beef cows (Bishop and Wettemann, 1993; Vizcarra et al., 1997, 1999; Hamilton et al., 1999). Hence, infrequent episodic release of GnRH may be linked to the anestrous condition of beef cows (Walters et al., 1982c; Moss et al., 1985; Short et al., 1990; Rasby et al., 1992).

Pituitary content of LH in cattle is reduced during late gestation, probably by high circulating concentrations of steroids (Nett, 1987) produced by the ovaries and/or the placenta. Concentrations of progesterone and estradiol decrease at parturition, thus, the hypothalamus-pituitary axis may recover from the negative feedback caused by these steroids, allowing secretion of LH to increase with time after parturition (Rawlings et al., 1980; Walters et al., 1982a).

Mean concentrations of LH in serum began to increase as early as 5 to 8 d after parturition (Erb et al., 1971; Ingalls et al., 1973) and continued to increase to reach maximal levels after 20 to 30 d postpartum (Peters et al., 1981; Riley et al., 1981; Humphrey et al., 1983). These

increases are coincident with increases in LH pulsatility and follicular development (Savio et al., 1990a).

Pulsatility of LH secretion during the postpartum anestrous period ranged from 1 to 2 pulses per 6 h (Walters et al., 1982c; Humphrey et al., 1983; Schallenberger, 1985; Nett et al., 1988). The frequency of LH pulses increased to approximately 1 to 2 pulses per h before the first postpartum ovulation (Peters et al., 1981; Terqui et al., 1982; Schallenberger, 1985; Savio et al., 1990a). Amplitude of LH pulses may also contribute to increased systemic concentrations of LH because LH pulse amplitude increased with time after parturition (Leung et al., 1986). Since the number and the affinity constant of GnRH binding sites did not change during the postpartum period (Moss et al., 1985; Leung et al., 1986), the reduced frequency of LH pulses is possibly due to limited secretion of GnRH from the hypothalamus. Frequency of LH pulses increases before the onset of puberty, at times when ovarian activity is reestablished after seasonal or lactational anestrous (Haresign et al., 1983), and during the follicular phase of the bovine estrous cycle that precedes the LH surge and ovulation (Rahe et al., 1980; Walters and Schallenberger, 1984; Yoshioka et al., 2001). A decrease in progesterone

and an increase in estradiol concentrations are required to elicit the preovulatory surge of gonadotropins (Kesner et al., 1982; Hansel and Convey, 1983). Cows with increased LH pulsatility had estrogen-active large follicles (Spicer et al., 1986b) and a 10- to 14- fold increase in serum estradiol compared with cows with minimal LH pulsatility (Crowe et al., 2001).

Concentrations of FSH in serum of cows increased more rapidly than concentrations of LH after parturition (Schallenberger et al., 1982; Peters and Lamming, 1984). Secretion of FSH occurs within 5 d after parturition (Schallenberger et al., 1982; Schallenberger, 1985; Crowe et al., 1998) and is constant from 14 to 45 d postpartum (Convey et al., 1983; Leung et al., 1986; Nett et al., 1988). Pituitary content of FSH (Moss et al., 1985; Nett et al., 1988) and GnRH-induced release of FSH from the pituitary gland (Leung et al., 1986) did not change during the first 60 d after parturition. Regulation of FSH secretion may require a minimal input of GnRH and is mainly regulated at the pituitary by ovarian estradiol and inhibin (Clarke, 1989; McNeilly, 1997). Concentrations of FSH, unlike those of LH, do not appear to limit the initiation

of ovarian function during the postpartum period (Walters et al., 1982a; Roche et al., 1992; McNeilly, 1997).

Steroids. Concentrations of progesterone are minimal at parturition in beef and dairy cows (Henricks et al., 1972; Arije et al. 1974; Humphrey et al., 1983) and are < 1 ng/mL during postpartum anovulation, indicating the absence of a functional corpus luteum (Schams et al., 1978; Savio et al., 1990a; Perry et al., 1991b). Transient increases in concentrations of progesterone in serum usually occurred during a 3- to 9-d period before the first postpartum estrus of lactating beef cows (Donaldson et al., 1970; Perry et al., 1991b; Werth et al., 1996). These initial increases in serum progesterone were attributed to the corpus luteum formed after the first ovulation (Lauderdale, 1986; Perry et al., 1991b) or to luteinized ovarian follicles, which may also produce progesterone (Donaldson et al., 1970; Corah et al., 1974). The physiological significance of initial increases in progesterone prior to the first postpartum estrus remains unknown. Progesterone secreted after ovulation without estrus may remove the hypothalamic refractoriness to estradiol that triggers estrous behavior (Allrich, 1994), may enhance GnRH

secretion and pituitary sensitivity to estradiol (Schallenberger, 1985), and/or increase synthesis of LHβ in gonadotropes (Looper, 1999).

Concentrations of estradiol in serum decrease abruptly within 1 to 6 d after parturition and are minimal during the early postpartum period (Henricks et al., 1972; Echternkamp and Hansel, 1973; Humphrey et al., 1983). During postpartum anovulation, concentrations of estradiol in serum may increase occasionally due to growth of dominant follicles (Yavas and Walton, 2000), although, concentrations of estradiol in serum and follicular growth may not be associated during nonovulatory follicular waves (Murphy et al., 1990, Stagg et al., 1995). Concentrations of estradiol in serum increased just before the first postpartum estrus (Henricks et al., 1972; Echternkamp and Hansel, 1973; Arije et al., 1974) and are associated with growth of the ovulatory follicle (Murphy et al., 1990; Perry et al., 1991b; Stagg et al., 1995). Secretion of estradiol was greater in cows treated with intermittent doses of GnRH (Walters et al., 1982a) or LH (Duffy et al., 2000) during the early postpartum period than in control cows. Whether the first postpartum ovulation (usually followed by a short luteal phase) is preceded by normal

increases in estradiol in serum is still unknown, but the preovulatory increases in estradiol in serum before the first postpartum estrus are similar in magnitude and duration to those during the estrous cycle (Spicer and Echternkamp, 1986).

Follicular development after parturition. Stevenson and Britt (1980) proposed the following endocrine events to explain the resumption of follicular growth after parturition: 1) follicles start growing after parturition in response to surges in FSH secretion in the presence of low LH concentrations, 2) growing follicles increase estrogen secretion, which induces a positive feedback on episodic LH secretion, 3) increased LH secretion and sustained FSH secretion stimulate further follicular growth and differentiation prior to first ovulation, and 4) greater secretion of estrogen induces a surge of LH and FSH which lead to ovulation.

Follicular waves occur recurrently during early pregnancy (Ginther et al., 1989), however, follicular growth decreases gradually between 7 to 9 mo of pregnancy until follicles ≥ 6 mm are undetectable during 21 d before parturition (Ginther et al., 1996). Maximal concentrations

of progesterone and estradiol in plasma during the last third of pregnancy may suppress gonadotropin secretion and arrest follicular growth (Nett, 1987). Follicular development and establishment of the first dominant follicle occurs within 10 to 20 d after parturition in beef and dairy cows (Murphy et al., 1990; Savio et al., 1990a; Stagg et al., 1995). A transient increase in FSH concentrations after parturition (Beam and Butler, 1997; Crowe et al., 1998) precedes the emergence of each follicular wave (Adams et al., 1992; Stagg et al., 1998). Periods of growth and regression of medium size follicles (5 to 9 mm) were detected before any follicles became dominant (≥ 10 mm; Murphy et al., 1990; Savio et al., 1990a). Number of small (1 to 3.9 mm) and large (≥ 8 mm) follicles did not change whereas number of medium (4 to 7.9 mm) size follicles increased during days 7 to 42 after parturition in anovulatory suckled cows (Spicer et al., 1986c). Thus, the growth and regression of dominant follicles in the early postpartum period (i.e. < 42 d) is likely not a limiting step to resumption of ovarian function in beef and dairy cows.

The majority (73 %) of dairy cows ovulated the first postpartum dominant follicle (DF; Savio et al., 1990a) in

contrast to a small percentage (11 %) of beef cows, which on the average turned over three (range 2 to 6) DF before the first ovulation (Murphy et al., 1990). Ovulation in beef cows may be delayed by suckling (Nett, 1987; Williams, 1990; McNeilly, 1997) and/or inadequate energy intake during the early postpartum period (Dunn and Kaltenbach, 1980; Eadson et al., 1985; Randel, 1990). Cows with suckled calves (Henao et al., 2000) or receiving a low postpartum energy intake (Stagg et al., 1995) had 3 or more follicular waves before the first estrus than nonsuckled or well-fed cows, respectively. Primiparous cows ovulated 30 d later than mature cows after the appearance of the first postpartum DF (Dimmick et al., 1991). Suckling (Short et al., 1990; Williams, 1990) and restricted nutrient intake (Day et al., 1986; Richards et al., 1989a; Kurz et al., 1990) reduce the pulsatile secretion of LH. The absence of sufficient LH pulses is associated with atresia rather than ovulation of DF (Sirois and Fortune, 1990; Savio et al., 1993; Stock and Fortune, 1993). Conversely, calf removal (Walters et al., 1982a; Edwards, 1985; Williams et al., 1993) or increased nutrient intake (Kurz et al., 1990; Perry et al., 1991a; Wright et al., 1992) increases LH pulse frequency and hastens the initiation of luteal

activity. Administration of hourly LH pulses either prolonged the dominance phase or caused the ovulation of the first DF after calving (Duffy et al., 2000). In addition, LH pulse frequency was doubled within 5 d after restricted suckling, leading to a rapid resumption of ovulation in 50 % of cows during the early postpartum period (Stagg et al., 1998). Collectively, these observations indicate that the pulsatile secretion of LH regulates final growth and fate of DF during the early postpartum period.

Spicer and Echternkamp (1986) suggested that the steroidogenic capacity of large follicles changes during the postpartum period. Since large follicles are present on the ovarian surface for considerable lengths of time prior the first ovulation, and because exogenous GnRH (Kesler et al., 1977, 1980; Wettemann et al., 1982) or estradiol (Short et al., 1979) can elicit normal LH surges during the early postpartum period, it is possible that the concentrations of estradiol produced by large follicles may be unable to stimulate preovulatory LH surges during the postpartum period, or, large follicles may be incapable of producing sufficient estradiol for appearance of the normal proestrual increase in estradiol (Spicer and Echternkamp,

1986). Size of the largest follicle was correlated (r=0.53) with concentrations of estradiol in plasma at 48 d postpartum and cows with follicles ≥ 10 mm released considerably more LH in response to a GnRH challenge than those with follicles ≤ 10 mm in diameter (Lishman et al., 1979).

Steroids in follicular fluid. Steroidogenic capabilities of small and medium follicles did not change during 56 d after parturition, and both sizes of follicles produced mainly progesterone (Spicer et al., 1986a). However, large follicles shifted from progesterone to estradiol production as time postpartum increased, which suggests that an increase in concentrations of estradiol of large follicles may be an essential step to restore the ovarian activity after parturition (Spicer et al., 1986). Large follicles collected at 15, 30 or 45 d postpartum contain approximately 20 % of the estradiol, testosterone and androstenedione found in preovulatory follicles collected during the estrous cycle (Braden et al., 1986). Intrafollicular concentrations of total IGF-I did not change with time postpartum (Rutter and Manns, 1991) and were similar between estrogen-inactive and estrogen-active

follicles (Spicer et al., 1988). However, a possible role of IGF-I in modulating follicular steroidogenesis in postpartum cows cannot be ruled out until concentrations of IGF-I binding protein activity in follicular fluid are characterized.

Steroidogenic capabilities of postpartum follicles may be depressed by suckling (Bellin et al., 1984), low body condition at calving (Prado et al., 1990) and/or inadequate energy intake (Kendrick et al., 1999). How these factors suppress ovarian function is unknown, but decreased LH pulse frequency (Prado et al., 1990), minimal concentrations of IGF-I in follicular fluid (Ryan et al., 1994; Kendrick et al., 1999) and/or reduced aromatase activity (Prado et al., 1990) could be involved. Concentrations of steroids in follicular fluid of large follicles during postpartum anovulation were similar to those of luteal phase follicles (Braden et al., 1986). Pulsatile treatment with LH during the midluteal phase of the estrous cycle increased follicular fluid concentrations of estradiol and androstenedione, and increased the expression of mRNAs for cytochrome P450 17α-hydroxylase (P450c17) and 3β-hydroxysteroid dehydrogenase (3β-HSD) but not for cytochrome P450 aromatase (P450arom) (Manikkam et

al., 2001). There may be a threshold level of estrogenicity, below which the physiological changes to trigger ovulation cannot be initiated (Pinto Andrade et al., 1995).

Luteal function. Luteal function after the first postpartum ovulation is generally abnormal. The first corpus luteum formed in postpartum cows has a life span that is shorter than normal. Short luteal phases are common after spontaneous ovulation (Perry et al., 1991b; Werth et al., 1996; Looper, 1999) or after ovulation induced by weaning (Odde et al., 1980; Copelin et al., 1987; Breuel et al., 1993), GnRH (Kesler et al., 1980; Wettemann et al., 1982), PMSG (Wettemann et al., 1982) or hCG injections (Pratt et al., 1982; Sheffel et al., 1982). Because maternal recognition of pregnancy in the cow occurs between 15 to 17 d after estrus (Northey and French, 1980), a corpus luteum cannot support pregnancy if it regresses before 15 d after estrus.

The first ovulation that occurs after parturition usually is not preceded by estrous behavior and is followed by a short ovarian cycle. Estrous behavior was not detected in 86% of cows (range: 70-100%) prior the first postpartum

ovulation (Graves et al., 1968; Murphy et al., 1990; Savio et al., 1990a; Perry et al., 1991b), which was followed by a short luteal phase in 83% of cows (range: 70-100%) before the first estrus (Corah et al., 1974; Rawlings et al., 1980; Humphrey et al., 1983; Murphy et al., 1990, Perry et al., 1991b; Stagg et al., 1995; Werth et al., 1996; Looper, 1999; Mackey et al., 2000). Length of the estrous cycle, after the first postpartum estrus, often is normal (80% of the time, range: 72-94 %; Corah et al., 1974; Odde et al., 1980; Looper, 1999). During a short luteal phase, all cows ovulated the first dominant follicle of a wave (Murphy et al., 1990; Savio et al., 1990b; Stagg et al., 1995). The incidence of short luteal phases decreased after consecutive postpartum ovulations (Eger et al., 1988). Short luteal phases and/or interovulatory intervals averaged 7 to 10 d in length between the first and second postpartum ovulation (Murphy et al., 1990; Perry et al., 1991b; Werth et al., 1996). Similarly, interestrus intervals were commonly 7 to 10 d in length after weaning-induced ovulations (Odde et al., 1980; Ramirez-Godinez et al., 1982). A transient pre-estrus increase in progesterone enhanced pregnancy rate at the first postpartum estrus in primiparous beef cows (Werth et al., 1996).

Concentrations of progesterone in plasma were similar during the first 5 d after estrus for cows with short or normal life span corpora lutea (CL), but progesterone secretion from short-live CL declined abruptly on d 5 to 7 after estrus (Ramirez-Godinez et al., 1981, 1982; Copelin et al., 1987, 1989a). Size of preovulatory follicles was similar for those expected to develop CL with short or normal life span (Braden et al., 1989; Perry et al., 1991b), indicating that preovulatory size is not associated with abnormal CL development. Although concentrations of luteal progesterone and LH receptors, ratio of large/small luteal cells, sensitivity to the luteolytic signal, pre- and postovulatory gonadotropin support, and concentrations of LH receptors in follicular cells may be related to less than normal progesterone secretion by CL with a short life, a premature luteolytic signal seems to be responsible for short luteal phases (for review see: Hunter, 1991; Garverick et al., 1992).

The uterine luteolysin, $PGF_2\alpha$, is associated with short luteal phases in postpartum cows and ewes. Inhibition of $PGF_2\alpha$ synthesis with indomethacin after GnRH-induced ovulation, decreased concentrations of 13,14-dihydro-15-keto $PGF_2\alpha$ (PGFM), normalized progesterone secretion, and

prolonged luteal function of suckled beef cows (Troxel and Kesler, 1984). Immunization against a $PGF_2\alpha$-ovalbumin conjugate maintained the life span and progesterone secretion from the corpora lutea induced by calf weaning at 30-35 d postpartum (Copelin et al., 1989b). Hysterectomy prevented the early regression of CL that were expected to develop a short luteal phase in postpartum cows (Copelin et al., 1987) and anestrous ewes (Southee et al., 1988; Lassoued et al., 1997). Basal and oxytocin-stimulated secretion of $PGF_2\alpha$ from bovine endometrium explants were greater on day 5 of a short compared to a normal luteal phase (Zollers et al., 1991). Suppression of $PGF_2\alpha$ release and hysterectomy extend the life span of short-lived corpora lutea, indicating that a premature release of uterine $PGF_2\alpha$ may be the cause of early luteal regression.

Regression of CL in ruminants involves complex interactions between pulses of neurohypophyseal/luteal oxytocin and episodic secretion of uterine $PGF_2\alpha$ (Silvia et al., 1991; McCracken et al., 1999). Concentrations of oxytocin and PGFM increased simultaneously during luteolysis in cows with short or normal estrous cycles (Peter et al., 1989). An increase in concentrations of $PGF_2\alpha$ occurred on days 4 to 9 after a hCG injection in cows that

had a short luteal phase but not in cows with normal length cycles (Cooper et al., 1991). An oxytocin injection induced a greater PGFM release on day 5 in cows with a short compared with a normal luteal phase (Zollers et al., 1989). Cows treated with progesterone, prior to weaning or GnRH-induced ovulation, formed corpora lutea with normal life spans (Ramirez-Godinez et al., 1981; Sheffel et al., 1982), indicating that progesterone priming of the uterus may delay the premature release of $PGF_2\alpha$.

Greater development of oxytocin binding sites in the uterus may determine the life span of CL during the early luteal phase. Concentrations of endometrial oxytocin receptors were similar at d 1 after GnRH-induced ovulation in anestrous ewes treated with progesterone or controls, but the presence of oxytocin receptors was greater at d 5 in the control group (Hunter, 1991). On d 5 after the first postpartum induced-ovulation, concentrations of progesterone in plasma were less and concentrations of oxytocin endometrial receptors were greater in cows expected to have short compared with those having a normal estrous cycles (Zollers et al., 1993). Presence of oxytocin receptors in endometrial explants collected from ovariectomized cows is inhibited by progesterone in a dose-

dependant manner (Mann, 2001). Progesterone directly inhibited oxytocin binding to their receptors in the rat uterus (Grazzini et al., 1998). Oxytocin-induced $PGF_2\alpha$ release was less in cows (Zollers et al., 1989) and ewes (Vallet et al., 1990) pretreated with progesterone compared with control ewes. Thus, the uterus that is not exposed to progesterone may be more responsive to oxytocin during the early luteal phase, setting up the conditions for development of a $PGF_2\alpha$-oxytocin positive feedback loop that may initiate luteal regression.

Dietary lipids may alter luteal function in beef cows. Luteal synthesis and secretion of progesterone is regulated by multiple factors, including steroidogenic enzymes and availability of its main precursor, cholesterol (Niswender et al., 2000). Feeding diets with greater amounts of lipids after parturition increased concentrations of cholesterol in plasma (Williams, 1989; Hightshoe et al., 1991) and follicular fluid (Wehrman et al., 1991), and increased life span of CL and progesterone secretion (Williams, 1989, Hightshoe et al., 1991). High-lipid diets enhanced, by 2-3 fold, the ability of preovulatory granulosa cells to secrete pregnenolone and progesterone in vitro (Wehrman et al., 1991). Dietary-induced increases in serum lipids were

directly associated with total steroidogenic area in luteal tissue occupied by lipids and concentrations of progesterone in plasma, and inversely related with rate of progesterone clearance (Hawkins et al., 1995). In theory, dietary lipids may enhance luteal function of postpartum beef cows, possibly by increased luteal tissue steroidogenic capacity and/or reduced progesterone clearance.

Estrous behavior. Expression of estrous behavior is essentially controlled by relative concentrations of ovarian steroids, estradiol and progesterone. Injections of estradiol benzoate can induce normal estrous behavior in ovariectomized cows (Carrick and Shelton, 1969; Katz et al., 1980; Vailes et al., 1992) and the percentage of cows exhibiting estrus (Cook et al., 1986) and duration and intensity of estrous behavior may be dose dependant (Nessan and King, 1981; Lyimo et al., 2000). However, no relationship was detected between concentrations of estradiol in plasma and duration or intensity of estrous behavior at natural (Glencross et al., 1981; Coe and Allrich, 1989) or induced estrus (Cook et al., 1986). Estrous behavior did not occur in beef heifers immunized

against estradiol benzoate, even when they were exposed to concentration of estradiol adequate to induce estrus in normal heifers (Martin et al., 1978). These results indicate that estradiol concentrations in plasma must increase, until a threshold is reached, to induce estrous behavior. However, the relationship between concentrations of estradiol in plasma and duration and/or intensity of estrus is still unclear.

Progesterone has inhibitory effects on expression of estrous behavior in cyclic cows. Simultaneous administration of progesterone blocked the ability of estradiol benzoate to induce estrous behavior in ovariectomized heifers (Rajamahendran et al., 1979) and cows (Vailes et al., 1992). Concentrations of progesterone in plasma equivalents to those that can be detected during the early or middle-late luteal phases, or supraphysiological doses, lineraly decreased the expression of estrous behavior traits and delayed the onset of standing behavior (Davidge et al., 1987). Standing behavior was suppressed to a greater extent than mounting activity when high concentrations of progesterone and estradiol ocurred at the same time (Davidge et al., 1987; Vailes et al., 1992). Pretreatment with progesterone did not

facilitate the estrus-inducing actions of estradiol in ovariectomized cows (Allrich et al., 1989). After a threshold concentrations of progesterone was reached, estrous behavior was inhibited even in the presence of estrus-inducing concentrations of estradiol (Allrich, 1994).

Several other factors can interact to alter the expression of estrous behavior. These include: age of the cow (Nebel et al., 1996; Mathew et al., 1999), number of cows in estrus (Hurnik et al., 1975; Helmer and Britt, 1985; Floyd, 2001), environmental temperature (Gangwar et al., 1965; Abilay et al., 1975; White, 2000), and days postpartum (King et al., 1976; Britt et al., 1986; Pennington et al., 1986). Suckling decreased the intensity (LaVoie et al., 1981) and the percentage of cows exhibiting estrous behavior (Wiltbank and Cook, 1958; Graves et al., 1968) at the first detected ovulation. Similarly, negative energy balance decreased the percentage of cows exhibiting estrous behavior at the first detected ovulation (Spicer et al., 1990). Duration of estrus is highly variable (3 to 28 h; Allrich, 1994; White et al., 2002) between cows, prossibly because hypothalamic sensitivity to threshold

concentrations of estradiol may be different among cows (Darwash et al., 2001).

Progesterone may stimulate, rather than inhibit, estrous behavior in postpartum cows as they become reproductively active. The first postpartum ovulation is frequently not preceded by estrous behavior (Wettemann, 1980; Short et al., 1990). Estrus is usually expressed prior to the second ovulation in the majority of cows (King et al., 1976; Perry et al., 1991b). A transitory increase in concentrations of progesterone commonly preceded the first pubertal (Rutter and Randel, 1986) and postpartum estrus in beef cattle (Perry et al., 1991b; Werth et al., 1996; Looper, 1999). An injection of estradiol benzoate after short-term progesterone treatment increases the estrous response of anestrous cows (McDougall et al., 1992; Fike et al., 1997). It is likely that short-term transient luteal activity must precede the first postpartum estrus. Progesterone acting on neural centers may enhance the effect of estradiol on estrous behavior. High levels of estradiol produced during late pregnancy may induce a refractory state to estradiol in the brain, which is reversed by progesterone exposure (Carrick and Shelton, 1969). Alternatively, progesterone may act at the ovarian

level to stimulate estrus and ovulation by altering LH secretion. Treatment of anestrous cows with progesterone, increased synthesis of LHβ mRNA in the pituitary (Looper, 1999), LH secretion (Anderson et al., 1996), and number of LH receptors and concentrations of estradiol within the largest follicle (Inskeep et al., 1988), a sequence of events that can cause estrus and ovulation.

Nutrition, BCS, suckling, and postpartum reproduction

Introduction. Nutrition is important in postpartum cows because several biological events are overlapping and competing for a limited amount of nutrients: uterine involution, maximal lactation, return to cyclic ovarian function, pregnancy and early embryo development (Smidt and Farries, 1982). Even though suckling is a major factor affecting the duration of the postpartum anestrous period (Stagg et al., 1998), the onset of luteal activity in response to early weaning depends on body energy reserves of cows at weaning (Bishop et al., 1994). This indicates that nutritional status can modulate the length of the postpartum anestrous interval when suckling influences become less restrictive. Nutrition may influence

reproductive endocrine function at the hypothalamus (Imakawa et al., 1987; Rasby et al., 1992; Vizcarra et al., 1997), pituitary (Beal et al., 1978; Sen et al., 1979), and/or ovaries (Harrison and Randel, 1986; Rhind et al., 1989; Quesnel et al., 2000).

Prepartum nutrition. The relationship between nutritional status and reproductive performance has been extensively studied in beef cows and the conclusion is that cows on a high level of nutrition before parturition resume postpartum ovarian activity earlier than cows on a low level of nutrition. Restricted nutrient intake prepartum results in thin cows at parturition, a prolonged postpartum anestrous period, and less cows in estrus during the breeding season (Wiltbank et al., 1962; Bellows and Short, 1978; Dunn and Kaltenbach, 1980; Wright et al., 1987). Nutrient intake before calving also influences the subsequent interval from parturition to pregnancy (Dunn et al., 1969) and pregnancy rate (Dunn et al., 1969; Selk et al., 1988). However, variations in nutrient intake during the last third of gestation did not affect reproductive performance in postpartum mature or first-calf cows (Selk et al., 1988; Whittier et al., 1988; DeRouen et al., 1994),

suggesting that postpartum reproduction may be independent of prepartum nutrient intake if cows calve with adequate body energy reserves. Cows with large changes in body condition during the last 90 d of gestation, but with similar body energy reserves (BCS 5) at calving, had similar reproductive performance (Morrison et al., 1999). Greater levels of energy prior to parturition may increase pregnancy rates without alteration of cow body condition or weight (Marston et al., 1995).

The adverse effects of restricted energy intake prepartum on subsequent reproductive performance may be partially overcome by increasing energy intake after parturition (Wiltbank et al., 1962; Dunn et al., 1969; Houghton et al., 1990a). However, the response of cows to greater energy intake postpartum is influenced by the severity of prepartum energy restriction, which is reflected in body condition at calving. Thin primiparous or mature cows (BCS $\leq$ 4) had improved reproductive performance in response to high energy intake postpartum compared with cows in moderate to good condition (BCS $\geq$ 5) (Richards et al., 1986; Wright et al., 1987, 1992; Spitzer et al., 1995). Increased energy intake postpartum enhanced follicular development and the incidence of ovulation in

cows (Perry et al., 1991a) and shortened the postpartum anovulatory interval by 20 d in primiparous cows with a BCS of 4 at calving (Lalman et al., 1997). Even when postpartum nutrient intake improved reproductive performance of thin cows it did not reverse completely the undesirable effects of inadequate prepartum nutrition, and this effect is more evident in first-calf than in mature cows. Wiltbank and coworkers (1962) suggested that prepartum energy intake affects the interval from calving to first estrus whereas postpartum energy intake influences conception rates.

Body condition. Amount of subcutaneous fat is closely related to reproductive performance of postpartum cows. In beef cows, subcutaneous fat reserves are subjectively quantified by visual appraisal or palpation of specific animal fat depots at vertebral processes, over the ribs, pins and hooks, and around the tail insertion. According to the amount of fat present in the animal, a body condition scores on a nine-point scale is assigned, with 1 representing a severely emaciated and 9 a very obese cow (BCS; Wagner et al., 1988). The amount of carcass lipids/fats and the total energy content of carcass were highly correlated with BCS (Wright and Russel, 1984; Wagner

et al., 1988; Houghton et al., 1990b; Yelich et al., 1995). Thus, BCS can be used to assess the amount of body energy reserves in cows under field and research situations.

Decreased energy intake causes loss in live weight and BCS (Wiltbank et al., 1962; Corah et al., 1975; Richards et al., 1989a; Bossis et al., 1999). However, live weight at calving did not affect subsequent reproductive performance (Osoro and Wright, 1992), which is more strongly related to body condition at calving than to prepartum change in BCS or live weight (Dunn and Kaltenbach, 1980; Selk et al., 1988; DeRouen et al., 1994). Selk et al. (1988) concluded that body condition at calving and at the start of breeding are dominant factors determining the occurrence of pregnancy in range cows, with changes in live weight between 2 and 4 mo prepartum modulating the response. They also indicated that the increase in pregnancy rate per unit of increase in body condition is greater (20%) for cows that calved with a BCS between 4 and 6 than for thiner or fatter cows.

Reproductive and calf performance should be optimized if mature cows calve with a BCS $\geq$ 5 (Richards et al., 1986; Morrison et al., 1999), primiparous cows calve with BCS $\geq$ 6 (DeRouen et al., 1994; Spitzer et al., 1995), and both age

groups maintain weight during lactation. Thin primiparous or mature cows at calving will respond to increased postpartum energy intake by enhanced reproductive performance (Dunn et al., 1969; Dunn and Kaltenbach, 1980; Richards et al., 1986; Wettemann et al., 1986; Wright et al., 1992; Spitzer et al., 1995; Vizcarra et al., 1998), although postpartum reproduction may be still less than optimal for primiparous cows. Nonlactating cows (Richards et al., 1989a; Bishop and Wettemann, 1993) and heifers (Vizcarra et al., 1995; Bossis et al., 1999) become anovulatory when BCS is about 3.5.

Mechanisms relating body energy reserves with an early resumption of ovarian function during the postpartum period are still under research. Available evidence suggests that body condition may have direct effects on the GnRH pulse generator and/or the ovary. Thin cows at calving had increased amounts of met-enkephalin in the preoptic area and had similar hypothalamic GnRH content before or at 48 h after calf removal (Connor et al., 1990). Body condition of thin nonovulatory cows was negatively associated with total GnRH content in the infundibular stalk-median eminence and maximum LH secreted after GnRH challenge (Rasby et al., 1992), suggesting that inadequate body energy reserves may

depress GnRH release, and thus, LH secretion. Pulsatile GnRH infusion induced luteal function in thin anestrous cows (Bishop and Wettemann, 1993) and pituitary gland sensitivity to GnRH was independent of body condition at calving (Wright et al., 1990). Body condition at calving was not related to LH pulse frequency at 14 d postpartum but it was positive associated at 42 and 70 d postpartum (Perry et al., 1991a). This indicates that body condition may modulate LH secretion after the GnRH pulse generator escapes from other inhibitory factors that control its activity during the early postpartum period. Body condition at calving is associated with LH pulse frequency, peripheral concentrations of IGF-I, and onset of ovarian function after early weaning at 40 d postpartum (Bishop et al., 1994). Follicular development during the early postpartum was delayed in thin cows compared with cows in moderate or good body condition at calving, and concentrations of IGF-I in serum and follicular fluid increased with BCS at calving (Ryan et al., 1994). Steroidogenic capacity of large, estrogen-active follicles, was affected by body condition at parturition (Prado et al., 1990; Pinto Andrade et al., 1995). Systemic concentrations of IGF-I were positively associated with BCS

during the postpartum period but not with days to first large follicle (Spicer et al., 2002).

Suckling. Secretion of LH is responsible for the resumption of reproductive function after parturition (Short et al., 1972; Wettemann, 1980; Short et al., 1990; Yavas and Walton, 2000). Pulsatile release of LH is reduced during the early postpartum period because pituitary LH content is minimal after parturition (Moss et al., 1985; Nett et al., 1988). Suckling delayed the onset of LH secretion in cows (Short et al., 1972; Williams et al., 1982), whereas suppression of the suckling stimulus after 20 to 30 d postpartum increased LH secretion (Walters et al., 1982b; Myers et al., 1989; Shively and Williams, 1989; Stagg et al., 1998). Milking twice a day with the cow's calf absent or present did not inhibit pulsatility of LH, suggesting that suckling is a more potent inhibitor of LH secretion than milking (Lamb et al., 1999). Therefore, minimal pulsatile release of LH occurs independently of the suckling inhibition soon after parturition but suckling is a major inhibitor of LH secretion (Nett, 1987) after 20 d postpartum.

Episodic LH secretion at 7 and 14 d postpartum was less in suckled cows than in cows that had calves weaned at 12 h after birth (Williams et al., 1983). Complete (Walters et al., 1982b; Griffith and Williams, 1996), temporary (i.e., 48 to 72 h) (Edwards, 1985; Shively and Williams, 1989; Myers et al., 1989; Salfen et al., 2001), or partial (i.e., once suckling per day) (Stagg et al., 1998; Mackey et al., 2000) suppression of the suckling stimulus increased LH secretion. Calf return after temporary weaning decreased LH pulse frequency within 8 h in anovulatory cows (Edwards, 1985; Shively and Williams, 1989). Early weaning or once-daily suckling after 65 d postpartum did not affect length of the anovulatory interval of primiparous beef cows that calved and were fed to maintain a body condition score (BCS) ≥ 5 until breeding (Bell et al., 1998). Therefore, suckling may influence the anovulatory interval from d 20 to d 65 postpartum after which nutrition plays the primary role.

Suckled beef cows have a longer interval after calving to first estrus or ovulation than milked or nonsuckled cows (Wiltbank and Cook, 1958; Williams, 1990; Stevenson and Lamb, 1997). Continuous presence of a cow's own nonsuckling calf did not suppress the weaning-stimulated increase in LH

concentrations (Williams et al., 1987; Hoffman et al., 1996) and shorter interval to ovulation (Hoffman et al., 1996; Lamb et al., 1997, 1999). However, twice-daily suckling was sufficient to decreased LH release and prolonged the postpartum anovulatory period (Lamb et al., 1999). Duration of the postpartum anovulatory period was not related to suckling behavior of single calves that nursed ad libitum, even when frequency and duration of suckling events decreased with days of lactation (Day et al., 1987). Extreme suckling intensity, induced by twins (Sinclair et al., 1994) or adoption of a foster calf (Wettemann et al., 1978), and frequency of daily suckling greater than once (Randel, 1981; Lamb et al., 1999) extended the postpartum anovulatory period. Thus, it appears that presence of a cow's own nonsuckling calf does not affect weaning-induced LH secretion and reduce the length of the postpartum anovulatory period, whereas at least twice daily suckling, but not milking two or five times per day, can inhibit LH release and ovulation. Calf access to the cow's inguinal area, without suckling, was sufficient to prolong the postpartum anovulatory period (Viker et al., 1993; Stevenson et al., 1994).

Maternal recognition of a cow's own calf may be a critical component of suckling-mediated anovulation. Cows that were suckled by an alien (foster) calf, or had calves weaned, had increased LH pulse frequency and a shorter anovulatory interval than cows that were suckled by their own calf. This indicates that maternal recognition of a calf, not only the suckling event, may be require to inhibit LH release and ovulation (Silveira et al., 1993). Cows suckled ad libitum by alien calves, in presence or in absence of their own calf, had similar intervals to first ovulation as cows suckled by their own calves; both had longer anovulatory intervals than cows that had calves weaned (Lamb et al., 1997). These results indicate that the establishment of cow-calf bond, between a cow and her own or with an unrelated calf, can postpone ovulation in postpartum beef cows. Vision may be an important sense for ewes to recognize lambs, although olfactory and auditory cues are also involved (Lindsay and Fletcher, 1968). Deprivation of a cow's visual and olfactory senses blocked recognition of her calf and released the cow from the suckling-mediated inhibition of LH secretion (Griffith and Williams, 1996). Overall, twice-daily suckling may inhibit pulsatile secretion of LH and prolong the anovulatory

interval if the cow-calf bond has been previously established.

Quantity of GnRH in the hypothalamic area was greater in suckled than in cycling cows (Nett et al., 1987, 1988) and remained unchanged during at least 30 to 45 d postpartum (Moss et al., 1985; Nett et al., 1988). Amount of GnRH receptors in pituitary tissue of suckled cows remained constant (Leung et al., 1986) or increased (Moss et al., 1985; Nett et al., 1988) during the first 7 to 15 d after calving. Concentration of anterior pituitary estradiol receptors on d 15 postpartum in suckled cows was similar to that observed prior to the preovualtory LH surge in cycling cows (Nett et al., 1987, 1988). Pituitary LH content (Walters et al., 1982b) and pituitary LH released after a GnRH challenge (Walters et al., 1982c; Williams et al., 1982) were similar between suckled and nonsuckled anestrous cows. Suckling impaired GnRH release from the median eminence and decreased concentration of LH in plasma (Zalesky et al., 1990). Weaning increased GnRH secretion into cerobrospinal fluid and increased LH pulse frequency (Gazal et al., 1998). These results indicate that suckling suppresses hypothalamic GnRH release and delays LH release and first postpartum ovulation.

Endogenous opioid peptides can potentially mediate the suckling-induced inhibition of LH secretion in postpartum beef cows (Malven et al., 1986; Whisnant et al., 1986b; Myers et al., 1989). Concentrations of opioid peptides and GnRH in the preopic area and hypothalamic tissue were positively associated (Malven et al., 1986). Administration of naloxone, an opioid receptor antagonist, increased LH secretion in suckled cows (Whisnant et al., 1986a, 1986c), but it was ineffective in nonsuckled cows that had high basal LH secretion (Whisnant et al., 1986a). The ability of opioids to inhibit LH secretion may decrease with days after parturition (Whisnant et al., 1986b). Suckled anestrous cows had greater concentrations of opioid receptors in the preoptic-basal forebrain areas than suckled cyclic cows (Trout and Malven, 1988). However, nonsuckled, ovariectomized cows responded to naloxone with increased secretion of LH, suggesting that mechanisms, other than ovarian and/or suckling-induced, may be involved in opioid-mediated inhibition of LH secretion during the early postpartum period (Rund et al., 1989). Suppression of endogenous opioid peptides stimulated LH secretion in dairy cows in negative energy balance (Ahmadzadeh et al., 1998; Kadokawa and Yamada, 2000). The increase in opioidergic

tone has been included as a component of an hypothetical mechanism that integrates maternal behavior, suckling and hypothalamic-pituitary activity to maintain the anovulatory state in postpartum beef cows (Williams and Griffith, 1995). Alternatively, the suppressive effects of suckling on pulsatile secretion of LH may be due to increased sensitivity of the hypothalamus to the negative feedback of estradiol (Acosta et al., 1983). However, the postovariectomy increase in concentrations of LH occurred sooner in nonsuckled than in suckled cows, even when they had similar concentrations of estradiol at ovariectomy (Hinshelwood et al., 1985). Physiological concentrations of estradiol may enhance LH secretion in ovariectomized one-daily suckled cows (Garcia-Winder et al., 1986).

Postpartum nutrition. Increased energy intake after parturition shortened the intervals from calving to ovulation (Dunn et al., 1969; Perry et al., 1991a, Stagg et al., 1995; Lalman et al., 1997, 2000) and increased the percentage of cows exhibiting estrus during the breeding season (Wiltbank et al., 1962, 1964; Richards et al., 1986; Spitzer et al., 1995). Postpartum energy intake did not affect the interval to detection of the first dominant

follicle, but the number of dominant follicles undergoing atresia before first ovulation was greater in cows receiving a low energy diet (Stagg et al., 1995). Poor nutrition caused an increase in turnover of dominant follicles (Murphy et al., 1991). This increase in turnover of dominant follicle may be due to decreased IGF-I secretion (see next section). Restricted energy intake during the early postpartum period depressed LH pulsatility and decreased the size the largest ovarian follicle, suggesting a delay in the establishment of functional dominance in underfed cows (Grimard et al., 1995). Conversely, mean LH concentration, LH pulse frequency, and follicular development were increased in cows fed high energy diets compared with those that received less energy after calving (Perry et al., 1991a). Dietary energy intake after calving may not affect the length of the postpartum interval if cows calve and maintain adequate body energy reserves during lactation (Richards et al., 1986; Spitzer et al., 1995; Stagg et al., 1998). As mentioned earlier, positive effects of high energy intake postpartum on reproduction depends on body condition score at calving and are more evident in thin cows (Dunn and Kaltenbach, 1980; Richards et al., 1986; Wright et al., 1992; Spitzer et al.,

1995). Greater postpartum energy intake increased body condition score at 90 d postpartum, concentrations of IGF-I and insulin, and decreased the length of the postpartum period in primiparous cows that were thin at calving (Lalman et al., 2000). However, feeding a high energy diet postpartum was unable to decrease the mean postpartum interval to estrus to less than 110 d (Lalman et al., 1997, 2000). Low body condition score at calving may exacerbate inhibitory actions of suckling to suppress LH release and, therefore, delay return to estrus. Both negative effects are more pronounced in primiparous than in mature cows.

Increasing dietary energy intake after calving enhanced pregnancy rates of beef cows (Wiltbank et al., 1962, 1964; Dunn et al., 1969; Richards et al., 1986). Randel (1990) summarized results from those experiments and reported that pregnancy rates for cows fed diets restricted in energy content, after calving, varied from 50 to 76 % compared with 87 to 92 % for well-fed cows. Restricted nutrient intake during the postpartum period decreased pregnancy rate even in cows that had calved with good body condition (Rakestraw et al., 1986). Conception rates at the first service ranged from 38 to 62 % compared with 66 to 84 % for energy-restricted and well-fed cows, respectively

(Randel, 1990). These results indicate that restriction of energy intake postpartum may depress fertility at a single insemination, but this presumption has not been tested using an adequate number of cows. Hill et al. (1970) suggested that fertilization failure, not early embryonic mortality, is the cause of depressed conception rates in underfed heifers. Conversely, Spitzer et al. (1978) suggested that conception rates of energy-restricted heifers are reduced by embryo mortality after 4 d postmating.

Increasing energy intake elicits endocrine and metabolic changes that may act directly on the ovary to influence fertility of beef cows. Cows fed high energy postpartum had larger follicles and greater ovarian volume during at least 3 wk prior to first estrus, and conception rate at first service was greater than for energy-restricted cows (Wiltbank et al., 1964). Postpartum energy restriction decreased size of the dominant follicle and this event was not related to changes in LH secretion (Grimard et al., 1995). Growth rate of the dominant follicle and concentrations of insulin and IGF-I were greater in cows fed high energy compared with those fed low energy diet (Armstrong et al., 2001). These results and

those mentioned earlier suggest that increased energy intake may influence growth and perhaps differentiation of the DF, independent of LH secretion. Endocrine secretions from larger preovulatory follicles may enhance oocyte development or uterine ability to support pregnancy and enhance fertility. Britt (1995) proposed that undernutrition during the early postpartum period may alter gene expression of preantral follicles, resulting in abnormal mature follicles that produce low quality oocytes or form CL with abnormal function and, therefore, decrease fertility.

Metabolic signals and reproduction in female mammals

Introduction. Nutritional cues influence the reproductive process in cattle by altering the hypothalamic GnRH pulse generator, which controls the synthesis and secretion of luteinizing hormone (Randel, 1990; Short et al., 1990; Keisler and Lucy, 1996; Wettemann and Bossis, 2000). Restricted nutrient intake delays puberty (Day et al., 1986; Yelich et al., 1996) prolongs the postpartum anestrous interval (Wright et al., 1992; Bishop et al., 1994) and induces anestrus (Richards et al., 1989a; Bossis

et al., 1999) in bovine females by reducing LH pulse frequency. Continuous growth and dominance of preovulatory follicles requires frequent pulses of LH (Stock and Fortune, 1993). Increased nutrient intake increases maximum diameter of the dominant follicle in nutritionally anestrous (Bossis et al., 2000), cyclic (Spicer et al., 1991; Armstrong et al., 2001) and postpartum anestrous (Grimard et al., 1995; Lents et al., 2000) beef cows. Energy intake after parturition affects LH pulse frequency (Perry et al., 1991a) and therefore, modulates final stages of follicular growth. If follicular growth varies with level of nutrient intake, follicular diameter may reflect the endocrine/metabolic status of postpartum cows.

Metabolic signals, either hormones, substrates or products of metabolism, are continuously monitored by the brain and they may affect, directly or indirectly, the GnRH pulse generator, the pituitary gland, and/or the ovaries (Randel, 1990; Schillo, 1992; Stevenson and Lamb, 1997; Wettemann and Bossis, 2000) triggering events that end the anestrous period and lead to the first ovulation. Nutrition may modulate reproductive function by its effects on blood concentrations of metabolites and/or metabolic hormones.

Insulin and Glucose. Insulin, directly or indirectly, affects energy metabolism of practically every tissue or organ in the body. However, specific effects of insulin on the reproductive axis are less clear. Insulin receptors have been detected in different regions of the brain, pituitary gland (Lesniak et al., 1988), and ovarian tissue (Poretsky and Kalin, 1987). Insulin may stimulate gonadotropin secretion, follicular growth and steroidogenesis. Perfused rat hypothalamic fragments had a 8-fold increase in GnRH release in response to low concentrations of insulin only when glucose was available (Arias et al., 1992). Intracerebroventricular infusion of insulin increases basal LH concentration in underfed ewes (Daniel et al., 2000). Insulin can exert gonadotropin-like effects on ovarian tissues (Poretsky and Kalin, 1987), including stimulation of thecal androgen production, granulosa cell proliferation, and estradiol production in cows (Spicer and Echternkamp, 1995). Restricted nutrient intake decreases circulating concentrations of insulin in cows (Richards et al., 1989b; Armstrong et al., 1993; Bossis et al., 1999; Armstrong et al., 2001). Administration of insulin increases concentrations of estradiol in large follicles (Simpson et al., 1994) and

increases ovulation rate of energy restricted superovulated cows (Harrison and Randel, 1986). Follicular recruitment (Gutierrez et al., 1997b) or the ability of follicles to ovulate (Harrison and Randel, 1986) may be enhanced by insulin.

Secretion of GnRH may depend on the availability of metabolic energy. Glucose transport into the central nervous system is mainly mediated by insulin-independent glucose transporters (GLUT-1, -2 and -3), whereas the hypothalamus expresses an insulin-dependant glucose transporter, GLUT-4 (Livingstone et al., 1995). Under dietary restriction, low levels of insulin may redistribute glucose as a function of tissue priorities. Likely, hypoinsulinemia may inhibit glucose uptake by the hypothalamus but not by vital areas of the brain. Nutritional anestrous cows had lower plasma concentrations of glucose and insulin than cows on maintenance diets (Richards et al., 1989b; Bossis et al., 1999). Administration of 2-deoxy-D-glucose (2DG), a glucose antagonist, inhibited estrus and ovulation in well-fed cows (McClure et al., 1978). Furthermore, phlorizin-induced hypoglycemia prevented the increase in insulin and LH concentrations that follow early weaning in beef cows

(Rutter and Manns, 1987). Secretion of LH, but not pituitary sensitivity to GnRH, was abruptly reduced when ovariectomized ewes were injected with 2DG even when concentrations of insulin were increased (Funston et al., 1995b). Similarly, 2DG decreases LH secretion in gonadectomized male lambs (Bucholtz et al., 1996) and female and male rats (Nagatani et al., 1996). However, in male Rhesus monkey, stimulation of LH secretion may depend on the total availability of metabolic fuels instead of a specific increase in glucose concentrations (Schreihofer et al., 1996). Estrous cycles in hamsters occur recurrently when glycolysis or fatty acid oxidation is inhibited, but simultaneous pharmacologic blockage of both oxidative pathways suppresses estrous behavior (Schneider and Wade, 1989). Simultaneous inhibition of glucose and fatty acids oxidation decreased pulsatile secretion of LH in ovariectomized lambs (Hileman et al., 1991). These results indicate that the reproductive system may be more sensitive to total than to individual availability of metabolic fuels.

Glucose concentrations may influence ovarian function because glucose is the primary energy source used by the bovine ovary (Rabiee et al., 1997). In addition, ovarian

uptake of glucose and cholesterol are positively associated (Rabiee and Lean, 2000) suggesting that ovarian glucose uptake may influence steroidogenesis because circulating cholesterol is the main substrate for steroidogenic tissues (Gwynne and Strauss, 1982). In vitro, glucose stimulates bovine thecal cell steroidogenesis (Stewart et al., 1995). Therefore, nutritional status influences concentrations of insulin and/or glucose that can modulate reproductive function at CNS and/or ovarian tissue, even though concentrations of insulin and glucose in plasma are not good predictors of the onset of luteal activity in postpartum cows (Vizcarra et al., 1998).

Nonesterified fatty acids (NEFA). As an adaptation to negative energy balance, ruminants release nonesterified fatty acids from the adipose tissue as result of triglyceride lipolysis by hormone sensitive lipase. Glycerol and NEFA metabolism yield glucose and ketone bodies that are used as sources of energy by peripheral tissues, thus sparing circulating glucose for the central nervous system. Concentrations of NEFA increased with negative energy balance in dairy cows (Staples and Thatcher, 1990; Lucy et al., 1991) and with losses in body

weight and body condition score in beef cows (Richards et al., 1989b; Bossis et al., 1999). Beef cows that were underfed at calving had reduced LH pulse frequency at 30 d postpartum, and increased concentrations of NEFA. However, LH pulsatility and NEFA concentrations in plasma were similar in underfed and maintenance cows at 70 d postpartum (Grimard et al., 1995). Nutrient intake increased LH pulsatility by 82% without an alteration in NEFA concentrations in lambs, and lipid infusion increased NEFA concentrations without an effect on LH pulse frequency (Estienne et al., 1990). In addition, concentrations of NEFA in plasma increased during the last two follicular waves preceding resumption of ovulation during realimentation of nutritionally induced anestrous heifers (Bossis et al., 2000). Although concentrations of NEFA in plasma reflect fat mobilization and may be a good indicator of nutrient intake or energy status in beef cows, the onset of luteal activity of postpartum beef cows could not be predicted from plasma NEFA concentrations (Garmendia, 1984; Vizcarra et al., 1998). Therefore, it is unlikely that concentrations of NEFA in plasma directly affect LH secretion in ruminants.

Thyroid hormones. Thyroid gland function may be associated with reproduction in cattle. Thyroidectomy prevents the transition to seasonal anestrus and allows continuous estrous cycles in sheep (Nicholls et al., 1988). Thyroidectomized ewes maintained pulsatile secretion of LH in presense or absence of estradiol, whereas pulsatile LH secretion at the end of the breeding season was blocked by estradiol in thyroid-intact ewes (Moenter et al., 1991). Oral administration of thyroprotein during lactation of dairy cows prolonged the postpartum anestrous period (Wagner and Hansel, 1969) and induced hyperthyroidism increased the incidence of anestrous in Brahman cows (De Moraes et al., 1998). Concentrations of thyroxine were at the nadir when nonlactating cows reached nutritionally induced anestrus, although concentrations of LH and thyroxine were not related (Richards et al., 1995). In ovariectomized, nonlactating, nonpregnant Holstein cows, the presence of thyroid glands decreased basal concentrations of LH and FSH, but pulse frequency and amplitude were unaffected (Stewart et al., 1994). Hypothyroidism did not inhibit the expression of estrous behavior in ovariectomized-thyroidectomized nonlactating Holstein cows treated with estradiol benzoate with or

without progesterone (Stewart et al., 1993). Thyroid hormones may influence ovarian function. Thyroid status affected ovarian response to exogenous FSH (Bernal et al., 1999) although induced hyper- or hypothyroidism did not alter follicular dynamics and corpus luteum function in cows (De Moraes et al., 1998). Physiological concentrations of thyroid hormones enhanced LH-induced androstenedione output of bovine theca cells in vitro, which could cause an increased estradiol production by granulosa cells (Spicer et al., 2001). Dietary intake may alter concentrations of thyroid hormones in bovine serum (Beaver et al., 1989; Richards et al., 1995). Overall, a role for thyroid glands in regulation of gonadal function seems more likely for Brahman cows, which have more seasonally-influenced reproductive patterns than Bos taurus cows. The effects of thyroid hormones on reproductive function are minimal and inconsitent in Bos taurus cattle (Wagner and Hansel, 1969; Stewart et al., 1994; Richards et al., 1995).

Insulin-like growth factor-I. Insulin-like growth factor-I (IGF-I) is a mitogenic GH-dependent serum peptide with structure and functions closely related to insulin and IGF-II (Spicer and Echternkamp, 1995). The binding of IGF-I

to its cell membrane receptor in cultured cells of mesodermal origin stimulates glucose uptake and synthesis of glycogen, lipids, proteins, DNA, and cell differentiation (Froesch et al., 1985). In biological fluids, IGF-I and -II (IGFs) are normally bound to high affinity binding proteins (IGFBPs), designated as IGFBP-1 to -6 (Jones and Clemmons, 1995). The functions of IGFBPs include: 1) to prolong the half-life of IGFs in serum, 2) to prevent IGFs induced hypoglycemia, 3) to regulate the passage of IGFs from intra- to extravascular space, 4) to restrict free IGFs binding to its receptor, 5) to enhance the biological actions of IGFs by forming a slow release IGFs pool, and 6) to exert IGFs-independent, cellular actions (Collett-Solberg and Cohen, 2000). In addition to endocrine effects of IGFs, locally produced IGFs have paracrine, as well as autocrine effects on cell proliferation (Jones and Clemmons, 1995). The IGF system also includes the IGFBP proteases, which may provide a mechanism to increase the availability of free IGFs to cell receptors by degradation of IGFBPs (Maile and Holly, 1999).

The IGF type I receptor (IGF-IR) is present in tissues directly involved with reproductive function. A high density of IGF-IR has been detected in the median eminence

as well as the choroid plexus, the olfactory bulb, and the suprachiasmatic nucleus of the brain in rats (Lesniak et al., 1988; Hiney et al., 1996), indicating a neuromodulatory role of IGF-I in specific areas of the brain. Incubation of GT1-GnRH neuronal cells with 10 ng/mL of IGF-I stimulated GnRH gene expression (Longo et al., 1998) and produced a two-fold increase in GnRH secretion at two h after treatment (Anderson et al., 1999). Treatment of median eminence fragments of prepubertal female rats with IGF-I elicited a dose-related increase in GnRH (Hiney et al., 1991). Moreover, IGF-I administration may decrease hypersensitivity of the hypothalamic-pituitary axis to estradiol negative feedback and hastened the onset of puberty by 4 months in female monkeys (Wilson, 1995). The pituitary gland may also be affected by IGF-I because labeled IGF-I binding activity and IGF-IR mRNA were detected in the ovine pituitary gland (Adam et al., 2000). Output of LH from rat (Soldani et al., 1995) and ovine (Adam et al., 2000) pituitary cells is augmented by IGF-I in vitro. Concentrations of IGFBP in the bovine pituitary gland, but not in serum, changed during the estrous cycle and were positively related with progesterone concentrations (Funston et al., 1995a), and inversely

related to serum concentrations of LH. During the estrous cycle, pituitary and serum concentrations of IGF-I did not vary. Thus changes in pituitary concentrations of IGFBPs may alter availability of IGF-I in the pituitary gland of cows during the estrous cycle. Roberts et al. (2001) suggested that IGFBPs could act within the pituitary mediating endocrine feedback mechanisms that regulate gonadotrope function.

Receptors for IGF-I are also present in bovine follicular cells and oocytes. Bovine granulosa and theca layers express the gene for IGF-IR and synthesize IGF-IR. A greater number of IGF-IR are located in granulosa than theca cells (Stewart et al., 1996; Perks et al., 1999; Armstrong et al., 2000). IGF-I interacts synergistically with gonadotropins to promote growth and differentiation of ovarian follicular cells in several mammalian species (Monget and Monniaux, 1995; Spicer and Echternkamp, 1995; Adashi, 1998). Acting alone (Gutierrez et al., 1997a) or in combination with FSH (Spicer et al., 1993b) or LH (Stewart et al., 1995), IGF-I stimulated mitogenesis, progesterone production, and aromatase activity in bovine granulosa cells, as well as mitogenesis, progesterone and androstenedione production, and LH binding in theca cells.

In immature rat granulosa cells, the IGF-I/FSH synergism enhanced expression of the FSH receptor, possibly by prolonging FSH receptor mRNA stability in vitro (Minegishi et al., 2000). Overall, the interactions between IGF-I and gonadotropins seem to be determinants of growth and steroidogenic capacity of bovine ovarian follicles. IGF-I may also be involved in oocyte maturation, since oocytes collected from bovine preantral and antral follicles express mRNA for IGF-RI (Armstrong et al., 2001).

Negative energy balance during early lactation (Spicer et al., 1990; Vicini et al., 1991; Sharma et al., 1994), chronic (Richards et al., 1991, 1995; Bossis et al., 1999) or acute restriction of nutrient intake (Armstrong et al., 1993; Armstrong et al., 2001; White et al., 2001), and 48-h fasting (Spicer et al., 1992; Amstalden et al., 2000) reduce plasma concentrations of IGF-I in cattle. Reduced nutrient intake uncouples the GH-IGF-I endocrine axis (Thissen et al., 1994). Cattle fed restricted diets have increased GH secretion (Thomas et al., 1990; Breier and Gluckman, 1991; Armstrong et al., 1993; Bossis et al., 1999) whereas serum concentrations of IGF-I and hepatic IGF-I mRNA are decreased (Vandehaar et al., 1995), probably due to an insulin-dependent (Thissen et al., 1994;

Kobayashi et al., 1999; Butler and Butler, 2001) down-regulation of the GH receptor (Breier and Gluckman, 1991; Vandehaar et al., 1995). Concentrations of IGF-I in plasma increased in association with onset of puberty (Renaville et al., 1993; Yelich et al., 1996) and with the resumption of ovarian activity in postpartum (Roberts et al., 1997; Beam and Butler, 1997, 1998; Stagg et al., 1998) and nutritionally induced anestrous cows (Bossis et al., 2000). Concentrations of IGF-I in plasma are positively related to body energy reserves and nutrient intake (Rutter et al., 1989; Bishop et al., 1994; Yelich et al., 1996; Armstrong et al., 2001) and increased nutrient intake promotes the development of ovulatory follicles in prepubertal heifers (Bergfeld et al., 1994) and anestrous cows (Jolly, 1995; Stagg et al., 1995; Bossis et al., 2000). Intraovarian concentrations of IGF-I did not change with increased nutrient intake (Spicer et al., 1991, 1992) or days postpartum (Spicer et al., 1988) indicating that total amount of IGF-I in follicular fluid is not strongly regulated by nutrient supply. However, the availability of biologically active IGF-I in serum is influenced by nutrition because relative amounts of IGFBPs in serum (McGuire et al., 1992; Thissen et al., 1994; Vandehaar et

al., 1995), hypothalamus, pituitary (Snyder et al., 1999), and ovarian follicles (Armstrong et al., 2001) change with nutritional status. Ovarian inactivity in postpartum beef cows is probably caused by inadequate gonadotropin support (Wettemann, 1980) to allow early dominant follicles to produce adequate amounts of estradiol to induce ovulation (Murphy et al., 1990; Stagg et al., 1995; Duffy et al., 2000). Changes in energy status of cows have been directly associated with concentrations of IGF-I and estradiol in serum and the ability of dominant follicles to ovulate (Beam and Butler, 1999; Bossis et al., 2000). Concentrations of IGF-I in serum may be a major determinant of IGF-I concentrations in the ovary (Leeuwenberg et al., 1996) and infusion of an IGF-I analog into the ovarian artery increased estradiol secretion during the follicular phase of ewes with autotransplanted ovaries (Scaramuzzi et al., 1999). Increased nutrient intake postpartum could stimulate hepatic IGF-I secretion. Systemic IGF-I, and/or IGF-I produced locally by tissues, may influence the brain to increase gonadotropin secretion, and/or IGF-I may act synergistically with gonadotropins to enhance follicular steroidogenisis and ovarian function.

Insulin-like growth factor-II. Actions of IGF-II are also mediated through the IGF type I receptor (Adashi et al., 1989; LeRoith et al., 1995). Bovine ovarian cells contain IGF-II mRNA. Expression of mRNA for IGF-II occurs theca cells whereas weak or no expression is detected in granulosa cells (Armstrong et al., 2000, 2001; Schams et al., 2002). In luteal cells, IGF-II mRNA expression was maximal during the early luteal phase (Schams et al., 2002), but this was not observed by Woad et al.(2000). An intrapituitary IGF system exists in ewes, but in situ hybridization signals for IGF-II mRNA were not as intense as for IGF-I mRNA (Adam et al., 2000). Intrafollicular concentrations of IGF-II were greater in dominant than in small antral follicles collected during the first follicular wave of cyclic cows (Stewart et al., 1996). Insulin like growth factor-II may not affect FSH-induced aromatase activity (Spicer et al., 1993a) and may inhibit insulin-induced aromatase activity in small and large bovine granulosa cells (Spicer et al., 1994). Intrafollicular concentrations of IGF-II were reduced in ewes fed to maintain weight compared with those fed to gain weight (O'Callaghan et al., 2000). IGF-II has not been extensively studied as IGF-I, however, IGF-II may be

involved in selection of the dominant follicle (Yuan et al., 1998), vascularization of luteal tissue (Schams et al., 2002), and/or production of luteal progesterone (Maciel et al., 2001).

Leptin. Leptin, a protein encoded by the OB gene (Zhang et al., 1994) and primarily secreted by adipocytes, has profound effects on feed intake, metabolism, and neuroendocrine secretion (Houseknecht et al., 1998a; Barb, 1999; Ahima and Flier, 2000). Leptin may also regulate reproductive functions (Barash et al., 1996; Finn et al., 1998; Cunningham et al., 1999; Keisler et al., 1999). Leptin alters endocrine functions in rodents and farm animals, by influencing secretion of hormones from the anterior pituitary (Ahima et al., 1996, Yu et al., 1997; Barb et al., 1998), ovary (Spicer and Francisco, 1997, 1998), and adrenal gland (Heiman et al., 1999). Leptin receptors are localized in several reproductive tissues (for review see: Houseknecht and Portocarrero, 1998; Spicer 2001) and brain of rats (Zamorano et al., 1997), monkeys (Finn et al., 1998) and sheep (Dyer et al., 1997). The large form of leptin receptor mRNA, or protein, was detected in several hypothalamic areas including the

arcuate nuclei, which has been implicated in the regulation of feed intake and reproduction of rats (Kalra et al., 1998). Intracerebral administration of leptin in sheep reduced feed intake and also suppressed LH pulse frequency (Henry et al., 1999; Blache et al., 2000b; Morrison et al., 2001). However, the decreased LH pulsatility could be a consequence of feed intake reduction rather than a direct action on GnRH neurons (Blache et al., 2000a). In nonruminants, leptin treatment stimulates the secretion of gonadotropins (Barash et al., 1996), prevents the delay in puberty induced by food restriction (Cheung et al., 1997) and advances the onset of puberty (Chehab et al., 1997). In addition, treatment with leptin prevented the suppression of estrous cycles (Ahima et al., 1996; Schneider et al., 1998) and pulsatile secretion of LH (Ahima et al., 1996; Nagatani et al., 1998; Finn et al., 1998) induced by fasting in nonruminants. In contrast, cows fasted for 48 h had significant reductions in leptin mRNA in adipose tissue and plasma concentrations of leptin (Tsuchiya et al., 1998; Amstalden et al., 2000), but LH concentrations and amplitude of LH pulses were not changed (Amstalden et al., 2000). Short-term nutritional deprivation may be less disruptive to the reproductive axis in ruminants than

nonruminants. The rumen may act as a buffer to delay the rapid decreased in LH pulse frequency that occurs in nonruminants during fasting. Exogenous leptin prevented the fasting-induced decline in LH pulse frequency of steroid-implanted wethers (Nagatani et al., 2000) but central infusion of leptin did not increase LH pulsatility in well-fed ewes (Henry et al., 1999). This indicates a permissive role of leptin in modulation of LH secretion. Perhaps decreased concentrations of leptin may coordinate neuroendocrine events that partition energy availility away from nonprioritized functions, such as reproduction, during early lactation period.

Reproductive function in cows is strongly influenced by body fat stores since lean cows reproduce less efficiently than cows with moderate body fat (Selk et al., 1988; Randel, 1990; Short et al., 1990). An important component of this fat-reproduction relationship is a hormone or signal that reflects the amount of fat stored in the body. Concentrations of leptin in plasma are linearly related to the amount of body fat in humans (Considine et al., 1996; Ostlund et al., 1996) and rodents (Maffei et al., 1995; Schneider et al., 2000). The same relationship may exist in ruminants (Blache et al., 2000b; Delavaud et

al., 2000; Ehrhardt et al., 2000), but there is not adequate information to confirm that presumption. Leptin may serve as a signal of body fat stores to the central nervous system and therefore, it may act as an endocrine link between nutritional status and reproductive function. Mobilization of body fat reserves during the early postpartum period prolongs the postpartum anestrous period (Roche et al., 2000). Concentrations of leptin were reduced in dairy cows during the first eigth wk after calving, were negatively correlated with plasma concentrations of growth hormone and NEFA, and were positively correlated with plasma concentrations of glucose and insulin (Block et al., 2001). Days from parturition to the nadir of leptin concentrations were directly associated with days to first ovulation, suggesting that a delay in recovery of leptin secretion may prolong postpartum anestrus (Kadokawa et al., 2000). Thus, increased concentrations of leptin in plasma may stimulate an early return to reproductive activity in postpartum cows. Plane of nutrition affects leptin mRNA synthesis (Tsuchiya et al., 1998; Amstalden et al., 2000) and plasma concentrations of leptin in cattle and sheep (Ehrhardt et al., 2000; Marie et al., 2001). Nutritional management can be used to stimulate leptin secretion in

vivo to test if increased concentrations of leptin have a key role in the resumption of ovulation in postpartum beef cows. Feed restriction, which is associated with decreased concentrations of leptin in plasma, up-regulates the expression of leptin receptor in the arcuate and ventromedial hypothalamic nuclei in ewes (Dyer et al., 1997) possibly to stimulate feed intake.

Summary

Endocrine functions that control energy metabolism preceding the first ovulation postpartum in primiparous cows are not completely defined. The roles of plasma concentrations of IGF-I and leptin during the reestablishment of ovarian activity should be investigated. Prepartum and postpartum nutrient intake may interact to differentially influence energy status, estrous activity, ovarian function and pregnancy rate of cows at the first estrus. Concentrations of steroids in follicular fluid should reflect steroidogenic capacity of preovulatory follicles immediately before the first ovulation. Therefore, the objectives of this research are: 1) to determine the effects of BCS at calving and postpartum nutrient intake on estrous behavior, ovarian function,

pregnancy rate at first estrus, and concentrations of IGF-I, leptin, NEFA, glucose, insulin, and thyroxine in primiparous beef cows; 2) to explore potential relationships between postpartum endocrine and reproductive functions, and 3) to determine the effect of nutrition on concentrations of steroids in follicular fluid aspirated at the first estrus and luteal function during the subsequent estrous cycle.

CHAPTER III

Influence of Body Condition at Calving and Postpartum Nutrition on Endocrine Function and Reproductive Performance of Primiparous Beef Cows

ABSTRACT

The influences of body condition score (BCS) at calving and postpartum nutrition on endocrine and ovarian function, and reproductive performance were determined by randomly allocating thin (BCS 4.4 ± 0.1) or moderate (BCS 5.5 ± 0.1) Angus x Hereford primiparous cows to receive either one of two nutritional treatments after calving. Cows were group-fed and targeted to gain 0.45 kg/d (M, n=17) or 0.90 kg/d (H, n=17) for the first (mean ± SD) 71 ± 17 d postpartum. Then, all cows were fed the M diet until the first estrus. A replication (M, n=25; H, n=23) was used to determine pregnancy rate. Concentrations of IGF-I, leptin, insulin, glucose, NEFA, and thyroxine (T_4) were quantified in plasma samples collected weekly during treatment and during 7 wk before the first estrus. Estrous behavior was detected by

radiotelemetry and ovulation was determined using plasma progesterone. All cows were AI between 14 and 20 h after onset of estrus, and pregnancy status was determine at 35 to 55 d post-AI by ultrasonography. Cows that calved with a BCS of 4 or 5 had similar endocrine function and reproductive performance at the first estrus. During treatment, H cows gained BW and BCS ($P < 0.01$), and had greater ($P < 0.05$) concentrations of IGF-I, leptin, insulin, glucose, and T_4 in plasma than M cows. However, during the 7 wk before the first estrus, plasma concentrations of IGF-I, leptin, insulin, glucose, NEFA, and T_4 were not affected by time. Cows previously on the H treatment had a shorter ($P < 0.01$) interval to first postpartum estrus and ovulation, and a larger dominant follicle ($P < 0.01$) at first estrus, than M cows, but duration and intensity of estrus were not influenced by nutrient intake. Pregnancy rate from AI at the first estrus was greater ($P < 0.03$) for H (76%, n=38) than for M (57%, n=33) cows. In summary, postpartum nutrient intake, but not BCS (4 or 5), affected endocrine and reproductive function of primiparous beef cows. Increased nutrient intake after calving stimulated secretion of anabolic hormones, promoted fat tissue deposition during the early postpartum period, shortened the postpartum interval, and increased pregnancy

rate at the first estrus. Concentrations of IGF-I and leptin in plasma were constant during 7 wk before the first estrus, indicating that acute changes in these hormones are not limiting factors for resumption of ovarian function in primiparous beef cows.

Key Words: Postpartum, Nutrition, IGF-I, Leptin, Fertility, Beef cows

Introduction

Optimal reproductive performance in beef cows is often limited by prolonged postpartum anestrous periods. Heifers bred to calve at 2-yr of age resume ovarian function 20 to 40 d later than mature cows (Wiltbank, 1970). Stress of calving and the combined effects of growth and first lactation impose nutritional requirements that are often not fulfilled when cows graze low quality pastures. Thus, inadequate nutrient intake before (Bellows et al., 1982) or after calving (Grimard et al., 1995) has greater detrimental effects on postpartum reproduction in primiparous than in mature cows.

Suckling (Williams, 1990; Stagg et al., 1998) and nutrition (Selk et al., 1988; Randel, 1990) are major regulators of the length of postpartum anestrous period.

Restricted nutrient intake prepartum results in thin cows at calving, a prolonged postpartum anestrous period, and less cows in estrus during the breeding season (Dunn and Kaltenbach, 1980; Richards et al., 1986; Spitzer et al., 1995). Greater postpartum nutrient intake can enhance LH pulsatility and follicular growth (Perry et al., 1991a; Grimard et al., 1995), and effects of nutrition on reproduction may be more pronounced in thin cows than in cows with adequate BCS (Dunn and Kaltenbach, 1980; Richards et al., 1986; Spitzer et al., 1995).

Metabolites and metabolic hormones could mediate the effects of nutrient intake on reproductive function (Keisler and Lucy, 1996, Wettemann and Bossis, 2000). In postpartum beef cows, the roles of plasma concentrations of IGF-I (Stagg et al., 1998, Bossis et al., 2000) and leptin (Nagatani et al., 1998, Cunningham et al., 1999), in regulation of resumption of ovulation are not established. Therefore, this study was designed to determine the effects of BCS at calving and postpartum nutrient intake on endocrine and ovarian function, and reproductive performance at the first postpartum estrus of primiparous suckled beef cows.

Materials and Methods

Animals, diets, and treatments. Hereford and Hereford x Angus primiparous cows maintained on pasture were studied in two non-consecutive years (1998 = YEAR 1, n = 34; 2000 = YEAR 2, n = 49). During the last third of gestation, cows were supplemented with either 0.9 or 1.8 kg/d of a 40% CP soybean meal-based supplement, so that they would calve with a body condition score (BCS: 1 = emaciated; 9 = obese; Wagner et al., 1988) between 4 and 5. At calving, in February and March, cows were stratified by body condition and calving date and randomly assigned to one of two nutritional treatments for the first (mean ± SD) 71 ± 17 d postpartum. Late calving cows were on nutritional treatments for at least 45 d before the end of the feeding period (May 19). Cows were group-fed and targeted to gain 0.45 kg/d (Moderate = M) or 0.90 kg/d (High = H). During treatments, cows were offered prairie hay (4% CP) ad libitum. Moderate cows were supplemented with 2 kg/d of 38% CP range cubes whereas H cows had free access to a high-energy feed (1.61 Mcal NE_m/kg DM, 0.90 Mcal NE_g/kg DM, and 11.1% CP). The ration was composed (% DM) of rolled corn (39.7%), ground alfalfa pellets (35.5%), cottonseed hulls (22%), cane molasses (2.5%) and salt (0.3%). Cows on the high-energy feed consumed on average of 16 kg/head/d. After

the end of nutritional treatments, all cows were maintained in the same pasture and fed the M diet until detected in estrus. Body weight and BCS were determined monthly, from 90 d before to 150 after parturition, after cows were denied access to feed and water for 16 h. The last BCS recorded prior to calving was assigned as BCS at calving. The first weight recorded after calving was used to determine BW changes during treatments. Calves were weighed within 48 h of birth and at 30-d intervals until weaning. Calves remained with cows and were denied access to feed and water for 16 h prior to weighing.

Blood sampling and hormone and metabolite assays. Blood samples were obtained three (Monday, Wednesday, and Friday) or two (Tuesday and Thursday) times a week during YEAR 1 and YEAR 2, respectively. Samples were collected from 30 d after calving to 3 wk after the first estrus or 23 wk after calving for estrous and anestrous cows, respectively. Cows had access to feed prior to sampling. Caudal vein blood was collected in vacutainers (10 mL) containing EDTA (0.1 ml of a 15% solution). Tubes were immediately placed on ice, centrifuged (2500 x g for 15 min) at 4 °C within 3 h after collection, and plasma was

recovered and stored at -20 °C until hormones and metabolites were quantified.

Metabolic hormones and metabolites were quantified in samples collected during YEAR 1. Concentrations of insulin, IGF-I, leptin, thyroxine (T_4), glucose, and NEFA were determined in weekly samples collected from 3 wk before the end of the nutritional treatment to the week of first estrus. Concentrations of insulin in plasma were quantified with a solid phase RIA for human insulin (Coat-A-Count Insulin kit, Diagnostic Products Corp., Los Angeles, CA; Bossis et al., 1999) using bovine pancreatic insulin for standards (Sigma Chemical Co., St. Louis, MO) and 0.2 mL sample volume. Intra- and interassay CV (n = 7 assays) were 12 and 17%, respectively. Concentrations of IGF-I in plasma were quantified by RIA (Echternkamp et al., 1990) after acid-ethanol extraction (16 h at 4 °C). Intra- and inter-assay CV (n = 3 assays) were 11 and 14%, respectively. Concentrations of leptin in plasma were determined by RIA specific for ovine leptin and validated for use in bovine serum (Delavaud et al., 2000). Concentrations of T_4 in plasma were quantified with a solid phase RIA for human T_4 (Coat-A-count Total T4 kit, Diagnostic Products Corp.). Sensitivity of the assay was 10 ng/mL of plasma and the

addition of 16 ng of T_4 to 1 mL of plasma resulted in 95% recovery (n=4). When 5, 10, 15, 20, and 25 μL of bovine plasma were assayed, concentrations of T4 were parallel to those of the standard curve. The intraassay CV was 23%. Concentrations of glucose in plasma were determined with an enzymatic colorimetric procedure (no. 510, Sigma Chemical Co.) and intra- and interassay CV (n = 7 assays) were 3 and 7%, respectively. Concentrations of NEFA in plasma were quantified with a modified (McCutcheon and Bauman, 1986) colorimetric procedure (Wako-NEFA C, Wako Chemicals USA Inc., Dallas, TX). Inter- and intrassay CV (n = 7 assays) were 7 and 5.5%, respectively. Plasma concentrations of progesterone were quantified with a solid phase RIA (Coat-A-Count Progesterone kit, Diagnostic Products Corp.; Vizcarra et al., 1997). Ovulation at the first estrus was confirmed by at least two consecutive plasma samples with concentrations of progesterone greater than 0.5 ng/mL. Plasma concentrations of estradiol 17-β were determined by RIA (Estradiol MAIA, Polymedco Inc., New York, NY) with modifications (Vizcarra et al., 1997). Estradiol 17-β concentrations were quantified in plasma samples collected at 18 to 30 h prior to the onset of the first estrus followed by ovulation.

Estrous behavior, ovarian function, and reproductive performance. The number of mounts received by cows was continuously monitored using a radiotelimetric pressure-sensitive device (HeatWatch, DDx Inc., Denver, CO) attached to the rump of cows at 30d postpartum. Date, time, and duration of each mount received were recorded and used to calculate the total number of mounts received and duration of estrus for each cow. Onset of estrus was defined as the first of two mounts received within 4 h. The end of estrus was defined as the last mount received with a mount 4 h before and with no mount during the next 12 h. Concentrations of progesterone were used to determine the incidence of luteal activity before and after the first postpartum estrus during YEAR 1. Luteal phases were classified as normal if at least 5 consecutive plasma samples (or at least 10 consecutive days) had at least 0.5 ng/mL of progesterone, otherwise they were classified as short luteal phases. Size of the dominant follicle (DF) was measured (YEAR 1 and 2), between 4 to 14 h after onset of estrus, by transrectal ultrasonography (Aloka 500-V ultrasound equipment with a 7.5-MHz probe; Corometrics Medical Systems, Wallingford, CT). Ovarian images were recorded on videotape. Size of the DF was the mean of the length and width of the largest follicle. Duration of

postpartum anestrus, anovulatory interval (PPI, YEAR 1 and 2) was the number of days from calving to first estrus and ovulation. Cows that remained anovulatory and anestrus (H = 1 vs M = 9) at 23 wk postpartum were assigned a PPI of 168 d (1 wk after the end of blood sampling) and were included in the statistical analyses. All cows were artificially inseminated between 14 and 20 h after onset of estrus by a single technician using semen from one bull in each year. Pregnancy status was determine at 35 to 55 d post-AI by ultrasonography and confirmed by calving date.

Statistical analyses. Changes in BCS and BW, number of mounts received, duration of estrus, PPI, and maximum diameter of DF were analyzed as a randomized complete block (year) design with a 2 x 2 treatment structure using a mixed model (PROC MIXED; Little et al., 1996) with SAS (SAS Inst. Inc., Cary, NC). Model included year as a random effect, BCS at calving (4 or 5), postpartum nutrition (H or M), the first order interaction as fixed effects, and days on nutritional treatment as a covariable. All effects were tested with the pooled residual error term. Calf growth and weaning weights were analyzed adding calf sex and all interactions to the previous model. Weaning weights were adjusted to 205 d of age before analysis. Birth weights were analyzed using a mixed model that included year as a

random effect, and BCS at calving, postpartum nutrition, calf sex, and all possible interactions as fixed effects. Proestrus concentrations of estradiol were analyzed as a completely randomized design with nutritional treatment, time of sampling (18 or 30 h) prior to first estrus, and its interaction as fixed effects with the GLM procedure of SAS.

Concentrations of metabolites and metabolic hormones across time postpartum were analyzed using generalized least squares and a mixed model for a randomized complete block (assay) design with repeated measures over the same experimental unit using the MIXED procedure of SAS. All samples for 2 or 3 cows per treatment were included in an assay, and samples were randomly distributed within assay. The statistical model included assay as a random effect and BCS at calving, PPN, week postpartum, and all first and second order interactions as fixed effects. Days on nutritional treatment were used as a covariable. Cow within BCS x nutritional treatment was used as error term to test treatment effects (BCS, nutritional treatment, and BCS x nutritional treatment) whereas the pooled residual was the error term to test the week effect and all interactions with week. Degrees of freedom for the pooled error term were calculated using Kenward-Roger's approximation. A

first order autoregressive function with lag equal to one was used to model the covariance structure for the repeated measures. If a significant treatment x week postpartum interaction was detected, simple effects of treatment were compared using the SLICE option of the LSMEANS statement of SAS. Pearson correlation coefficients (PROC CORR; SAS) were calculated to describe linear relationships among response variables. Logistic regression analysis was used to compare pregnancy rates. The model included year, BCS at calving, nutritional treatment, and all first order interactions as predictors, and number of cows pregnant over those inseminated at first estrus as the dependent variable. The model was fitted using the GENMOD procedure of SAS with logit as a link function and assuming a binomial distribution for the error term.

Results

Body condition score, body weights, and calf performance. Prepartum feeding management was effective to achieve a 4 or 5 BCS at calving ($P < 0.01$, Table 1). Body condition score was similar ($P = 0.60$) for cows on M or H diets at the beginning of postpartum treatments, and greater nutrient intake postpartum increased ($P < 0.01$) BCS at the end of feeding, independently ($P = 0.64$) of BCS at

calving. Cows on the M diet lost an average 0.26 BCS whereas cows on the H diet gained 0.45 of a BCS. Average body weight before calving did not differ ($P = 0.80$) among cows assigned to different postpartum nutritional treatments. Body weight changes after calving were similar to changes in BCS (Table 1). The effect of postpartum nutrition on BW change was independent ($P = 0.27$) of BCS at calving. Body condition score at calving did not influence ($P = 0.97$) change in BW during the feeding period, and H cows had a greater ADG during treatment than M cows (1.14 ± 0.10 vs 0.35 ± 0.10 kg/d, respectively, $P < 0.01$). Calf birth weights (33.3 ± 1.6 kg) were not influenced by BCS at calving (Table 2). However, calves that suckled H cows were heavier ($P < 0.01$) at the end of feeding than M calves. Average daily gain was 0.25 kg/d greater ($P < 0.01$) for calves that suckled H cows compared with those that suckled M cows. Calf weight at the end of feeding was directly related to change in BW of the cow during the same time period ($r = 0.56$; $P < 0.01$). Calves weaned from H cows were 10 kg heavier ($P < 0.05$) than those weaned from M cows.

Endocrine function during and after nutritional treatments. Concentrations of IGF-I in plasma during 3 wk before and 3 wk after the end of nutritional treatment were

greater ($P < 0.01$) for H cows (29.8 ± 1.7 ng/mL) than M cows (20.2 ± 2.0 ng/mL; Figure 1A). The end of nutritional treatment was an average of 9 wk (range: 4-12 wk) prior to first estrus. Neither BCS at calving ($P = 0.26$), week ($P = 0.61$), nor treatment x week ($P = 0.21$) affected concentrations of IGF-I in plasma.

There was a treatment x week effect ($P < 0.01$) for plasma concentrations of leptin, insulin, and glucose during the 3 wk before and 3 wk after treatment (Figure 1B, 1C, and 1D, respectively). Cows on H treatment had greater concentrations of leptin, insulin and glucose compared with M cows during the last 3 wk of nutritional treatment. Concentrations of leptin, insulin, and glucose for H vs M cows during the last 3 wk of feeding averaged 4.97 ± 0.6 vs 1.69 ± 0.7 ng/mL ($P < 0.01$, Figure 1B); 1.56 ± 0.1 vs 0.97 ± 0.1 ng/mL ($P < 0.01$, Figure 1C); and 68.6 ± 2.0 vs 62.0 ± 2.2 mg/dL ($P < 0.05$, Figure 1D), respectively. Concentrations of leptin, and insulin were similar in H and M cows during the first 3 wk after treatment when all cows were fed the M-diet. Concentrations of glucose were greater ($P < 0.05$) for H cows than for M cows at the first wk after treatment, thereafter concentrations of glucose were similar for H and M cows.

There was a treatment x week effect ($P < 0.01$) on plasma concentrations of NEFA during the 3 wk before and 3 wk after treatment (Figure 1E). Concentrations of NEFA were similar for H and M cows during treatment. However, concentrations of NEFA in plasma were greater ($P < 0.01$) for H than for M cows during the first 3 wk after the end of nutritional treatment (510 ± 30 vs 264 ± 30 μEq/mL, respectively).

There was a treatment x week effect ($P < 0.01$) on concentrations of T_4 in plasma during the last 3 wk before and 3 wk after nutritional treatment (Figure 1F). During the 3 wk of treatment and the first wk after treatment, plasma concentrations of T_4 were greater ($P < 0.01$) for H than for M cows (41.0 ± 2.3 vs 25.0 ± 2.4 ng/mL, respectively). However, concentrations of T_4 were similar for H and M cows during 2 and 3 wk after treatment.

Partial correlation coefficients, adjusted for cow, for concentrations of hormones and metabolites during the last 3 wk of nutritional treatment are in Table 3. Concentrations of IGF-I were positively correlated with concentrations of leptin ($P < 0.05$), insulin ($P < 0.01$) and glucose ($P < 0.01$). Concentrations of leptin were positively correlated with concentrations of insulin ($P <$

0.01), and glucose ($P < 0.05$). Concentrations of NEFA were not correlated with any of the hormones or metabolic compounds quantified. Concentrations of T_4 were positively ($P < 0.01$) correlated with concentrations of IGF-I, leptin, insulin, and glucose.

Endocrine function before the first postpartum estrus. There was not a significant ($P = 0.97$) treatment x BCS at calving x week effect on concentrations of IGF-I in plasma during 7 wk before the first estrus. There was a nutritional treatment x BCS at calving effect ($P < 0.03$) on concentrations of IGF-I in plasma. Concentrations of IGF-I during the 7 wk prior to the first estrus did not differ ($P > 0.60$) between cows that calved with BCS 4 and were on M (24.9 ± 2.6 ng/mL) or H (23.3 ± 2.4 ng/mL) nutrition after calving, but concentrations of IGF-I during 7 wk prior to first estrus were greater ($P < 0.01$) for H (35.3 ± 2.3 ng/mL) than for M cows (26.1 ± 2.1 ng/mL) when they calved with a BCS 5. Concentrations of IGF-I in plasma tended to vary ($P < 0.06$) with week before the first estrus, independently of BCS at calving ($P = 0.48$) and postpartum nutritional treatments ($P = 0.12$). Concentrations of IGF-I did not change ($P = 0.45$) during the last 4 wk before the first estrus (Figure 2A).

Concentrations of leptin during 7 wk before the first estrus were not affected by treatment x BCS at calving x week ($P = 0.29$), treatment x week ($P = 0.53$), treatment x BCS at calving ($P = 0.52$), BCS at calving x week ($P = 0.52$), BCS at calving (1.65 ± 0.3 vs 2.33 ± 0.3 ng/mL, $P = 0.11$, 4 vs 5, respectively), treatment (2.28 ± 0.3 vs 1.70 ± 0.3 ng/mL, $P = 0.18$, H vs M, respectively), or week ($P = 0.12$) before first estrus (Figure 2B).

Concentrations of insulin (Figure 2C) and glucose (Figure 2D) during 7 wk before the first estrus were not affected by treatment x BCS at calving x week ($P > 0.59$), treatment x week ($P > 0.35$), BCS at calving x week ($P > 0.63$), or treatment x BCS at calving ($P > 0.25$). Mean concentrations of insulin in plasma during 7 wk before first estrus were greater ($P < 0.05$) for cows with a BCS 5 at calving (1.08 ± 0.08 ng/mL) than for those with a BCS 4 (0.97 ± 0.08 ng/mL). Postpartum nutrition did not affect ($P = 0.45$) concentrations of insulin prior to estrus (1.01 ± 0.08 ng/mL and 1.04 ± 0.08 ng/mL, for M and H cows, respectively). Concentrations of glucose in plasma did not differ ($P = 0.30$) between cows that calved with BCS 4 or 5 (61.5 ± 1.5 mg/dL and 62.5 ± 1.4 mg/dL, respectively). Cows that were previously on H nutrition (63.4 ± 1.4 mg/dL) had

greater ($P < 0.01$) concentrations of glucose than those on M nutrition (60.6 ± 1.4 mg/dL). There was a tendency ($P < 0.08$) for week postpartum to influence concentrations of insulin and glucose before the first estrus.

Concentrations of NEFA in plasma before first estrus were not affected by treatment x BCS at calving x week ($P = 0.28$), treatment x BCS at calving ($P = 0.95$), BCS at calving x week ($P = 0.76$), and treatment x week ($P = 0.15$). Concentrations of NEFA in plasma varied ($P < 0.05$) in magnitude and direction with week before first estrus, but there was no distinct time trend (Figure 2E). Concentrations of NEFA in plasma prior to first estrus were not affected ($P = 0.72$) by BCS (268 ± 7 vs 265 ± 7 μEq/mL; BCS 4 vs 5, respectively). Mean concentrations of NEFA during 7 wk before first estrus were greater ($P < 0.01$) in H cows (293 ± 7 μEq/mL) than in M cows (240 ± 7 μEq/mL).

Concentrations of T_4 in plasma prior to first estrus were not affected by treatment x BCS at calving x week ($P = 0.53$). Week ($P = 0.78$) and the first-order interactions of main effects with week did not affect ($P > 0.25$) concentrations of T_4 in plasma prior to estrus (Figure 2F). There was a treatment x BCS at calving effect ($P < 0.05$) on concentrations of T_4 before first estrus. When cows calved

with BCS 4, plasma concentrations of T_4 did not differ (P =0.23) during 7 wk before first estrus for cows that were on H (29.3 ± 2.1 ng/mL) or M (26.2 ± 2.0 ng/mL) nutrition after calving. When cows calved with BCS 5, plasma concentrations of T_4 during 7 wk before first estrus were greater ($P < 0.01$) in H cows (37.6 ± 2.0 ng/mL) than in M cows (26.8 ± 1.9 ng/mL).

Estrus, ovarian function and reproductive performance. Estrus, ovarian function, and reproductive performance were not affected ($P > 0.20$) by BCS at calving and BCS at calving x postpartum nutrition treatment, thus results are summarized for the postpartum nutrition main effect (Table 4). The incidence of short luteal phases prior to first postpartum estrus was not influenced ($P > 0.70$) by postpartum nutrition. Eighty-seven percent of the cows had a transient increase of progesterone in plasma (≥ 0.5 ng/mL for less than 10 d; maximum concentration = 1.60 ± 0.20 ng/mL) within 2 to 4 d before the first estrus. After first estrus, all cows had normal luteal phases (progesterone ≥ 0.5 ng/mL for at least 10 consecutive days).

Characteristics of the first postpartum estrus were similar ($P > 0.50$) for H and M (Table 4). Average duration of the first estrus was 5.6 ± 1.2 h and cows were mounted

16.7 ± 5.1 times. Concentrations of estradiol 17-β in plasma were not affected by time ($P = 0.40$) or treatment x time ($P > 0.50$) during 2 d before estrus, and were similar at 30 h (4.15 ± 0.41 pg/mL) and 18 h (4.78 ± 0.63 pg/mL) before first estrus. Averaged over time, concentrations of estradiol 17-β were not different ($P > 0.25$) for M (3.64 ± 0.39 pg/mL) and H (4.29 ± 0.41 pg/mL) cows. Maximum diameter of the dominant follicle at the first estrus was larger ($P < 0.01$) for H cows than for M cows (Table 4). Mean concentration of estradiol 17-β in plasma at 18 to 30 h before estrus and maximum diameter of the dominant follicle at estrus were not correlated ($r = 0.16$, $P = 0.40$, $n = 28$).

The interval from calving to first estrus and ovulation (postpartum anestrous interval) was shorter ($P < 0.01$) for H than for M cows (Table 4). Only 24 % of M cows had ovulated and initiated a normal luteal phase before 80 d postpartum compared with 41 % of H cows ($P = 0.13$). When anovulatory cows (M = 9 and H = 1) were assigned an ovulation date as one wk after the last sampling date, the length of the postpartum anestrous interval was 34 d longer for M cows compared with H cows. Pregnancy rate at the first estrus was 18.7 percent units greater ($P < 0.03$) for H than for M cows (Table 4).

Discussion

Increased nutrient intake for approximately 70 d after parturition increased BCS and BW of primiparous suckled beef cows that calved with a BCS of 4 or 5. In contrast, cows that had a moderate nutrient intake lost body energy reserves and weighed less than high cows at the end of nutritional treatment. High-energy diets fed after calving (Perry et al., 1991a; Stagg et al., 1995) or before puberty (Yelich et al., 1995) increase fat deposition in mature cows and growing heifers. Primiparous beef cows fed a high- compared with a moderate-energy diet postpartum partitioned a greater proportion of net energy (consumed) to grow maternal tissue (Lalman et al., 2000). Thus, high-energy diets after calving may increase fat and tissue deposition in primiparous suckled cows that calve with a thin or moderate BCS.

Calf birth weights were not influenced by BCS at calving. Nutrient intake of first-calf cows during gestation may (Corah et al., 1975; Bellows and Short, 1978; Spitzer et al., 1995) or may not (Whittier et al., 1988b; Goehring et al., 1989; Wiley et al., 1991) influence birth weight of calves. Calf birth weights of primiparous cows with a BCS 5 were an average 1.5 kg heavier than those from cows calving with a BCS 4 (Spitzer et al., 1995). In the

present experiment, calves born from cows with a BCS 5 were only 0.5 kg heavier than those born from cows with BCS 4. Several environmental and genetic factors affect birth weight of calves (Holland and Odde, 1992), and may influence the effect of nutrient intake on birth weight. In addition, nutrient intake during gestation must be reduced drastically to reduce calf birth weight because thin cows have enhanced placental growth, which may alleviate or diminish some of the negative effects of reduced nutrient intake on fetal growth (Rasby et al., 1990).

Preweaning and adjusted 205-d weaning weights of calves were not affected by BCS at calving, which is consistent with previous results with primiparous beef cows (Whittier et al., 1988b; DeRouen et al., 1994; Spitzer et al., 1995). Minimal differences in BCS at calving may not have a significant effect on milk production and growth rate of calves. However, calves reared by thin cows (BCS 3) at calving were lighter at 105 d postpartum (Houghton et al., 1990) or at weaning (Corah et al., 1975) than those reared by well-fed cows (BCS 5).

Postpartum nutrient intake affected calf performance. Increased nutrient intake during lactation increased calf weight at the end of feeding and at 205-d of age. Postpartum energy restriction reduce calf weight at 70 d of

age (Perry et al., 1991a) as well as actual and adjusted 205-d weaning weight (Richards et al., 1986; Spitzer et al., 1995). Increased energy intake during lactation increases daily milk production (Perry et al., 1991a; Marston et al., 1995; Lalman et al., 2000). In the present study, calves suckling cows on the high-energy diet were heavier at 70 d of age and at weaning. Increased energy intake probably increased milk production as well as body energy reserves in H cows. Milk yield and weaning weights are positively correlated in beef cattle (Totusek et al., 1973; Marston et al., 1992). Alternatively, calves suckling H cows may have consumed some of the ration fed to cows, which could increase daily gain independently of the greater milk production of H cows.

Amount of nutrient intake after calving influenced concentrations of IGF-I in plasma of primiparous lactating beef cows. This is consistent with previous studies in which concentrations of IGF-I in plasma were directly related with nutrient intake in heifers (Armstrong et al., 1993; Yelich et al., 1996; Armstrong et al., 2001), primiparous (Lalman et al., 2000), and mature (Richards et al., 1991) beef cows. Reduced nutrient intake uncouples the GH-IGF-I axis (Thissen et al., 1994). Undernutrition increases GH secretion in cattle (Armstrong et al., 1993;

Bossis et al., 1999) whereas serum concentrations of IGF-I and hepatic IGF-I mRNA are decreased (Vandehaar et al., 1995), probably due to an insulin-dependant down-regulation of the GH receptor (Thissen et al., 1994; Kobayashi et al., 1999; Butler and Butler, 2001). In the present study, cows on moderate nutrition after calving lost BCS during the treatment period, which reflects an inadequate nutritional status and, probably, an uncoupled the GH-IGF-I axis. In contrast, cows on the high nutrient intake, gained BCS and weight, and had greater concentrations of IGF-I in plasma. The later occurred simultaneously with increased concentrations of insulin in plasma that may have enhanced the hepatic sensitivity to GH in H cows. This nutritionally induced increase in concentrations of IGF-I in plasma could have influenced, directly and/or indirectly, the reproductive function of H cows.

Our results are the first to demonstrate that H nutrient intake after calving stimulated secretion of leptin in plasma of lactating beef cows. A positive association between nutrient intake and concentrations of leptin in plasma has been reported in sheep (Delavaud et al., 2000; Ehrhardt et al., 2000) and cattle (Ehrhardt et al., 2000; Delavaud et al., 2002). We determine an acute decrease in concentrations of leptin in H cows within the

first week (4 ± 0.1 d) after nutritional treatment. Similarly, concentrations of leptin in plasma and in cerebrospinal fluid of sheep were influenced acutely and increased by 5 d after a change from a low- to a high-energy diet (Blache et al., 2000). These increases were probably not linked to changes in BCS or BW because they occurred in a short period of time, and indicate that nutrient intake may affect secretion of leptin in plasma independently of BCS or BW. Similar concentrations of leptin in plasma of H and M cows during the first 3 wk after treatment, when they were fed the M-diet, supports the notion that nutrient intake may exert a short-term regulation of leptin secretion without changes in body energy reserves.

Cows and heifers fasted for 48 h (Tsuchiya et al., 1998; Amstalden et al., 2000, 2002) or in negative energy balance during early lactation (Block et al., 2001), had decreased amounts of leptin mRNA in adipose tissue and concentrations of leptin in plasma. On the other hand, plasma concentrations of leptin are also highly correlated with adipocyte volume in non-lactating well-fed and underfed cows (Delavaud et al., 2002). Thus, concentrations of leptin in bovine plasma may depend on amount of adipose

tissue in the long-term, but are influenced by changes in nutrient intake in the short-term.

Several metabolic compounds could mediate the effect of nutrient intake on plasma concentrations of leptin. In the current study, plasma concentrations of insulin and glucose were greater in primiparous cows that had a high compared with moderate nutrient intake after calving, which agrees with other reports (Vizcarra et al., 1998; Lalman et al., 2000). Insulin secretion is stimulated by propionate in ruminants (Harmon, 1992) and diets with greater amount of starch, such as the H diet, increase the proportion of propionate in plasma. Insulin is secreted in response to increased concentrations of glucose in plasma, probably as a consequence of an increase in propionic acid in rumen or from postruminal starch digestion. In the present study, concentrations of leptin, insulin, and glucose were positively correlated during the last 3 wk of nutritional treatments. Concentrations of leptin in plasma are directly related to those of insulin in dairy cows (Block et al., 2001; Delavaud et al., 2002) and beef heifers (Amstalden et al., 2000, 2002). Insulin secretion in fasted heifers increased within 3 h of central infusion of leptin (Amstalden et al., 2002), and insulin increases the expression and secretion of leptin from bovine (Houseknecht

et al., 2000) and rat (Barr et al., 1997) adipose tissue in vitro. Leptin secretion in sheep did not respond to insulin administration for 2 h (Kauter et al., 2000). Insulin-induced secretion of leptin depends on uptake of glucose into rat adipocytes (Barr et al., 1997), indicating leptin secretion may be related to energy metabolism in the cell. An energy deficit in postpartum dairy cows (Block et al., 2001) or, in primiparous beef cows in this study, reduced leptin and increased NEFA concentrations in plasma. Since leptin and NEFA are secreted into circulation from adipocytes, it is probable that secretion of leptin may be coupled to fat synthesis/degradation. Lipolysis and sensitivity to catecolamines are enhanced during early lactation in dairy cows (Ingvartsen and Andersen, 2000), and stimulation of β-adrenergic receptors enhances triglycerides breakdown (Ferlay and Chilliard, 1999) and reduces concentrations of leptin in bovine plasma (Chilliard et al., 1998). Restricted energy intake increases concentrations of GH in plasma (Armstrong et al., 1993; Bossis et al., 1999), and GH reduces the stimulatory effects of insulin on leptin secretion from bovine adipose tissue culture (Houseknecht et al., 2000). Lipolysis increases secretion of NEFA in plasma, which inhibited secretion of leptin from rat adipocytes (Shintani et al.,

2000). Conversely, lipogenesis may stimulate leptin secretion because cows that had increased fat reserves in response to nutritional treatment also had increased concentrations of insulin, glucose, and leptin in plasma in this study. Our results provide evidence of a positive relationship between nutrient intake and secretion of leptin in postpartum beef cows. However, the specific roles of insulin, glucose and NEFA in modulating leptin secretion are unsolved.

Greater nutrient intake post-partum (H cows) increased T_4 and leptin concentrations in plasma, similar to the report recently for non-lactating cows (Delavaud et al., 2002). Concentrations of T_4 and leptin were positively associated during nutritional treatment. In agreement with this response, leptin administration increased pro-TSH gene expression (Legradi et al., 1997) and concentrations of T_4 (Ahima et al., 1996) in fasted rodents. Basal metabolic rate and energy expenditure are directly regulated by T_4. These results indicate that concentrations of leptin in plasma may be associated with rate of metabolism, increased secretion of anabolic hormones, and tissue accretion.

Manipulation of dietary energy intake from 45 to 70 d after parturition improved the nutritional and metabolic status of primiparous suckled beef cows. However, only 24 %

of H cows resumed ovarian function before d 80 postpartum in Year 1. Lack of adequate pulsatile secretion of GnRH and LH is the major cause of postpartum anovulation in cows (Wettemann, 1980; Butler and Smith, 1989; Short et al., 1990). This indicates that the changes in metabolic status were not sufficient to override the negative influence of other factors on the GnRH pulse generator activity. Those factors probably were thin BCS at calving and suckling.

Concentrations of IGF-I, leptin, insulin, glucose, NEFA, and T_4 did not change during the 7 wk before first estrus in cows that were previously fed H or M nutrient intake. In agreement with our results, concentrations of IGF-I in plasma did not change with time postpartum in Bos taurus cows (Spicer et al., 2002). However, plasma concentrations of IGF-I increased linearly during 75 d before first postpartum ovulation in beef cows suckled ad libitum or once daily (Stagg et al., 1998). Systemic concentrations of IGF-I in cows are directly influenced by nutrient intake (Bossis et al., 2000), BCS (Bishop et al., 1994), and energy balance (Spicer et al., 1990). In the present study, H and M cows differed in only 0.75 unit of a BCS at the end of feeding, and were on the same diet during the 7 wk before the first estrus. Perhaps both reasons may

explain why concentrations of IGF-I did not change prior to first estrus.

Leptin possibly regulates reproductive function since it acts as a metabolic signal via the brain to control pulsatile secretion of LH (Nagatani et al., 1998; Cunningham et al., 1999). However, the effects of leptin on LH secretion may depend on the animal model used. Exogenous leptin prevented the fasting-induced decline in LH pulse frequency in rats and steroid-implanted wethers (Nagatani et al., 1998, 2000). Leptin also stimulated LH pulses in chronically-underfed sheep, but central infusion of leptin did not increase LH pulsatility in well-fed ewes (Henry et al., 1999) and adequately fed cows (Amstalden et al., 2002). These results suggest that leptin may have a role in maintaining LH secretion when the nutritional insult is severe. Minimal concentrations of leptin during the early postpartum period in dairy cows may promote feed intake and divert energy from nonprioritized functions, such as reproduction (Block et al., 2001). Days from parturition to the nadir of leptin concentrations were directly associated with days to first ovulation, indicating that a delay until leptin secretion increases may prolong postpartum anestrus in dairy cows (Kadokawa et al., 2000). However, leptin did not change after 3-4 wk postpartum in dairy cows (Kadokawa

et al., 2000; Block et al., 2001). Similarly, in the present study, concentrations of leptin in plasma did not change during 7 wk before the first postpartum estrus, indicating that leptin is not the primary signal that restores cyclic ovarian function in postpartum beef cows. Greater milk production and energy intake may be the cause of increased leptin in plasma before first ovulation in high-producing dairy cows (Kadokawa et al., 2000) but not in our study with beef cows. First ovulation occurred an average at 25 d and 110 d postpartum for the dairy and beef cows, respectively, which implies that feeding management and physiological status of cows prior to first ovulation were different between experiments.

Concentrations of insulin and glucose in plasma were not influenced by time during 7 wk before first ovulation. Similarly, concentrations of insulin and glucose did not change during the last 3 follicular waves that preceded the resumption of ovulation during realimentation of nutritionally induced anestrous heifers (Bossis et al., 2000). Infusions of insulin and glucose did not alter LH secretion in postpartum beef cows (Garmendia, 1986), and basal LH concentration and LH pulse frequency were similar for control and hypoglycemic cows (Rutter and Manns, 1988). Concentrations of insulin and glucose were not predictive

of the first luteal activity in primiparous postpartum beef cows (Vizcarra et al., 1998). These results indicate that the ovulatory process in beef cows may be initiated without significant changes in insulin and glucose concentrations during several weeks prior to ovulation. However, insulin may affect the time of first ovulation by LH-independent mechanisms. Insulin stimulates proliferation, and steroidogenesis of bovine follicular cells in vitro (Spicer and Echternkamp, 1995; Webb et al., 1999). Small increases in concentrations of insulin during the postpartum period may increase responsiveness of follicular cells to gonadotropins, enhancing maturation and estradiol production by the dominant follicle and ovulation (Beam and Butler, 1999).

Increased concentrations of NEFA in plasma are indicative of lipid mobilization in cows during negative energy balance (Richards et al., 1989b; Staples and Thatcher, 1990; Bossis et al., 1999). In our study, mean plasma concentrations of NEFA were greater for H cows than for M cows during 7 wk before first estrus. The increased NEFA in H cows could be related to a greater nutrient intake and milk production since H cows weaned heavier calves, as has been reported previously for the same experimental paradigm (Vizcarra et al., 1998). Plasma NEFA

concentrations were negatively correlated with LH pulse frequency at 30 d postpartum in beef cows (Grimard et al., 1995), but NEFA are not the only endocrine signal that changes when energy balance is negative. Moreover, plasma NEFA concentrations are probably not a direct signal for the resumption of ovarian function because: 1) lipid infusion that increased NEFA concentrations by 2-fold did not affect pulsatile secretion of LH in ovariectomized lambs (Estienne et al., 1990), 2) concentrations of NEFA in plasma did not predict the resumption of ovulation in postpartum primiparous cows (Vizcarra et al., 1998) and, 3) Concentrations of NEFA in plasma increased with time during realimentation of nutritionally induced anovulatory cows prior to resumption of ovulation (Bossis et al., 2000)

Cows that calved with a BCS of 5 and received a H nutrient intake postpartum had greater concentrations of T_4 in plasma before first estrus, which probably reflected a greater metabolic rate associated with increased milk production or greater feed intake (Richards et al., 1995). However, plasma T_4 did not change during 7 wk before first estrus. Thyroid hormones may regulate gonadal function in sheep (Webster et al., 1991) and deer (Shi and Barrell, 1992), species that are seasonally polyestrus. In contrast, thyroid gland activity may not be a regulator of ovarian

function in cows. Concentrations of T_4 were at the nadir when nonlactating beef cows reached nutritionally induced anovulation, but concentrations of LH and T_4 were not correlated (Richards et al., 1995). Hypothyroidism did not interfere with the expression of estrous behavior in ovariectomized nonlactating cows treated with estradiol benzoate, with or without progesterone (Stewart et al., 1993). Induced hyper- or hypothyroidism did not alter follicular dynamics and corpus luteum function in cyclic cows (De Moraes et al., 1998). These results and ours support that concentrations of T_4 in plasma may not be limiting ovarian function in beef cows.

The first ovulation that occurs after parturition usually is not preceded by estrous behavior and is followed by a short ovarian cycle prior to the first estrus and second ovulation (Murphy et al., 1990; Perry et al., 1991b; Werth et al., 1996). The current experiment is the first to determine that BCS at calving and postpartum nutrient intake do not influence the incidence of short luteal activity before the first estrus of primiparous beef cows. In agreement with other studies (Corah et al., 1974; Odde et al., 1980), length of the estrous cycle after first postpartum estrus usually is normal and not influenced by

BCS at calving and postpartum weight gain (Looper, 1999; Lents et al., 2000).

Neither BCS at calving nor postpartum nutrition influenced estrous behavior at the first postpartum estrus. Duration of estrus and number of mounts received per estrus were highly variable, but comparable to other reports for mature lactating beef cows (Hurnik and King, 1987; Lents et al., 2000). In contrast, duration of estrus and number of mounts per estrus ranged from 14 to 17 h and 25 to 59 mounts, respectively, for non-lactating beef cows after a synchronized estrus (Floyd, 2001; White et al., 2002). These results indicate that duration and intensity of estrous behavior may differ between cyclic cows and postpartum cows at the first estrus. Possible causes of these differences can not be inferred from our experimental design, but may involve cow age, management and environmental factors, suckling stimulus and/or social factors. Estrous synchronization promotes a great number of cows in estrus simultaneously. Mounting activity and duration of estrus increase with the number of cows in estrus (Hurnik et al., 1975; Helmer and Britt, 1985; Floyd, 2001). In contrast, resumption of estrus and ovulation in postpartum cows occurs during a prolonged time period, which reduces the number of cows in estrus at the same

time. Thus, fewer cows in estrus simultaneously may be related to the reduction in duration and mounts received in lactating cows at the first postpartum estrus compared with nonlactating cyclic cows.

Similar reproductive performance for primiparous cows that calved with thin (BCS 4) or moderate (BCS 5) condition was not expected. Reproductive performance is reduced in thin mature and primiparous cows (Richards et al., 1986; Wright et al., 1987; Spitzer et al., 1995). However, if cows were bred for a shorter period after calving than the one used in this study, an effect of BCS at calving on reproductive performance might have been observed. Since young cows have additional nutritional requirements for growth during lactation, spring calving primiparous cows should calve at BCS 6 to optimize reproductive performance (DeRouen et al., 1994; Spitzer et al., 1995).

Increased nutrient intake after parturition shorted the interval from calving to first estrus and ovulation in primiparous cows. Greater nutrient intake postpartum has a positive (Wright et al., 1992; Stagg et al., 1995; Vizcarra et al., 1998) or no effect (Wright et al., 1987; Whittier et al., 1988a; Stagg et al., 1998) on duration of the postpartum anovulatory interval. Lack of consistency among studies may involve amount of energy intake, duration of

feeding period, BCS at calving, age of cows, etc. However, thin cows or primiparous cows at calving respond to increased postpartum nutrient intake with enhanced reproductive performance (Richards et al., 1986; Spitzer et al., 1995; Lalman et al., 2000), although reproductive performance may be still unacceptable. Length of the postpartum interval may depend on body fat reserves and energy intake during the postpartum period. If primiparous cows calve in thin condition and have restricted nutrient intake postpartum, resumption of ovulation will be delayed. The mechanisms underlying the effects of nutrition on reproductive performance are still unknown. In the present study, nutrient intake induced changes in plasma concentrations of IGF-I, leptin, insulin, glucose, NEFA, and T_4, which have been proposed as mediators between nutrition, reproductive function, and fat deposition. Most changes in plasma constituents occurred about 50 to 70 d postpartum, the time that is coincident with maximal milk production in beef cows (NRC, 1996). Since suckling is a major inhibitor of pulsatile LH secretion in beef cows (Williams, 1990; Stagg et al., 1998), it is possible that it suppressed reproductive function during that time period. However, increased nutrition also induced fat deposition in H cows, which may be a prerequisite to

reestablish ovarian function in postpartum cows. Increased BCS is required to resume estrous cycles in nutritionally induced anestrous cows (Richards et al., 1989a) and heifers (Bossis et al., 2000), and body energy reserves influence the interval to ovulation after early weaning of beef cows (Bishop et al., 1994). Thus, fat reserves may influence the resumption of ovarian function and could influence the inhibitory effects of suckling on LH secretion. Amount of fat reserves could be the reason why H-cows resumed ovarian function 20 to 30 d earlier than M cows. However, the signals that link energy reserves with secretion of GnRH-LH are still under intensive investigation. Concentrations of leptin in plasma are linearly related to the amount of body fat in humans (Considine et al., 1996; Ostlund et al., 1996), rodents (Maffei et al., 1995; Schneider et al., 2000), and probably in ruminants (Blache et al., 2000; Delavaud et al., 2000; Ehrhardt et al., 2000), and they can act at brain centers that control GnRH secretion (Cunningham et al., 1999). However, our results indicate that leptin may not be a major factor that controls LH secretion in postpartum beef cows because leptin concentrations did not change during 7 wk preceding the first ovulation. Another possibility is that increased plasma concentrations of leptin in H cows during increased

nutrient intake were sufficient to permit the onset of ovarian function, but other required signal(s) were not adequate to initiate ovulation or inhibitory signals were present.

Increased energy intake after parturition enhanced pregnancy rate at the first postpartum estrus. This result confirms what was suggested in previous reports (Wiltbank et al., 1964; Richards et al., 1986; Randel, 1990). Hill et al. (1970) suggested that fertilization failure, not early embryo mortality, is the cause of depressed conception rates in underfed heifers. Conversely, Spitzer et al. (1978) suggested that conception rates of energy restricted heifers are reduced by embryo mortality after 4 d after mating. Body condition score at the time of breeding (Humblot et al., 1996) or at time of oocyte recovery (Snijders et al., 2000) were positively related to pregnancy rate and embryo development in vitro, respectively. Bovine oocytes may acquire the ability to complete nuclear maturation when follicular diameter is approximately 3 mm (Fair et al., 1995). Bovine follicles could require 35 d to growth from 0.4 mm to 3.7 mm and 7 d more to attain preovulatory size (Lussier et al., 1987). Thus, any factor that affects follicular growth during that time period could also interfere with normal oocyte

maturation, and compromise subsequent embryo development. In the current study, increased nutrient intake induced endocrine and metabolic changes during 50 to 70 d postpartum, that might have acted directly on the ovary to influence fertility of H cows at the first estrus. Undernutrition during the early postpartum period may influence preantral follicles gene expression, resulting in abnormal ovulatory follicles that could produce low quality oocytes and/or form corpora lutea with abnormal function (Britt, 1995).

In summary, increased energy intake after calving stimulated secretion of anabolic hormones during the first 3 mo of lactation. Concomitantly, primiparous cows that calved with a thin or moderate body condition increased fat tissue deposition and, probably, milk production, as reflected by increased growth rate of the calves. However, resumption of ovarian function was limited during the first 3 mo after calving. Cows that previously had a high nutrient intake postpartum not only resumed ovarian activity early, but also had a greater pregnancy rate from AI at the first estrus. Although concentrations of IGF-I and leptin in plasma were greater in H cows during consumption of a high nutrient intake, concentrations were similar for H and M cows during 7 wk before the first

estrus. This indicates that plasma concentrations of IGF-I and leptin may not limiting factors, or were not the only factors, that control the resumption of ovarian function in primiparous beef cows. Differences in endocrine function or metabolic signals during the first 3 mo after calving could have influenced ovarian activity and fertility at the first postpartum estrus.

Implications

Reproductive performance of primiparous cows that calve with thin or moderate body condition can be increased by feeding a high-energy supplement after calving. Cows that maintain or lose body condition during lactation have a prolonged interval from calving to estrus, are less fertile, and produce lighter calves at weaning. Estrus detection should be intensified in postpartum cows due to the short duration of estrus period and less mounts received. Concentrations of IGF-I, leptin, insulin, glucose, NEFA, or thyroxine in blood may not individually signal the onset of postpartum ovarian function but may act in concert with others factors to signal the adequacy of nutrients. Additional research is necessary to elucidate the mechanism(s) that control the postpartum anestrous interval to maximize reproductive efficiency in beef cattle.

Table 1. Effects of body condition score[a] (BCS) at calving and postpartum nutrition (PPN) on change in BCS and body weight (BW) of primiparous lactating beef cows

	Treatment[b]					P-value		
Item	4-M	4-H	5-M	5-H	SE	BCSC[c]	PPN	INT[d]
Cows, no.	14	15	28	26	-	-	-	-
BCS at calving	4.4	4.4	5.1	5.1	0.06	0.01	0.60	0.63
BCS at the end of feeding	4.3	5.0	4.6	5.4	0.15	0.01	0.01	0.64
Change in BCS	-0.1	0.6	-0.5	0.3	0.09	0.01	0.01	0.34
Precalving BW, kg	420	424	406	434	22	0.05	0.80	0.13
BW at the end of feeding, kg	370	440	390	456	14	0.07	0.01	0.85
ADG during feeding, kg	0.32	1.20	0.37	1.09	0.11	0.67	0.01	0.33

[a]1 = emaciated and 9 = obese (Wagner et al., 1988).
[b]4-M = BCS 4-Moderate, 4-H = BCS 4-High, 5-M = BCS 5-Moderate, 5-H = BCS 5-High.
[c]BCSC = body condition score at calving.
[d]INT = interaction between BCSC and PPN.

Table 2. Effects of body condition score[a] (BCS) at calving and postpartum nutrition (PPN) on calf performance

	Treatment[b]					P-value		
Item	4-M	4-H	5-M	5-H	SE	BCSC[c]	PPN	INT[d]
Calves, no.	14	15	28	26	-	-	-	-
Birth wt, kg	33.6	32.8	33.1	34.2	1.2	0.60	0.82	0.22
Weight at the end of feeding, kg	83.0	101.0	82.7	93.7	9	0.17	0.01	0.34
Change in wt, kg	50	68	49	60	8	0.13	0.01	0.20
Weight at 205-d, kg	174	185	174	183	17	0.83	0.05	0.77

[a]1 = emaciated and 9 = obese (Wagner et al., 1988).
[b]4-M = BCS 4-Moderate, 4-H = BCS 4-High, 5-M = BCS 5-Moderate, 5-H = BCS 5-High.
[c]BCSC = body condition score at calving.
[d]INT = interaction between BCSC and PPN.

Table 3. Partial correlation coefficients, adjusted for cow, among plasma concentrations of insulin-like growth factor-I (IGF-I), leptin (LEP), insulin (INS), glucose (GLU), nonesterified fatty acids (NEFA), and thyroxine (T4) of primiparous beef cows (n = 30) during the last 3 weeks of nutritional treatment

Variable	LEP[a]	INS[b]	GLU	NEFA	T4
IGF-I	0.29*	0.55**	0.50**	-0.17	0.53**
LEP	-	0.43**	0.27*	0.12	0.49**
INS	-	-	0.45**	0.02	0.60**
GLU	-	-	-	0.03	0.68**
NEFA	-	-	-	-	0.09

[a] Correlations involving LEP or IGF-I based on n = 68.
[b] Correlations do not involve LEP or IGF-I based on n = 90.
*P < 0.05; **P < 0.01

Table 4. Influences of postpartum nutrition on estrous behavior, ovarian function, and reproductive performance at the first estrus of primiparous beef cows

	Postpartum nutrition			
Item	Moderate	High	SE	P-value
Cows, no.	33	38	-	-
Short LA[a], %	87	88	-	0.74
Duration of estrus, h	5.5	5.6	1.2	0.88
Mounts received, no.	15.7	17.8	5.1	0.54
Diameter of DF[b], mm	13.5	14.8	0.3	0.01
Duration of PPI[c], d	120	100	7.1	0.01
Pregnancy rate, %				
Year 1	80 (15)[d]	100 (16)	-	-
Year 2	39 (18)	59 (22)	-	-
Both years	57.6	76.3	-	0.03

[a]Luteal activity prior to first estrus based on Year 1 data.
[b]Dominant follicle.
[c]Postpartum anestrous interval.
[d]Number of cows inseminated.

Figure 1. Least squares mean concentrations of IGF-I (A), leptin (B), insulin (C), glucose (D), NEFA (E), and thyroxine (F) in plasma during 3 weeks before and 3 weeks after the end of nutritional treatment (High, n = 15; Moderate, n = 15) of primiparous lactating beef cows. * Treatment effect ($P < 0.05$). ** Treatment effect ($P < 0.01$).

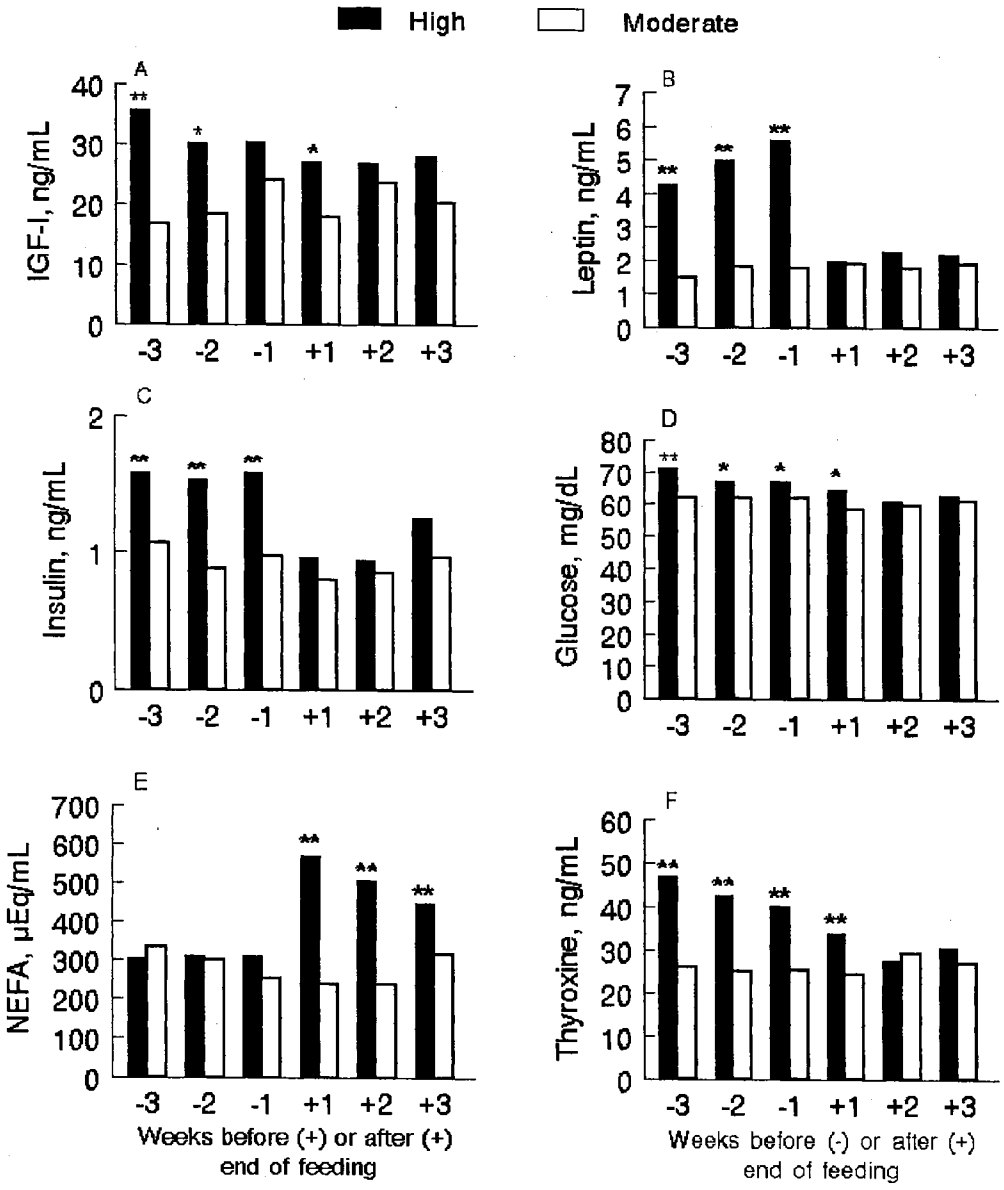
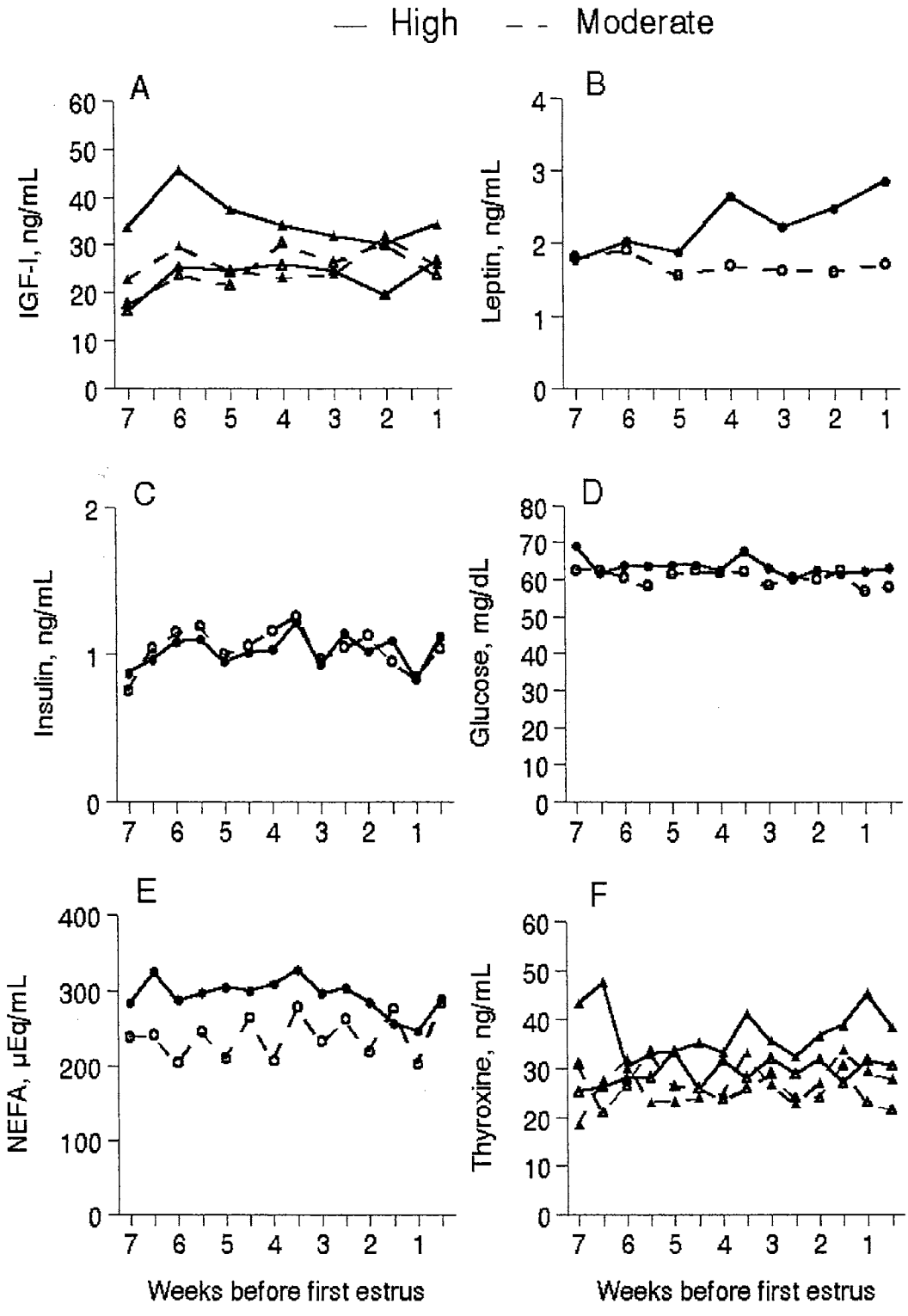


Figure 2. Least squares mean concentrations of IGF-I (A), leptin (B), insulin (C), glucose (D), NEFA (E), and thyroxine (F) in plasma during 7 weeks before the first estrus of primiparous lactating beef cows previously fed a High (n = 15) or a Moderate (n = 15) energy diet after calving. In panel A and F, lines with solid or open triangles represent cows that calved with body condition score 5 or 4, respectively.



CHAPTER IV

Effect of Nutrient Intake on Steroid Concentrations in the Dominant Follicle at First Postpartum Estrus of Beef Cows

ABSTRACT

Angus x Hereford cows maintained on pasture (n = 12; BW = 423 ± 2 kg) were used to determine if postpartum nutrient intake alters concentrations of steroids in follicular fluid (FFL) of dominant follicles (DF) at the first postpartum estrus. At calving, cows were stratified by body condition score (BCS = 4.0 ± 0.1) and calving date and randomly allotted to gain 0.45 kg/d (M; n = 6) or 0.90 kg/d (H; n = 6) for the first 65 ± 5 d postpartum. Onset of estrus was defined as the first of two mounts detected within 4 h using the Heatwatch system. At 4 to 14 h after onset of estrus, FFL from DF was aspirated using an ultrasound-guided needle. Concentrations of estradiol (E_2), progesterone (P_4), and androstenedione (A_4) in FFL were quantified by RIA. First estrus occurred at 78 ± 7 d after calving. Concentrations of E_2, P_4, and A_4 in FFL from DF

after onset of first estrus were not affected ($P > 0.18$) by nutrient intake. To determine if aspiration of the DF at estrus affects concentrations of P_4 in plasma during the subsequent estrous cycle, an additional group of Angus x Hereford nonaspirated cows (n = 28; BW = 401 ± 1 kg; BCS = 4.6 ± 0.01) was used as a control. After the first postpartum estrus, blood samples were collected daily or once a week for aspirated or control cows, respectively. Both groups of cows were fed to gain 0.45 kg/d (M diet) from the first estrus until the next estrus or 24 d later. From d 2 to d 10 of the subsequent estrous cycle, concentrations of P_4 in plasma increased ($P < 0.01$) at a greater rate per d (0.71 ng/mL/d) in control cows than in aspirated cows (0.48 ng/mL/d). In conclusion, nutrient intake during 65 d after calving did not alter concentrations of steroids in FFL after first estrus (78 ± 7 d postpartum), and aspiration of the preovulatory DF reduced secretion of progesterone during the subsequent estrous cycle.

Key Words: Nutrition, Dominant follicle, Aspiration, Estradiol, Beef cows

Introduction

Length of the postpartum anovulatory period in beef cows is affected by body condition score (BCS) at calving (Selk et al., 1988) and nutrient intake after calving (Dunn and Kaltenbach, 1980). Restricted nutrient intake reduces the diameter and growth rate of dominant follicles (DF) in cyclic (Murphy et al., 1991), nutritional anestrous (Bossis et al., 1999), and postpartum (Grimard et al., 1995; Lents et al., 2000) beef cows. However, it has not been determined if alterations in nutrient intake affect steroidogenic capacity of DF at first postpartum estrus.

Ultrasound-guided transvaginal follicular aspiration (Pieterse et al., 1988) is a technique mainly developed to harvest oocytes in vivo and can be used to sample follicular fluid from cows during follicular growth. Thus, the objectives of this study were: 1) to determine if alterations in nutrient intake after calving can influence concentrations of estradiol, progesterone, and androstenedione in follicular fluid aspirated from the DF at the first postpartum estrus, and 2) to determine if aspiration of DF at estrus influences plasma concentrations of progesterone and duration of the subsequent estrous cycle.

Materials and Methods

Animals, treatments and follicular aspiration. Angus x Hereford 3-yr old cows (n = 12, BW = 423 ± 2 kg) were maintained on pasture during gestation. At calving (March 1 ± 3 d), cows were stratified by body condition score (BCS = 4.0 ± 0.07, 1: emaciated, 9:obese; Wagner et al., 1988) and calving date and randomly allotted to moderate (M) or high (H) nutrient intake for the first 65 ± 5 d postpartum. During treatments, all cows were maintained on dormant or early spring native pasture and had prairie hay (4% CP) ad libitum. Cows on the M treatment were supplemented with 2 kg/d of 38% CP range cubes, whereas H cows had free access to a high-energy diet (1.61 Mcal NE_m/kg DM, 0.90 Mcal NE_g/kg DM, and 11.1% CP). The diet was composed (% DM) of rolled corn (39.7%), ground alfalfa pellets (35.5%), cottonseed hulls (22%), cane molasses (2.5%) and salt (0.3%). Body weight and BCS were determined monthly, from calving until the end of treatment, after cows were denied access to feed and water for 16 h. At 30 d postpartum, a radiotelimetric pressure-sensitive device (Heatwatch, DDx Inc., Denver, CO) was attached to the rump of each cow to monitor mounts received. Onset of estrus was defined as the first of two

mounts detected within 4 h. At 4 to 14 h after the onset of the first postpartum estrus, the DF was measured by ultrasonography and the FFL was aspirated using an ultrasound-guided needle via a vaginal approach (Pieterse et al., 1988). A real-time ultrasound scanner (Aloka 500-V; Corometrics Medical Systems, Wallingford, CT) equipped with a puncture line, a 5-MHz curved probe, and a needle guide were used. Briefly, the Heatwatch transmitter was removed, and epidiural anesthesia was induced with 5 mL of 2% lidocaine. The perineal region was disinfected, and an 18 G, 55 cm needle (Cook Veterinary Products, Spencer, IN) was inserted into the needle guide to puncture the vaginal wall and the DF. Follicular content was aspirated by vacuum into a 5-mL syringe attached to the aspiration needle. Immediately after collection, FFL samples were transferred to a 12-mL centrifuge tube, cooled on ice, and transported to the lab within 1 h. At the lab, samples were centrifuged at 4 °C (2000 g for 10 min), FFL was removed, and stored at -20 °C in cryogenic vials for steroid analysis. For technical reasons, FFL were not collected from 4 cows (2 cows per treatment).

Management of cows and blood sampling after follicular aspiration. After follicular aspiration, the Heatwatch transmitter was replaced on the rump of each cow. Then cows were maintained in a pen (25 x 30 m) with at least two other cows for estrous detection. Daily blood samples were collected by tail-vein puncture into vacutainers (10 mL) with EDTA (0.1 mL of a 15% solution), from the day of estrus (d 0) until the next estrus or d 24. To determine if aspiration of DF at the first postpartum estrus affects concentrations of progesterone during the subsequent estrous cycle, an additional group of Angus x Hereford 2-yr old cows (n = 28; BW = 401 ± 1 kg; BCS = 4.6 ± 0.01) was used as a control (non-follicular aspiration). Control cows were bled once a wk after the first estrus detected by Heatwatch. Aspirated and non-aspirated cows were fed the M-diet from first estrus until the end of blood sampling. Cows were AI between 14 to 20 h after onset of next estrus.

Hormone assays. Concentrations of androstenedione in FFL were determined in one assay using a solid-phase RIA (ICN Pharmaceuticals, Inc., Costa Mesa, CA) as previously described (Stewart et al., 1996). The intraassay CV was 9%. Concentrations of estradiol in FFL were quantified in one assay by RIA as previously described (Spicer and Enright,

1991). The intraassay CV was 11%. Plasma concentrations of estradiol 17-β were determined in one assay by RIA (Estradiol MAIA, Polymedco Inc., New York, NY) with modifications (Vizcarra et al., 1997). The intraassay CV was 15%. Concentrations of progesterone in FFL were quantified in one assay by RIA as previously described (Spicer and Enright, 1991). The intraassay CV was 8%. Concentrations of progesterone in plasma were quantified using a solid-phase RIA (Coat-A-Count Progesterone kit, Diagnostic Products Corp.; Vizcarra et al., 1997). Intra- and interassay CV (n=2) were 4 and 8%, respectively.

Statistical analyses. Concentrations of steroids in FFL were analyzed with the MIXED procedure of SAS (SAS Inst. Inc., Cary, NC), using a one-way ANOVA with nutrition as the main effect. When treatment means had heterogeneous variance, Satterthwaite's approximation was used for calculation of the effective degrees of freedom for the error term (Steel et al., 1997). The effect of follicular aspiration on plasma concentrations of progesterone was analyzed from d 2 to d 10 of the subsequent estrous cycle to avoid differences in luteal function between pregnant (n=15) and nonpregnant (n=13) control cows. Polynomial response curves, up to the fourth order, were fitted to

describe secretion of progesterone through day of the cycle in treated (ASP) and control (CON) cows. Linear equations best described secretion of progesterone in ASP and CON cows, so concentrations of progesterone were analyzed with a MIXED model that included treatment and day(treatment) as fixed effects, and assay (n=2) and cow(treatment) as random effects. The model was fitted without an overall intercept, thus, the solutions for the treatment and day(treatment) effects were the individual intercepts and slopes, respectively, for the linear regression of concentrations of progesterone on day of the cycle. The ESTIMATE option of SAS was used to determine if intercepts and slopes were different between treatments.

Results

Body condition score and postpartum interval. Nutritional management was effective to increase BCS of H cows at the end of feeding. Cows on the H diet gained ($P < 0.01$) 0.75 ± 0.14 unit of a BCS whereas cows on the M diet gained 0.25 ± 0.14. Postpartum interval was not different ($P = 0.35$) for H (85 ± 10 d) and M cows (71 ± 10 d). Aspiration of the DF occurred during the last two wk of nutritional

treatment in 75% (3 cows per each treatment) of cows. For the other two cows, the DF was aspirated 20 (M cow) and 48 (H cow) d after the end of nutritional treatment.

Size of dominant follicle and concentrations of steroids in follicular fluid. Maximum diameter of the DF at estrus immediately before follicular aspiration did not differ ($P = 0.52$) between treatments (16.6 ± 2.6 vs 13.5 ± 2.6 mm; H vs M cows, respectively). Concentrations of estradiol, progesterone, and androstenedione in follicular fluid aspirated from the DF after the onset of the first postpartum estrus were not affected ($P > 0.18$) by nutrient intake (Table 1). Mean concentrations of estradiol, progesterone, and androstenedione at 10.0 ± 1.7 h after estrus were 85.5 ± 48.5 ng/mL, 237 ± 54 ng/mL, and 5.7 ± 1.8 ng/mL, respectively. The ratio of estradiol:progesterone in FFL was similar ($P = 0.65$) for H and M cows. Concentrations of estradiol in plasma collected immediately before follicular aspiration were not influenced by nutritional treatment (0.86 vs 1.45 pg/mL, H vs M, respectively, $P = 0.23$).

Luteal function after follicular aspiration. Aspiration of the DF at the first postpartum estrus

affected concentrations of progesterone in plasma from d 2 to d 10 of the subsequent estrous cycle (Figure 1). Mean concentrations of progesterone in plasma increased ($P < 0.01$) at a greater rate per d (0.71 ng/mL/d) in CON-cows than in ASP-cows (0.48 ng/mL/d). Seventy-five percent (6/8) of the ASP-cows had an inter-estrus interval of 19 ± 1 d whereas two ASP-cows were not detected in estrus by d 24 after aspiration of the DF. Pregnancy rate after a single AI was 100% for ASP-cows detected in estrus (n = 6) within 24 d of aspiration of the DF.

Discussion

Concentrations of estradiol, progesterone, and androstenedione in follicular fluid of the DF at the first estrus were similar for suckled cows fed H or M nutrient intake postpartum. Growth of bovine ovulatory follicles is influenced by nutrient intake (Murphy et al., 1991; Grimard et al., 1995; Bossis et al., 1999; Ciccioli and Wettemann, 2000), but dietary effects on steroidogenic capacity of DF have not yet been established. Concentrations of estradiol in FFL of large follicles decrease with short-term fasting (Spicer et al., 1992), but they were similar for heifers fed 1.8% or 0.7% of BW in dry matter per day during 10 wk

(Spicer et al., 1991). Estradiol release into culture medium was similar for individual large follicles collected from nonlactating beef cows fed 130 or 65% of NRC requirements for 80 d (Staigmiller et al., 1982). Estradiol production of bovine granulosa cells from small follicles, but not from medium size follicles, was increased by a greater dietary intake (Armstrong et al., 2002). Rate of gain during realimentation of nutritional-induced anestrous cows had no effect on peripheral concentrations of estradiol during the follicular wave that preceded the first ovulation (Bossis et al., 2000). These results indicate that postpartum nutrition may not be associated with a greater production of estradiol by the DF at the first estrus. However, the prolonged postpartum interval in cows with low body condition score at calving may be due to reduced capacity to convert androgens to estradiol in estrogen-active follicles (Prado et al., 1990). Elucidation of this hypothesis requires investigating the effect of nutrient intake on aromatase activity in follicles collected at different times during the postpartum period.

The time of FFL aspiration relative to follicular growth and the ovulatory surge may have influenced concentrations of steroids in FFL. Onset of estrus occurs

concurrently with the preovulatory surge of LH in the cow (Swanson and Hafs, 1971; Chenault et al., 1975). Secretion of estradiol from the largest follicle in vitro decreases rapidly within 2 h after the LH surge (Staigmiller et al., 1982). Amounts of P450 17α-hydroxylase and P450 aromatase mRNA in follicular cells, and concentrations of estradiol in FFL decrease in preovulatory bovine follicles within 6 h after the gonadotropin surge (Voss and Fortune, 1993; Komar et al., 2001). In the present study, follicular fluid was aspirated from the DF an average 10 h after onset of estrus. Concentrations of progesterone were greater and concentrations of estradiol and androstenedione were less than those previously observed in our lab for FFL collected from DF not exposed to the LH surge (Stewart et al., 1996). For instance, concentrations of progesterone, estradiol, and androstenedione in FFL in our study were 423%, 55%, and 4%, respectively, of those determined from DF of the first follicular wave in lactating dairy cows (Stewart et al., 1996). These results indicate that our samples were collected when granulosa cells of DF had been luteinized, so the steroidogenic pathways had changed from estradiol to progesterone biosynthesis. In the present study, ratios of estradiol to progesterone in FFL of all follicles were less

than one, which also indicates that DF had become estrogen-inactive (Ireland and Roche, 1982). Therefore, we can not evaluate the effect of nutrient intake on estradiol synthesis in the DF at the first postpartum estrus because our samples were collected after DF became estrogen-inactive.

Aspiration of the DF after onset of estrus caused plasma concentrations of progesterone to increase at a lower daily rate from d 2 to d 10 of the subsequent estrous cycle. Previously, concentrations of progesterone in plasma were not affected by aspiration of the DF during early, mid, and late luteal phases in nonpregnant dairy cows (Amiridis et al., 1999), but in that experiment the corpus luteum had been formed before DF aspiration, which differs with the present study. Concentrations of progesterone in plasma depend on the balance between progesterone secretion by luteal tissue and metabolism of progesterone by the liver. Follicular aspiration probably decreased concentrations of progesterone in plasma by altering luteal secretion. Corpora lutea of domestic ruminants include two types of steroidogenic cells; small luteal cells and large luteal cells (Farin et al., 1986; Hansel et al., 1991). Large luteal cells are derived from granulosa cells whereas

small luteal cells are derived from the theca interna (Alila and Hansel, 1984). In vitro, both cell types produce progesterone, and production of progesterone is regulated differently by each cell type (Wiltbank, 1994). Large luteal cells produce about 80% of the progesterone secreted by the CL in vivo (Niswender et al., 1985). Apart from collection of follicular fluid, aspiration also removes the oocyte-cumulus complex and some mural granulosa cells. Probably, the number of granulosa cells available to differentiate into large luteal cells is less in the follicular wall after aspiration. Thus, the corpus luteum originating from aspirated follicles may secrete less progesterone.

The effect of aspiration of DF on duration of estrous cycles in cows is not established. Most studies have maximized the rate of oocyte recovery with long-term follicular aspiration, which alters ovarian and endocrine function and interferes with normal estrous cycles (Carlin et al., 1999). Ablation of the preovulatory DF at the first postpartum estrus did not alter length of the subsequent cycle. This is an expected result since the biochemical events associated with follicular luteinization and ovulation had already occurred and thus were not altered by

follicular aspiration. Ablation of the DF occurred about 10 h after the onset of estrus and the LH surge. The process of luteinization is triggered by the preovulatory LH surge (Murphy, 2000), indicating that development of corpora lutea had started when DF were aspirated. Ablation of the preovulatory DF 10 h after estrus could be similar to a premature ovulation which would have been 21 h earlier than normal ovulation in beef cows (White et al., 2002). Although progesterone concentrations were less after follicular aspiration, they were sufficient to allow normal estrous cycles. The 19 d estrous cycles observed in this study are considered within the normal range in cows (Cupps, 1991; Senger, 1997). Aspiration of DF during early, mid, or late luteal phase did not affect length of estrous cycles (Amiridis et al., 1999). Fertility after a single AI was 100% for the limited number of cows that were detected in estrus after aspiration, even though plasma concentrations of progesterone secretion from d 2 to d 10 of the previous cycle was less than for control cows.

Implications

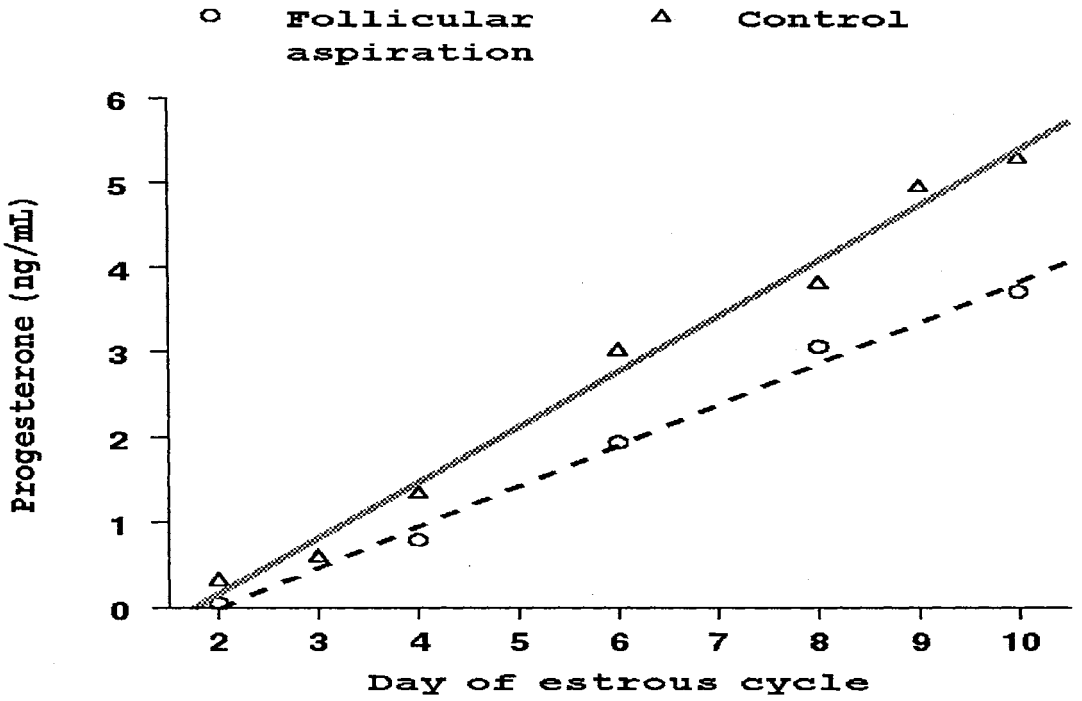
If the dominant follicle of postpartum cows produces sufficient estradiol to cause estrus, previous nutrient

intake does not influence concentrations of estradiol, progesterone, and androstenedione in follicular fluid. Aspiration of the preovulatory DF reduced rate of progesterone increase in blood during the early to mid luteal phase of the subsequent estrous cycle. However, length of the cycle and fertility at the next estrus were not compromised.

Table 1. Effect of nutrient intake during the first 65 d postpartum on concentrations of estradiol (E_2), progesterone (P_4), and androstenedione (A_4) in follicular fluid (FFL) of dominant follicles after onset of the first postpartum estrus of beef cows

	Nutrient intake			
Variable	Moderate	High	Pooled SE	P-value
FFL samples, no.	4	4	-	-
E_2, ng/mL	76.9	94.0	48.5	0.83
P_4, ng/mL	206.4	268.0	54.0	0.51
A_4, ng/mL	3.2	8.1	1.8	0.18
E_2:P_4 ratio	0.38	0.67	0.37	0.65

Figure 1. Linear regressions (lines) and means (symbols) for concentrations of progesterone from d 2 to d 10 of the estrous cycle after aspiration or nonaspiration of dominant follicle at the first estrus in postpartum beef cows. Treatment effect ($P < 0.01$).



CHAPTER V

SUMMARY AND CONCLUSIONS

Reproductive potential of beef cows is strongly dependent on nutrient availability. An understanding of the physiological mechanisms that mediate nutritional effects on resumption of ovarian function after calving will provide a basis to design feeding strategies that can enhance reproductive performance. Two experiments were conducted to study the influence of nutrient intake on endocrine and reproductive function at the first postpartum estrus in beef cows. The specific objectives were: 1) to determine the effects of pre- and postpartum nutrient intake on estrous behavior, ovarian function, pregnancy rate at first estrus, and concentrations of IGF-I, leptin, NEFA, glucose, insulin, and thyroxine in primiparous postpartum beef cows; 2) to explore potential relationships between postpartum endocrine and reproductive functions; and 3) to determine the effect of nutrition on concentrations of steroids in follicular fluid aspirated at

the first estrus and on luteal function during the subsequent estrous cycle.

In the first experiment, the influence of body condition score (BCS) at calving and postpartum nutrition on endocrine and ovarian functions, and reproductive performance, was determined by randomly allocating thin (BCS 4.4) or moderate (BCS 5.5) Hereford x Angus primiparous cows at calving to receive either one of two nutritional treatments after calving. Cows were group-fed and targeted to gain 0.45 kg/d (M; n = 17) or 0.90 kg/d (H; n = 17) for the first 71 ± 17 d postpartum. Then, all cows were fed the same (M) diet until the first estrus. A replication (M, n = 25; H, n = 23) was added to assess pregnancy rate. Concentrations of IGF-I, leptin, insulin, glucose, NEFA, and thyroxine (T_4) were quantified in plasma samples collected weekly during treatment and during 7 wk before the first estrus. Estrous behavior was detected by radiotelemetry and ovulation was determined using plasma progesterone concentrations. All cows were AI between 14 and 20 h after onset of estrus, and pregnancy status was determined at 35 to 55 d post-AI by ultrasonography.

Heifers that calved with a BCS of 4 or 5 had similar endocrine function and reproductive performance at the

first estrus. During treatment, H cows gained BW and BCS, and had greater concentrations of IGF-I, leptin, insulin, glucose, and T_4 compared with M cows. However, during the 7 wk before the first estrus, plasma concentrations of metabolites and metabolic hormones were similar for H and M cows, and they were not affected by time. Cows fed the H diet had a shorter interval to first estrus and a larger dominant follicle at first estrus, but the number of mounts received and duration of estrus was similar to M cows. Pregnancy rate at the first postpartum estrus was greater for H (76%, n = 38) than for M (57%, n = 33) cows.

In the second experiment, Angus x Hereford cows were used to determine if postpartum nutrient intake alters concentrations of steroids in follicular fluid collected from the dominant preovulatory follicle at the first estrus. At calving, thin cows (BCS = 4.0 ± 0.1) were stratified by BCS and calving date, maintained on pasture, and fed to gain 0.45 kg/d (M) or 0.90 kg/d (H) for the first 65 d postpartum. At 4 to 16 h after onset of the first estrus, follicular fluid from the dominant follicle (DF) was aspirated using an ultrasound-guided needle via a vaginal approach. Concentrations of estradiol, progesterone, and androstenedione in follicular fluid from

the DF at the first estrus were similar for cows with M or H nutrient intake postpartum.

Increased energy intake after parturition stimulated secretion of anabolic hormones during the first 3 mo of lactation. Secretion of anabolic hormones promoted fat and tissue deposition in primiparous cows that calved with thin or moderate BCS. However, only 24 % of H cows resumed ovarian function during the first 90 d postpartum, indicating that changes in metabolic status were not sufficient to override the negative influence of other factors that delay the first estrus. Failure of ovulation during the postpartum period results from inadequate pulsatile secretion of GnRH and LH during the late growing phase of dominant follicles. Inadequate BCS at calving and suckling delay the return to normal pulsatile LH secretion.

Greater nutrient intake after calving stimulated secretion of leptin in plasma of lactating beef cows. Plasma concentrations of leptin decreased acutely within 4 d after cows were switched from high to moderate nutrient intake. This decrease was probably not linked to changes in BCS or BW since it occurred in a short time period, which indicates that nutrient intake affects secretion of leptin in plasma, independently of changes in BCS or BW.

Concentrations of leptin, insulin, and glucose were positively associated during nutritional treatment. Conversely, decreased nutrient intake resulted in reduced plasma concentrations of leptin and increased NEFA release from adipocytes. Secretion of leptin in plasma in response to nutrient intake was probably coupled to fat synthesis and degradation.

Concentrations of IGF-I, leptin, insulin, glucose, NEFA, and T_4 did not change during the 7 wk preceding resumption of ovulation. Leptin and IGF-I could mediate some of the effects of nutrition on reproductive function since they can act at the brain-pituitary-ovary level and are positively associated with nutrient intake. However, our results indicate that IGF-I and leptin may not be major regulators of pulsatile LH secretion in postpartum beef cows. Alternatively, it is possible that concentrations of IGF-I and leptin must be maintained over a threshold concentration in plasma for several weeks to restore normal activity of the hypothalamo-pituitary-ovarian axis. If this situation exists, it could take 30 d or longer to restore the frequency LH pulses to normal because concentrations of IGF-I and leptin remained constant during 7 wk before the first estrus.

Increased nutrient intake after calving enhanced pregnancy rate at the first postpartum estrus. There is not a clear explanation for this novel effect of nutrition on fertility. We speculate that the endocrine and metabolic changes induced by nutrient intake during 50 to 70 d postpartum, may influence follicular growth and oocyte maturation, and subsequent embryo development. This hypothesis could be tested using an embryo production system in vitro. Oocytes, collected from cows fed H or M nutrient intake postpartum, could be matured and fertilized in vitro to determine the subsequent rate of blastocyst formation.

The second experiment demonstrated postpartum nutrient intake did not influence concentrations of estradiol, progesterone, and androstenedione in follicular fluid of the dominant follicle at the first estrus. Effects of nutrient intake on follicular growth are well documented, but it may not be associated with a greater steroidogenic potential at first estrus. However, nutrient intake may influence the timing of events within the follicle, such as expression of the aromatase system.

Reproductive performance of primiparous cows that calve with thin or moderate body condition can be increased

by feeding a high-energy supplement after calving. Cows that maintain or lose body condition during lactation have a prolonged interval from calving to estrus, are less fertile, and produce lighter calves at weaning. Concentrations of IGF-I, leptin, insulin, glucose, NEFA, or thyroxine in blood may not individually signal the onset of postpartum ovarian function but may act in concert with others factors to signal the adequacy of nutrients. Differences in endocrine function or metabolic signals during the first three months after calving could influence ovarian activity and fertility at the first postpartum estrus. If dominant follicles of postpartum cows produce sufficient estradiol to cause estrus, previous nutrient intake does not influence concentrations of estradiol, progesterone, and androstenedione in follicular fluid. Additional research is necessary to elucidate the mechanism(s) that control the postpartum anestrous interval to maximize reproductive efficiency in beef cows.

LITERATURE CITED

Abilay, T. A., H. D. Johnson, and M. Madan. 1975. Influence of environmental heat on peripheral plasma progesterone and cortisol during the bovine estrous cycle. J. Dairy Sci. 58:1836-1840.

Acosta, B., G. K. Tarnavsky, T. E. Platt, D. L. Hamernik, J. L. Brown, H. M. Schoenemann, and J. J. Reeves. 1983. Nursing enhances the negative effect of estrogen on LH release in the cow. J. Anim. Sci. 57:1530-1536.

Adam, C. L., T. S. Gadd, P. A. Findlay, and D. C. Wathes. 2000. IGF-I stimulation of luteinizing hormone secretion, IGF-bindings proteins (IGFBPs) and expression of mRNAs for IGFs, IGF receptors and IGFBPs in the ovine pituitary gland. J. Endocrinol. 166:247-254.

Adams, G. P., R. L. Matteri, J. P. Kastelic, J. C. H. Ko, and O. J. Ginther. 1992. Association between surges of follicle-stimulating hormone and the emergence of follicular waves in heifers. J. Reprod. Fertil. 94:177-188.

Adashi, E. Y. 1998. The IGF family and folliculogenesis. J. Reprod. Immunology 39:13-19.

Adashi, E. Y., C. E. Resnik, and R. G. Rosenfeld. 1989. Insulin-like growth factor-I (IGF-I) and IGF-II hormonal action in cultured rat granulosa cells: mediation via type I but not type II IGF receptors. Endocrinology 126:216-222.

Ahima, R. S. and J. S. Flier. 2000. Leptin. Annu. Rev. Physiol. 62:413-437.

Ahima, R. S., D. Prabakaran, C. Mantzoros, D. Qu, B. Lowell, E. Maratos-Flier, and J. S. Flier. 1996. Role of leptin in the neuroendocrine response to fasting. Nature 382:250-252.

Ahmadzadeh, A., M. A. Barnes, and R. E. Pearson. 1998. Effect of naloxone on serum luteinizing hormone concentration in anovulatory Holstein cows during the early postpartum period. Domest. Anim. Endocrinol. 15:177-181.

Alila, H. W., and W. Hansel. 1984. Origin of different cell types in the bovine corpus luteum as characterized by specific monoclonal antibodies. Biol. Reprod. 31:1015-1025.

Allrich, R. D. 1994. Endocrine and neural control of estrus in dairy cows. J. Dairy Sci. 77:2738-2744.

Allrich, R. D., D. L. Cook, L. A. Horstman, R. J. Knutson, and T. A. Winters. 1989. Influence of dexamethasone, progesterone, gonadotropin-releasing hormone, and testosterone on estrous behavior of estradiol-treated ovariectomized heifers. J. Dairy Sci. 72:2707-2711.

Amiridis, G. S., L. Robertson, S. Reid, J. S. Boyd, P. J. O'Shaughnessy, and I. A. Jeffcoate. 1999. Plasma estradiol, FSH and LH concentration after dominant follicle aspiration in the cow. Theriogenology 52:995-1003.

Amstalden, M., M. R. Garcia, R. L. Stanko, S. E. Nizielski, C. D. Morrison, D. H. Keisler, and G. L. Williams. 2002. Central infusion of recombinant ovine leptin normalizes plasma insulin and stimulates a novel hypersecretion of luteinizing hormone after short-term fasting in mature beef cows. Biol. Reprod. 66:1555-1561.

Amstalden, M., M. R. Garcia, S. W. Williams, R. L. Stanko, S. E. Nizielski, C. D. Morrison, D. H. Keisler, and G. L. Williams. 2000. Leptin gene expression, circulating leptin, and luteinizing hormone pulsatility are acutely responsive to short-term fasting in prepubertal heifers: relationships to circulating insulin and insulin-like growth factor I. Biol. Reprod. 63:127-133.

Anderson, L. H., C. M. McDowell, and M. L. Day. 1996. Progestin-induced puberty and secretion of luteinizing hormone in heifers. Biol. Reprod. 54:1025-1031.

Anderson, R. A., I. H. Zwain, A. Arroyo, P. L. Mellon, and

S. C. S. Yen. 1999. The insulin-like growth factor system in the GT1-7 GnRH neuronal cell line. Neuroendocrinology 70:353-359.

Arias, P., M. Rodriguez, B. Szwarcfarb, I. R. Sinay, and J. A. Moguilevsky. 1992. Effect of insulin on LHRH release by perifused hypothalamic fragments. Neuroendocrinology 56:415-418.

Arije, G. R., J. N. Wiltbank, and M. L. Hopwood. 1974. Hormone levels in pre- and post-parturient beef cows. J. Anim. Sci. 39 :338-347.

Armstrong, D. G., J. G. Gong, J. O. Gardner, G. Baxter, C. O. Hogg, and R. Webb. 2002. Steroidogenesis in bovine granulosa cells: the effect of short-term changes in dietary intake. Reproduction 123:371-378.

Armstrong, D. G., C. G. Gutierrez, G. Baxter, A. L. M. G. E. Glazirin, K. J. Woad, C. O. Hogg, and R. Webb. 2000. Expression of mRNA encoding IGF-I, IGF-II and type 1 IGF receptor in bovine ovarian follicles. J. Endocrinol. 165:101-113.

Armstrong, D. G., T. G. McEvoy, G. Baxter, J. J. Robinson, C. O. Hogg, K. J. Woad, R. Webb, and K. D. Sinclair. 2001. Effect of dietary energy and protein on bovine follicular dynamics and embryo production in vitro: associations with the ovarian insulin-like growth factor system. Biol. Reprod. 64:1624-1632.

Armstrong, J. D., W. S. Cohick, R. W. Harvey, E. P. Heimer, and R. M. Campbell. 1993. Effect of feed restriction on serum somatotropin, insulin-like growth factor-I-(IGF-I) and IGF binding proteins in cyclic heifers actively immunized against growth hormone releasing factor. Domest. Anim. Endocrinol. 10:315-324.

Barash, I. A., C. C. Cheung, D. S. Weigle, H. Ren, E. B. Kabigting, J. L. Kuijper, D. K. Clifton, and R. A. Steiner. 1996. Leptin is a metabolic signal to the reproductive system. Endocrinology 137:3144-3147.

Barb, C. R. 1999. The brain-pituitary-adipose axis: role of leptin in modulating neuroendocrine function. J. Anim. Sci. 77:1249-1257.

Barb, C. R., X. Yan, M. J. Azain, R. R. Kraeling, G. B.

Rampacek, and T. G. Ramsay. 1998. Recombinant porcine leptin reduces feed intake and stimulates growth hormone secretion in swine. Domest. Anim. Endocrinol. 15:77-86.

Barr, V. A., D. Malide, M. J. Zarnowski, S. I. Taylor, and S. W. Cushman. 1997. Insulin stimulates both leptin secretion and production by rat white adipose tissue. Endocrinology 138:4463-4472.

Beal, W. E., R. E. Short, R. B. Staigmiller, R. A. Bellows, C. C. Kaltenbach, and T. G. Dunn. 1978. Influence of dietary energy intake on bovine pituitary and luteal function. J. Anim. Sci. 46:181-188.

Beam, S. W., and W. R. Butler. 1997. Energy balance and ovarian follicle development prior to the first ovulation postpartum in dairy cows receiving three levels of dietary fat. Biol. Reprod. 56:133-142.

Beam, S.W., and W. R. Butler. 1998. Energy balance, metabolic hormones, and early postpartum follicular development in dairy cows fed prilled lipid. J. Dairy Sci. 81:121-131.

Beam, S.W., and W. R. Butler. 1999. Effects of energy balance on follicular development and first ovulation in postpartum dairy cows. J. Reprod. Fert.(Suppl. 54):411-424.

Beaver, E. E., J. E. Williams, S. J. Miller, D. L. Hancock, S. M. Hannah, and D. L. O'Connor. 1989. Influence of breed and diet on growth, nutrient digestibility, body composition and plasma hormones of Brangus and Angus steers. J. Anim. Sci. 67:2415-2425.

Bell, D. J., J. C. Spitzer, and G. L. Burns. 1998. Comparative effects of early weaning or once-daily suckling on ocurrence of postpartum estrus in primiparous beef cows. Theriogenology 50:707-715.

Bellin, M. E., M. M. Hinshelwood, E. R. Hauser, and R. L. Ax. 1984. Influence of suckling and side of corpus luteum or pregnancy on folliculogenesis in postpartum cows. Biol. Reprod. 31:849-855.

Bellows, R. A. and R. E. Short. 1978. Effects of precalving feed level on birth weight, calving difficulty and

subsequent fertility. J. Anim. Sci. 46:1522-1528.

Bellows, R. A., R. E. Short, and G. V. Richardson. 1982. Effects of sire, age of dam and gestation feed level on dystocia and postpartum reproduction. J. Anim. Sci. 55:18-27.

Bergfeld, E. G. M., F. N. Kojima, A. S. Cupps, M. E. Wehrman, K. E. Peters, M. Garcia-Winder, and J. E. Kinder. 1994. Ovarian follicular development in prepuberal heifers is influenced by the dietary energy intake. Biol. Reprod. 51:1051-1057.

Bernal, A., G. V. DeMoraes, T. A. Thrift, C. C. Willard, and R. D. Randel. 1999. Effects of induced hypothyroidism on ovarian response to superovulation in Brahman (Bos indicus) cows. J. Anim. Sci. 77:2749-2756.

Bishop, D. K., and R. P. Wettemann. 1993. Pulsatile infusion of gonadotropin-releasing hormone initiates luteal activity in nutritionally anestrous beef cows. J. Anim. Sci. 71:2714-2720.

Bishop, D. K., R. P. Wettemann, and L. J. Spicer. 1994. Body energy reserves influence the onset of luteal activity after early weaning of beef cows. J. Anim. Sci. 72:2703-2708.

Blache, D., P. Celi, M. A. Blackberry, R. A. Dynes, and G. B. Martin. 2000a. Decrease in voluntary feed intake and pulsatile luteinizing hormone secretion after intracerebroventricular infusion of recombinant bovine leptin in mature male sheep. Reprod. Fertil. Dev. 12:373-381.

Blache, D., R. L. Tellam, L. M. Chagas, M. A. Blackberry, P. E. Vercoe, and G. B. Martin. 2000b. Level of nutrition affects leptin concentrations in plasma and cerepbrospinal fluid in sheep. J. Endocrinol. 165:625-637.

Block, S. S., W. R. Butler, R. A. Ehrhardt, A. W. Bell, M. E. Van Amburgh, and Y. R. Boisclair. 2001. Decreased concentration of plasma leptin in periparturient dairy cows is caused by negative energy balance. J. Endocrinol. 171:339-348.

Bossis, I., R. P. Wettemann, S. D. Welty, J. A. Vizcarra, and L. J. Spicer. 2000. Nutritionally induced anovulation in beef heifers: ovarian and endocrine function during realimentation and resumption of ovulation. Biol. Reprod. 62:1436-1444.

Bossis, I., R. P. Wettemann, S. D. Welty, J. A. Vizcarra, L. J. Spicer, and M. G. Diskin. 1999. Nutritionally induced anovulation in beef heifers: ovarian and endocrine function preceding cessation of ovulation. J. Anim. Sci. 77:1536-1546.

Braden, T. D., M. E. King, K. G. Odde, and G. D. Niswender. 1989. Development of preovulatory follicles expected to form short-lived corpora lutea in beef cows. J. Reprod. Fertil. 85:97-104.

Braden, T. D., J. G. Manns, D. L. Cermak, T. M. Nett, and G. D. Niswender. 1986. Follicular development following parturition and during the estrous cycle in beef cows. Theriogenology 25:833-843.

Breier, B. H., and P. D. Gluckman. 1991. The regulation of postnatal growth: nutritional influences on endocrine pathways and function of the somatotrophic axis. Liv. Prod. Sci. 27:77-94.

Breuel, K. F., P. E. Lewis, E. K. Inskeep, and R. L. Butcher. 1993. Endocrine profiles and follicular development in early-weaned postpartum beef cows. J. Reprod. Fertil. 97:205-212.

Britt, J. H. 1995. Effect of short- and long-term changes in energy balance on reproduction. Paper presented at Mid-South Rum. Nut. Conf., May 4-5, Dallas, TX.

Britt, J. H., R. G. Scott, J. D. Armstrong, and M. D. Whitacre. 1986. Determinants of estrous behavior in lactating Holstein cows. J. Dairy Sci. 69:2195-2202.

Bucholtz, D. C., N. M. Vidwans, C. G. Herbosa, K. K. Schillo, and D. L. Foster. 1996. Metabolic interfaces between growth and reproduction. V. Pulsatile luteinizing hormone secretion is dependent on glucose availability. Endocrinology 137:601-607.

Butler, S. T., and W. R. Butler. 2001. Insulin plays a key role in re-coupling the IGF-somatotropin axis in the

early postpartum dairy cow. J. Anim. Sci.(Suppl. 1) 79:34 (Abstr.).

Butler, W. R., and R. D. Smith. 1989. Interrelationships between energy balance and postpartum reproductive function in dairy cattle. J. Dairy Sci. 72:767-783.

Carlin, S. K., A. S. Garst, C. G. Tarraf, T. L. Bailey, M. L. McGillard, J. R. Gibbons, A. Ahmadzadeh, and F. C. Gwazdauskas. 1999. Effects of ultrasound-guided transvaginal follicular aspiration on oocyte recovery and hormonal profiles before and after GnRH treatment. Theriogenology 51:1489-1503.

Carrick, M. J. and J. N. Shelton. 1969. Oestrogen-progesterone relationships in the induction of oestrus in spayed heifers. J. Endocrinol. 45:99-109.

Chehab, F. F., K. Mounzih, R. Lu, and M. E. Lim. 1997. Early onset of reproductive function in normal female mice treated with leptin. Science 275:88-90.

Chenault, J. R., W. W. Thatcher, P. S. Kalra, R. M. Abrams, and C. J. Wilcox. 1975. Transitory changes in plasma progestins, estradiol, and luteinizing hormone approaching ovulation in the bovine. J. Dairy Sci. 58:709-717.

Cheung, C. C., J. E. Thornton, J. L. Kuijper, D. S. Weigle, D. K. Clifton, and R. A. Steiner. 1997. Leptin is a metabolic gate for the onset of puberty in the female rat. Endocrinology 138:855-858.

Chilliard, Y., F. Bocquier, C. Delavaud, M. Guerre-Millo, M. Bonnet, P. Martin, Y. Faulconnier, and A. Ferlay. 1998. Leptin in ruminants: effects of species, breed, adiposity, photoperiod, beta-agonists and nutritional status. In: Proc. Cornell Nutr. Conf. for Feed Manufacturers, Cornell Univesity, Ithaca, NY. pp. 65-74.

Clarke, I. J. 1989. The GNRH/gonadotropin axis in the ewe, cow and sow. Domest. Anim. Endocrinol. 6:1-14.

Clarke, I. J., and J. T. Cummins. 1982. The temporal relationship between gonadotropin releasing hormone (GnRH) and luteinizing hormone (LH) secretion in ovariectomized ewes. Endocrinology 111:1737-1739.

Coe, B. L. and R. D. Allrich. 1989. Relationship between endogenous estradiol-17B and estrous behavior in heifers. J. Anim. Sci. 67:1546-1551.

Collett-Solberg, P. F. and P. Cohen. 2000. Genetics, chemistry, and function of the IGF/IGFBP system. Endocrine 12:121-136.

Connor, H. C., P. L. Houghton, R. P. Lemenager, P. V. Malven, J. R. Parfet, and G. E. Moss. 1990. Effect of dietary energy, body condition and calf removal on pituitary gonadotropins, gonadotropin-releasing hormone (GnRH) and hypothalamic opioids in beef cows. Domest. Anim. Endocrinol. 7:403-411.

Considine, R. V., M. K. Sinha, M. L. Heiman, A. Kriauciunas, T. W. Stephens, M. R. Nyce, J. P. Ohannesian, C. C. Marco, L. J. McKee, T. L. Bauer, and J. F. Caro. 1996. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. N. Engl. J. Med. 334:292-295.

Convey, E. M., H. A. Tucker, and R. E. Short. 1983. Acute effect of suckling on gonadotropin, prolactin and glucocorticoid concentrations in serum of intact and ovariectomized beef cows. Theriogenology 20:661-674.

Cook, D. L., T. A. Winters, L. A. Horstman, and R. D. Allrich. 1986. Induction of estrus in ovariectomized cows and heifers: effects of estradiol benzoate and gonadotropin releasing hormone. J. Anim. Sci. 63:546-550.

Cooper, D. A., D. A. Carver, P. Villeneuve, W. J. Silvia, and E. K. Inskeep. 1991. Effects of progestagen treatment on concentrations of prostaglandins and oxytocin in plasma from the posterior vena cava of post-partum beef cows. J. Reprod. Fertil. 91:411-421.

Copelin, J. P., M. F. Smith, H. A. Garverick, and R. S. Youngquist. 1987. Effect of the uterus on subnormal luteal function in anestrous beef cows. J. Anim. Sci. 64:1506-1511.

Copelin, J. P., M. F. Smith, H. A. Garverick, and R. S. Youngquist. 1989a. Effect of termination of pregnancy or long-term progestogen exposure on subsequent estrous cycle length and concentration of progesterone

in plasma of heifers. J. Anim. Sci. 67:1552-1558.

Copelin, J. P., M. F. Smith, D. H. Keisler, and H. A. Garverick. 1989b. Effect of active immunization of pre-partum and post-partum cows against prostaglandin F-2 alpha on lifespan and progesterone secretion of short-lived corpora lutea. J. Reprod. Fertil. 87:199-207.

Corah, L. R., T. G. Dunn, and C. C. Kaltenbach. 1975. Influence of prepartum nutrition on the reproductive performance of beef females and the performance of their progeny. J. Anim. Sci. 41:819-824.

Corah, L. R., A. P. Quealy, T. G. Dunn, and C. C. Kaltenbach. 1974. Prepartum and postpartum levels of progesterone and estradiol in beef heifers fed two levels of energy. J. Anim. Sci. 39:380-385.

Crowe, M. A., P. Kelly, M. A. Driancourt, M. P. Boland, and J. F. Roche. 2001. Effects of follicle-stimulating hormone with and without luteinizing hormone on serum hormone concentrations, follicle growth, and intrafollicular estradiol and aromatase activity in gonadotropin-releasing hormone-immunized heifers. Biol. Reprod. 64:368-374.

Crowe, M. A., V. Padmanabhan, M. Mihm, I. Z. Beitins, and J. F. Roche. 1998. Resumption of follicular waves in beef cows is not associated with periparturient changes in follicle-stimulating hormone heterogeneity despite major changes in steroid and luteinizing hormone concentrations. Biol. Reprod. 58:1445-1450.

Cunningham, M. J., D. K. Clifton, and R. A. Steiner. 1999. Leptin's actions on the reproductive axis: Perspectives and mechanisms. Biol. Reprod. 60:216-222.

Cupps, P. T. 1991. Reproduction in Domestic Animals. 4th ed. Academic Press, Inc., San Diego, CA.

Daniel, J. A., M. G. Thomas, C. S. Hale, J. M. Simmons, and D. H. Keisler. 2000. Effect of cerebroventricular infusion of insulin and (or) glucose on hypothalamic expression of leptin receptor and pituitary secretion of LH in diet-restricted ewes. Domest. Anim. Endocrinol. 18:177-185.

Darwash, A. O., G. E. Lamming, and L. M. Hicking. 2001. Oestrus in relation to peak oestradiol levels in ovariectomized Galloway cows. Animal Science 72:401-405.

Davidge, S. T., J. L. Wiebold, P. L. Senger, and J. K. Hillers. 1987. Influence of varying levels of blood progesterone upon estrous behavior in cattle. J. Anim. Sci. 64:126-132.

Day, M. L., K. Imakawa, A. C. Clutter, P. L. Wolfe, D. D. Zalesky, M. K. Nielsen, and J. E. Kinder. 1987. Suckling behavior of calves with dams varying in milk production. J. Anim. Sci. 65:1207-1212.

Day, M. L., K. Imakawa, D. D. Zalesky, R. J. Kittok, and J. E. Kinder. 1986. Effects of restriction of dietary energy intake during the prepubertal period on secretion of luteinizing hormone and responsiveness of the pituitary to luteinizing hormone-releasing hormone in heifers. J. Anim. Sci. 62:1641-1648.

De Moraes, G. V., H. R. Vera-Avila, A. W. Lewis, J. W. Koch, D. A. Neuendorff, D. M. Hallford, J. J. Reeves, and R. D. Randel. 1998. Influence of hypo- or hyperthyroidism on ovarian function in Brahman cows. J. Anim. Sci. 76:871-879.

Delavaud, C., F. Bocquier, Y. Chilliard, D. H. Keisler, A. Gertler, and G. Kann. 2000. Plasma leptin determination in ruminants: effect of nutritional status and body fatness on plasma leptin concentration assessed by a specific RIA in sheep. J. Endocrinol. 165:519-526.

Delavaud, C., A. Ferlay, Y. Faulconnier, F. Bocquier, G. Kann, and Y. Chilliard. 2002. Plasma leptin concentration in adult cattle: Effects of breed, adiposity, feeding level, and meal intake. J. Anim. Sci. 80:1317-1328.

DeRouen, S. M., D. E. Franke, D. G. Morrison, W. E. Wyatt, D. F. Coombs, T. W. White, P. E. Humes, and B. B. Greene. 1994. Prepartum body condition and weight influences on reproductive performance of first-calf beef cows. J. Anim. Sci. 72:1119-1125.

Dimmick, M. A., T. Gimenez, and J. C. Spitzer. 1991.

Ovarian endocrine activity and development of ovarian follicles during the postpartum interval in beef cows. Anim. Reprod. Sci. 24:173-183.

Donaldson, L. E., J. M. Bassett, and G. D. Thorburn. 1970. Pheripheral plasma progesterone concentration of cows during puberty, oestrous cycles, pregnancy and lactation, and the effects of undernutrition or exogenous oxytocin on progesterone concentration. J. Endocrinol. 48:599-614.

Duffy, P., M. A. Crowe, M. P. Boland, and J. F. Roche. 2000. Effect of exogenous LH pulses on the fate of the first dominant follicle in postpartum beef cows nursing calves. J. Reprod. Fertil. 118:9-17.

Dunn, T. G., J. E. Ingalls, D. R. Zimmerman, and J. N. Wiltbank. 1969. Reproductive performance of 2-year-old Hereford and Angus heifers as influenced by pre- and post-calving energy intake. J. Anim. Sci. 29:719-726.

Dunn, T. G. and C. C. Kaltenbach. 1980. Nutrition and postpartum interval of ewe, sow and cow. J. Anim. Sci. 51(Suppl.2):29-39.

Dyer, C. J., J. M. Simmons, R. L. Matteri, and D. H. Keisler. 1997. Leptin receptor mRNA is expressed in ewe anterior pituitary and adipose tissues and is differentially expressed in hypothalamic regions of well-fed and feed-restricted ewes. Domest. Anim. Endocrinol. 14:119-128.

Eadson, M. P., J. M. Chesworth, M. B. E. Aboul-ela, and G. D. Henderson. 1985. The effect of undernutrition of beef cows on blood hormone and metabolite concentrations post partum. Reprod. Nut. Dev. 25:113-126.

Echternkamp, S. E., and W. Hansel. 1973. Concurrent changes in bovine plasma hormone levels prior to and during the first postpartum estrous cycle. J. Anim. Sci. 37:1362-1370.

Echternkamp, S. E., L. J. Spicer, K. E. Gregory, S. F. Canning, and J. M. Hammond. 1990. Concentrations of insulin-like growth factor-I in blood and ovarian follicular fluid of cattle selected twins. Biol. Reprod. 43:8-14.

Edwards, S. 1985. The effects of short term calf removal on pulsatile LH secretion in the postpartum cow. Theriogenology 23:777-785.

Eger, S., M. Shemesh, H. Schindler, S. Amir, and R. H. Foote. 1988. Characterization of short luteal cycles in the early post-partum period and their relation to reproductive performance of dairy cows. Anim. Reprod. Sci. 16:215-224.

Ehrhardt, R. A., R. M. Slepetis, J. Siegal-Willott, M. E. Van Amburgh, A. W. Bell, and Y. R. Boisclair. 2000. Development of a specific radioimmunoassay to measure physiological changes of circulating leptin in cattle and sheep. J. Endocrinol. 166:519-528.

Erb, R. E., A. H. Surve, C. J. Callahan, R. D. Randel, and H. A. Garverick. 1971. Reproductive steroids in the bovine. VII. Changes postpartum. J. Anim. Sci. 33:1060-1071.

Estienne, M. J., K. K. Schillo, S. M. Hileman, M. A. Green, S. H. Hayes, and J. A. Boling. 1990. Effects of free fatty acids on luteinizing hormone and growth hormone secretion in ovariectomized lambs. Endocrinology 126:1934-1940.

Fair, T., P. Hyttel, and T. Greve. 1995. Bovine oocyte diameter in relation to maturational competence and transcriptional activity. Mol. Reprod. Dev. 42:437-442.

Farin, C. E., C. L. Moeller, H. R. Sawyer, F. Gamboni, and G. D. Niswender. 1986. Morphometric analysis of cell types in the ovine corpus luteum throughout the estrous cycle. Biol. Reprod. 35:1299-1308.

Ferlay, A., and Y. Chilliard. 1999. Effects of the infusion of non-selective beta-, and selective beta1- or beta2-adrenergic agonists, on body fat mobilisation in underfed or overfed non-pregnant heifers. Reprod. Nutr. Dev. 39:409-421.

Fike, K. E., M. L. Day, E. K. Inskeep, J. E. Kinder, P. E. Lewis, R. E. Short, and H. D. Hafs. 1997. Estrus and luteal function in suckled beef cows that were anestrous when treated with an intravaginal device containing progesterone with or without a subsequent

injection of estradiol benzoate. J. Anim. Sci. 75:2009-2015.

Finn, P. D., M. J. Cunningham, K. Y. Pau, H. G. Spies, D. K. Clifton, and R. A. Steiner. 1998. The stimulatory effect of leptin on the neuroendocrine reproductive axis of the monkey. Endocrinology 139:4652-4662.

Floyd, L. N. 2001. Effects of the number of cows in estrus and confinement area on estrous behavior of beef cows. M. Sc. Thesis. Oklahoma State University.

Froesch, E. R., C. Schmid, J. Schwander, and J. Zapf. 1985. Actions of insulin-like growth factors. Annu. Rev. Physiol. 47:443-467.

Funston, R. N., G. E. Moss, and A. J. Roberts. 1995a. Insulin-like growth factor-I (IGF-I) and IGF-binding proteins in bovine sera and pituitaries at different stages of the estrous cycle. Endocrinology 136:62-68.

Funston, R. N., A. J. Roberts, D. L. Hixon, D. M. Hallford, D. W. Sanson, and G. E. Moss. 1995b. Effect of acute glucose antagonism on hypophyseal hormones and concentrations of insulin-like growth factor (IGF)-I and IGF-binding proteins in serum, anterior pituitary, and hypothalamus of ewes. Biol. Reprod. 52:1179-1186.

Gangwar, P. C., C. Branton, and D. L. Evans. 1965. Reproductive and physiological responses of Holstein heifers to controlled and natural climatic conditions. J. Dairy Sci. 48:222-227.

Garcia-Winder, M., K. Imakawa, M. L. Day, D. D. Zalesky, R. J. Kittok, and J. E. Kinder. 1986. Effects of suckling and low doses of estradiol on luteinizing hormone secretion during the postpartum period in beef cows. Domest. Anim. Endocrinol. 3:79-86.

Garmendia, J. C. 1984. Prepartum nutrition, plasma constituents and reproductive performance of beef cows. M. Sc. Thesis. Oklahoma State University.

Garmendia, J. C. 1986. Energy metabolites in blood, luteinizing hormone secretion and reproductive performance of beef cows. Ph.D. dissertation. Oklahoma State University.

Garverick, H. A., W. G. Zollers, and M. F. Smith. 1992. Mechanisms associated with corpus luteum lifespan in animals having normal or subnormal luteal function. Anim. Reprod. Sci. 28:111-124.

Gazal, O. S., L. S. Leshin, R. L. Stanko, M. G. Thomas, D. H. Keisler, L. L. Anderson, and G. L. Williams. 1998. Gonadotropin-releasing hormone secretion into third-ventricle cerebrospinal fluid in cattle: Correspondence with the tonic and surge release of luteinizing hormone and its tonic inhibition by suckling and Neuropeptide Y. Biol. Reprod. 59:676-683.

Ginther, O. J., L. Knopf, and J. P. Kastelic. 1989. Ovarian follicular dynamics in heifers during early pregnancy. Biol. Reprod. 41:247-254.

Ginther, O. J., K. Kot, L. J. Kulick, S. Martin, and M. C. Wiltbank. 1996. Relationships between FSH and ovarian follicular waves during the last six months of pregnancy in cattle. J. Reprod. Fertil. 108:271-279.

Glencross, R. G., M. J. Esslemont, M. J. Bryant, and G. S. Pope. 1981. Relationships between the incidence of pre-ovulatory behaviour and the concentrations of oestradiol-17B and progesterone in bovine plasma. Appl. Anim. Ethol. 7:141-148.

Goehring, T. B., L. R. Corah, and J. J. Higgins. 1989. Effects of energy and lasalocid on productivity of first-calf heifers. J. Anim. Sci. 67:1879-1888.

Graves, W. E., J. W. Lauderdale, E. R. Hauser, and L. E. Casida. 1968. Relation of postpartum interval to pituitary gonadotropins, ovarian follicular development and fertility of beef cows. Research Bulletin 270: Studies on the postpartum cow, University of Wisconsin, Madison, pp 23-26.

Grazzini, E., G. Guillon, B. Mouillac, and H. H. Zingg. 1998. Inhibition of oxytocin receptor function by direct binding of progesterone. Nature 392:509-512.

Griffith, M. K., and G. L. Williams. 1996. Roles of maternal vision and olfaction in suckling-mediated inhibition of luteinizing hormone secretion, expression of maternal selectivity, and lactational performance of beef cows. Biol. Reprod. 54:761-768.

Grimard, B., P. Humblot, A. A. Ponter, J. P. Mialot, D. Sauvant, and M. Thibier. 1995. Influence of postpartum energy restriction on energy status, plasma LH and oestradiol secretion and follicular development in suckled beef cows. J. Reprod. Fertil. 104:173-179.

Gutierrez, C. G., B. K. Campbell, and R. Webb. 1997a. Development of a long-term bovine granulosa cell culture system: induction and maintenance of estradiol production, response to follicle- stimulating hormone, and morphological characteristics. Biol. Reprod. 56:608-616.

Gutierrez, C. G., J. Oldham, T. A. Bramley, J. G. Gong, B. K. Campbell, and R. Webb. 1997b. The recruitment of ovarian follicles is enhanced by increased dietary intake in heifers. J. Anim. Sci. 75:1876-1884.

Gwynne, J. T., and J. F. III. Strauss. 1982. The role of lipoproteins in steroidogenesis and cholesterol metabolism in steroidogenic glands. Endocr. Rev. 3:299-329.

Hamilton, T. D., J. A. Vizcarra, R. P. Wettemann, B. E. Keefer, and L. J. Spicer. 1999. Ovarian function in nutritionally induced anoestrous cows: effect of exogenous gonadotrophin-releasing hormone in vivo and effect of insulin and insulin-like growth factor I in vitro. J. Reprod. Fertil. 117:179-187.

Hansel, W., H. W. Alila, J. P. Dowd, and R. A. Milvae. 1991. Differential origin and control mechanisms in small and large bovine luteal cells. J. Reprod. Fertil. (Suppl. 43):77-89.

Hansel, W., and E. M. Convey. 1983. Physiology of the estrous cycle. J. Anim. Sci. 57(Suppl. 2):404-424.

Haresign, W., G. R. Foxcroft, and G. E. Lamming. 1983. Control of ovulation in farm animals. J. Reprod. Fertil. 69:383-395.

Harmon, D. L. 1992. Impact of nutrition on pancreatic exocrine and endocrine secretion in ruminants: a review. J. Anim. Sci. 70:1290-1301.

Harrison, L. M., and R. D. Randel. 1986. Influence of insulin and energy intake on ovulation rate,

luteinizing hormone and progesterone in beef heifers. J. Anim. Sci. 63:1228-1235.

Hawkins, D. E., K. D. Niswender, G. M. Oss, C. L. Moeller, K. G. Odde, H. R. Sawyer, and G. D. Niswender. 1995. An increase in serum lipids increases luteal lipid content and alters the disapperance rate of progesterone in cows. J. Anim. Sci. 73:541-545.

Heiman, M. L., K. W. Sloop, Y. Chen, and J. F. Caro. 1999. Extension of neuroendocrine axes to include leptin. J. Anim. Sci. 77:33-42.

Helmer, S. D., and J. H. Britt. 1985. Mounting behavior as affected by stage of estrous cycle in Holstein heifers. J. Dairy Sci. 68:1290-1296.

Henao, G., M. Olivera-Angel, and J. G. Maldonado-Estrada. 2000. Follicular dynamics during postpartum anestrus and the first estrous cycle in suckled or non-suckled Brahman (Bos indicus) cows. Anim. Reprod. Sci. 63:127-136.

Henricks, D. M., J. F. Dickey, J. R. Hill, and W. E. Johnston. 1972. Plasma estrogen and progesterone levels after mating, and during late pregnancy and postpartum in cows. Endocrinology 90:1336-1342.

Henry, B. A., J. W. Goding, W. S. Alexander, A. J. Tilbrook, B. J. Canny, F. Dunshea, A. Rao, A. Mansell, and I. J. Clarke. 1999. Central administration of leptin to ovariectomized ewes inhibits food intake without affecting the secretion of hormones from the pituitary gland: evidence for a dissociation of effects on appetite and neuroendocrine function. Endocrinology 140:1175-1182.

Hightshoe, R. B., R. C. Cochran, L. R. Corah, G. H. Kiracofe, D. L. Harmon, and R. C. Perry. 1991. Effects of calcium soaps of fatty acids on postpartum reproductive function in beef cows. J. Anim. Sci. 69:4097-4103.

Hileman, S. M., K. K. Schillo, J. B. Kearnan, J. B. Hall, and S. Mohapatra. 1991. Effects of metabolic fuel restriction on patterns of LH and GH in ovariectomized lambs. Biol. Reprod. 44(Suppl. 1):87 (Abstr.).

Hill Jr, J. R., D. R. Lamond, D. M. Henricks, J. F. Dickey, and G. D. Niswender. 1970. The effects of undernutrition on ovarian function and fertility in beef heifers. Biol. Reprod. 2:78-84.

Hiney, J. K., S. R. Ojeda, and W. L. Dees. 1991. Insulin-like growth factor I: a possible metabolic signal involved in the regulation of female puberty. Neuroendocrinology 54:420-423.

Hiney, J. K., V. Srivastava, C. L. Nyberg, S. R. Ojeda, and W. L. Dees. 1996. Insulin-like growth factor I of peripheral origin acts centrally to accelerate the initiation of female puberty. Endocrinology 137:3717-3728.

Hinshelwood, M. M., D. J. Dierschke, and E. R. Hauser. 1985. Effect of suckling on the hypothalamic-pituitary axis in postpartum beef cows, independent of ovarian secretions. Biol. Reprod. 32:290-300.

Hoffman, D. P., J. S. Stevenson, and J. E. Minton. 1996. Restricting calf presence without suckling compared with weaning prolongs postpartum anovulation in beef cattle. J. Anim. Sci. 74:190-198.

Holland, M. D., and K. G. Odde. 1992. Factors affecting calf birth weight: a review. Theriogenology 38:769-798.

Houghton, P. L., R. P. Lemenager, L. A. Horstman, K. S. Hendrix, and G. E. Moss. 1990a. Effects of body composition, pre- and postpartum energy level and early weaning on reproductive performance of beef cows and preweaning calf gain. J. Anim. Sci. 68:1438-1446.

Houghton, P. L., R. P. Lemenager, G. E. Moss, and K. S. Hendrix. 1990b. Prediction of postpartum beef cow body composition using weigth to height ratio and visual body condition score. J. Anim. Sci. 68:1428-1437.

Houseknecht, K. L., C. A. Baile, R. L. Matteri, and M. E. Spurlock. 1998. The biology of Leptin: a review. J. Anim. Sci. 76:1405-1420.

Houseknecht, K. L., and C. P. Portocarrero. 1998. Leptin and its receptors: Regulators of whole-body energy homeostasis. Domest. Anim. Endocrinol. 15:457-475.

Houseknecht, K. L., C. P. Portocarrero, S. Ji, R. Lemenager, and M. E. Spurlock. 2000. Growth hormone regulates leptin gene expression in bovine adipose tissue: correlation with adipose IGF-1 expression. J. Endocrinol. 164:51-57.

Humblot, P., B. Grimard, O. Ribon, B. Khireddine, V. Dervishi, and M. Thibier. 1996. Sources of variation of post-partum cyclicity, ovulation and pregnancy rates in primiparous Charolais cows treated with Norgestomet implants and PMSG. Theriogenology 46:1085-1096.

Humphrey, W. D., C. C. Kaltenbach, T. G. Dunn, D. R. Koritnik, and G. D. Niswender. 1983. Characterization of hormonal patterns in the beef cow during postpartum anestrus. J. Anim. Sci. 56:445-453.

Hunter, M. G. 1991. Characteristics and causes of inadequate corpus luteum. J. Reprod. Fertil. (Suppl. 43):91-99.

Hurnik, J. F., and G. J. King. 1987. Estrous behavior in confined beef cows. J. Anim. Sci. 65:431-438.

Hurnik, J. F., G. J. King, and H. A. Robertson. 1975. Estrous and related behaviour in postpartum Holstein cows. Appl. Anim. Ethol. 2:55-68.

Imakawa, K., M. L. Day, D. D. Zalesky, A. Clutter, R. J. Kittok, and J. E. Kinder. 1987. Effects of 17-B estradiol and diets varying in energy on secretion of luteinizing hormone in beef heifers. J. Anim. Sci. 64:805-815.

Ingalls, W. G., E. M. Convey, and H. D. Hafs. 1973. Bovine serum LH, GH, and prolactin during late pregnancy, parturition and early lactation. Proc. Soc. Exp. Biol. Med. 143:161-164.

Ingvartsen, K. L. and J. B. Andersen. 2000. Integration of metabolism and intake regulation: a review focusing on periparturient animals. J. Dairy Sci. 83:1573-1597.

Inskeep, E. K., T. D. Braden, P. E. Lewis, M. Garcia-Winder, and G. D. Niswender. 1988. Receptors for luteinizing hormone and follicle-stimulating hormone in largest follicles of postpartum beef cows. Biol.

Reprod. 38:587-591.

Ireland, J. J., and J. F. Roche. 1982. Development of antral follicles in cattle after prostaglandin-induced luteolysis: changes in serum hormones, steroids in follicular fluid, and gonadotropin receptors. Endocrinology 111:2077-2085.

Jolly, P. D. 1995. Physiological effects of undernutrition on postpartum anoestrus in cows. J. Reprod. Fertil.(Suppl. 49):477-492.

Jones, J. I., and D. R. Clemmons. 1995. Insulin-like growth factors and their binding proteins: biological actions. Endocr. Rev. 16:3-34.

Kadokawa, H., D. Blache, Y. Yamada, and G. B. Martin. 2000. Relationships between changes in plasma concentrations of leptin before and after parturition and the timing of first post-partum ovulation in high-producing Holstein dairy cows. Reprod. Fertil. Dev. 12:405-411.

Kadokawa, H., and Y. Yamada. 2000. Effect of a long-lasting opioid receptor antagonist (naltrexone) on pulsatile LH release in early postpartum Holstein dairy cows. Theriogenology 54:75-81.

Kalra, S. P., B. Xu, M. C. Dube, and P. S. Kalra. 1998. Nutritional infertility: mismanagement in central neuropeptidergic and peripheral insulin-leptin signaling. Pennington Center Nutrition Series. Nutrition and Reproduction. pp. 25-40. Lousiana State University Press, Baton Rouge.

Katz, L. S., E. A. Oltenacu, and R. H. Foote. 1980. The behavioral responses in ovariectomized cattle to either estradiol, testosterone, androstenedione, or dihydrotestosterone. Horm. Behav. 14:224-235.

Kauter, K., M. Ball, P. Kearney, R. Tellam, and J. R. McFarlane. 2000. Adrenaline, insulin and glucagon do not have acute effects on plasma leptin levels in sheep: development and characterisation of an ovine leptin ELISA. J. Endocrinol. 166:127-135.

Keisler, D. H. and M. C. Lucy. 1996. Perception and interpretation of the effects of undernutrition on reproduction. J. Anim. Sci. 74 (Suppl.3):1-17.

Keisler, D. H., J. A. Daniel, and C. D. Morrison. 1999. The role of leptin in nutritional status and reproductive function. J. Reprod. Fert.(Suppl. 54):425-435.

Kendrick, K. W., T. L. Bailey, A. S. Garst, A. W. Pryor, A. Ahmadzadeh, R. M. Akers, W. E. Eyestone, R. E. Pearson, and F. C. Gwazdauskas. 1999. Effects of energy balance on hormones, ovarian activity, and recovered oocytes in lactating Holstein cows using transvaginal follicular aspiration. J. Dairy Sci. 82:1731-1740.

Kesler, D. J., H. A. Garverick, R. S. Youngquist, R. G. Elmore, and C. J. Bierschwal. 1977. Effect of days postpartum and endogenous reproductive hormones on GnRH-induced LH release in dairy cows. J. Anim. Sci. 46:797-803.

Kesler, D. J., T. R. Troxel, and D. L. Hixon. 1980. Effect of days postpartum and exogenous GnRH on reproductive hormone and ovarian changes in postpartum suckled beef cows. Theriogenology 13:287-295.

Kesner, J. S., V. Padmanabhan, and E. M. Convey. 1982. Estradiol induces and progesterone inhibits the preovulatory surges of luteinizing hormone and follicle-stimulating hormone in heifers. Biol. Reprod. 26:571-578.

King, G. J., J. F. Hurnik, and H. A. Robertson. 1976. Ovarian function and estrus in dairy cows during early lactation. J. Anim. Sci. 42:688-692.

Kiracofe, G. H. 1980. Uterine involution: its role in regulating postpartum intervals. J. Anim. Sci. 51 (Suppl. 2):16-28.

Kobayashi, Y., C. K. Boyd, C. J. Bracken, W. R. Lamberson, D. H. Keisler, and M. C. Lucy. 1999. Reduced growth hormone receptor(GHR) messenger ribonucleic acid in liver of periparturient cattle is caused by a specific down-regulation of GHR 1A that is associated with decreased insulin-like growth factor I. Endocrinology 140:3947-3954.

Komar, C. M., A. K. Berndtson, A. C. O. Evans, and J. E. Fortune. 2001. Decline in circulating estradiol during the periovulatory period is correlated with decreases

in estradiol and androgen, and in messenger RNA for P450 aromatase and P450 17α-hydroxylase, in bovine preovulatory follicles. Biol. Reprod. 64:1797-1805.

Kurz, S. G., R. M. Dyer, Y. Hu, M. D. Wright, and M. L. Day. 1990. Regulation of luteinizing hormone secretion in prepubertal heifers fed an energy-deficient diet. Biol. Reprod. 43:450-456.

Lalman, D. L., D. H. Keisler, J. E. Williams, E. J. Scholljegerdes, and D. M. Mallett. 1997. Influence of postpartum weight and body condition change on duration of anestrus by undernourished suckled beef heifers. J. Anim. Sci. 75:2003-2008.

Lalman, D. L., J. E. Williams, B. W. Hess, M. G. Thomas, and D. H. Keisler. 2000. Effect of dietary energy intake on milk production and metabolic hormones in thin, primiparous beef cows. J. Anim. Sci. 78:530-538.

Lamb, G. C., J. M. Lynch, D. M. Grieger, J. E. Minton, and J. S. Stevenson. 1997. Ad libitum suckling by an unrelated calf in the presence or absence of a cow's own calf prolongs postpartum anovulation. J. Anim. Sci. 75:2762-2769.

Lamb, G. C., B. L. Miller, J. M. Lynch, K. E. Thompson, J. S. Heldt, C. A. Loest, D. M. Grieger, and J. S. Stevenson. 1999. Twice daily suckling but not milking with calf presence prolongs postpartum anovulation. J. Anim. Sci. 77:2207-2218.

Lassoued, N., G. Khaldi, P. Chemineau, Y. Cognie, and J. Thimonier. 1997. Role of the uterus in early regression of corpora lutea induced by the ram effect in seasonally anoestrous Barbarine ewes. Reprod. Nutr. Dev. 37:559-571.

Lauderdale, J. W. 1986. A review of patterns of change in luteal function. J. Anim. Sci. 62(Suppl. 2):79-91.

LaVoie, V., D. K. Han, D. B. Foster, and E. L. Moody. 1981. Suckling effect on estrus and blood plasma progesterone in postpartum beef cows. J. Anim. Sci. 52:802-812.

Leeuwenberg, B. R., N. L. Hudson, L. G. Moore, P. R. Hurst, and K. P. McNatty. 1996. Peripheral and ovarian IGF-I

concentrations during the ovine oestrous cycle. J. Endocrinol. 148:281-289.

Legradi, G., C. H. Emerson, R. S. Ahima, J. S. Flier, and R. M. Lechan. 1997. Leptin prevents fasting-induced suppression of prothyrotropin-releasing hormone messenger ribonucleic acid in neurons of the hypothalamic paraventricular nucleus. Endocrinology 138:2569-2576.

Lents, C. A., F. J. White, D. L. Lalman, and R. P. Wettemann. 2000. Effects of body condition of beef cows at calving and protein supplementation on estrous behavior and follicle size. Okla. Agr. Exp. Sta. Res. Rep. P-980:164-168.

Lents, C. A., F. J. White, D. L. Lalman, and R. P. Wettemann. 2000. The effects of body condition and protein supplementation of postpartum beef cows on estrous behavior and follicle size. J. Anim. Sci. 78(Suppl.1):205 (Abstr.).

LeRoith, D., H. Werner, D. Beitner-Johnson, and C. T. Roberts Jr. 1995. Molecular and cellular aspects of the insulin-like growth factor I receptor. Endocr. Rev. 16:143-163.

Lesniak, M. A., J. M. Hill, W. Kiess, M. Rojeski, C. B. Pert, and J. Roth. 1988. Receptors for insulin-like growth factors I and II: autoradiographic localization in rat brain and comparison to receptors for insulin. Endocrinology 123:2089-2099.

Leung, K., V. Padmanamhan, L. J. Spicer, H. A. Tucker, R. E. Short, and E. M. Convey. 1986. Relationship between pituitary GnRH-binding sites and pituitary release of gonadotrophins in post-partum beef cows. J. Reprod. Fertil. 76:53-63.

Lindsay, D. R. and I. C. Fletcher. 1968. Sensory involvement in the recognition of lambs by their dams. Anim. Behav. 16:415-417.

Littell, R. C., G. A. Milliken, W. W. Stroup, and R. D. Wolfinger. 1996. SAS System for Mixed Models. SAS Inst. Inc., Cary, NC.

Lishman, A. W., S. M. Allison, R. L. Fogwell, R. L.

Butcher, and E. K. Inskeep. 1979. Follicular development and function of induced corpora lutea in underfed postpartum anestrous beef cows. J. Anim. Sci. 48:867-875.

Livingstone, C., H. Lyall, and G. W. Gould. 1995. Hypothalamic GLUT 4 expression: a glucose- and insulin-sensing mechanism? Mol. Cell. Endocrinol. 107:67-70.

Longo, K. M., Y. Sun, and A. C. Gore. 1998. Insulin-like growth factor-I effects on gonadotropin-releasing hormone biosynthesis in GT1-7 cells. Endocrinology 139:1125-1132.

Looper, M. L. 1999. Body energy reserves and steroids regulate gonadotropins and reproduction in beef cows. Ph.D. dissertation. Oklahoma State University.

Lucy, M. C., C. R. Staples, F. M. Michel, and W. W. Thatcher. 1991. Energy balance and size and number of ovarian follicles detected by ultrasonography in early postpartum dairy cows. J. Dairy Sci. 74:473-482.

Lussier, J. G., P. Matton, and J. J. Dufour. 1987. Growth rates of follicles in the ovary of the cow. J. Reprod. Fertil. 81:301-307.

Lyimo, Z. C., M. Nielen, W. Ouweltjes, T. A. M. Kruip, and F. van Eerdenburg. 2000. Relationship among estradiol, cortisol and intensity of estrous behavior in dairy cattle. Theriogenology 53:1783-1795.

Maciel, S. M., C. S. Chamberlain, R. P. Wettemann, and L. J. Spicer. 2001. Dexamethasone influences endocrine and ovarian function in dairy cattle. J. Dairy Sci. 84:1998-2009.

Mackey, D. R., J. M. Sreenan, J. F. Roche, and M. G. Diskin. 2000. The effect of progesterone alone or in combination with estradiol on follicular dynamics, gonadotropin profiles, and estrus in beef cows following calf isolation and restricted suckling. J. Anim. Sci. 78:1917-1929.

Maffei, M., J. Halaas, E. Ravussin, R. E. Pratley, G. H. Lee, Y. Zhang, H. Fei, S. Kim, R. Lallone, S. Ranganathan, P. A. Kern, and J. M. Friedman. 1995.

Leptin levels in human and rodent: measurement of plasma leptin and ob RNA in obese and weight-reduced subjects. Nat. Med. 1:1155-1161.

Maile, L. A., and J. M. P. Holly. 1999. Insulin-like growth factor binding protein (IGFBP) proteolysis: ocurrence, identification, role and regulation. Growth Horm. IGF Res. 9:85-95.

Malven, P. V., J. R. Parfet, D. W. Gregg, R. D. Allrich, and G. E. Moss. 1986. Relationships among concentrations of four opioid neuropeptides and luteinizing hormone-releasing hormone in neural tissues of beef cows following early weaning. J. Anim. Sci. 62:723-733.

Manikkam, M., M. D. Calder, B. E. Salfen, R. S. Youngquist, D. H. Keisler, and H. A. Garverick. 2001. Concentrations of steroids and expression of messenger RNA for steroidogenic enzymes and gonadotropin receptors in bovine ovarian follicles of first and second waves and changes in second wave follicles after pulsatile LH infusion. Anim. Reprod. Sci. 67:189-203.

Mann, G. E. 2001. Hormone control of prostaglandin $F_{2\alpha}$ production and oxytocin receptor concentrations in bovine endometrium in explant culture. Domest. Anim. Endocrinol. 20:217-226.

Marie, M., P. A. Findlay, L. Thomas, and C. L. Adam. 2001. Daily patterns of plasma leptin in sheep: effects of photoperiod and food intake. J. Endocrinol. 170:277-286.

Marston, T. T., K. S. Lusby, R. P. Wettemann, and H. T. Purvis. 1995. Effects of feeding energy or protein supplements before or after calving on performance of spring-calving cows grazing native range. J. Anim. Sci. 73:657-664.

Marston, T. T., D. D. Simms, R. R. Schalles, K. O. Zoellner, L. C. Martin, and G. M. Fink. 1992. Relationship of milk production, milk expected progeny difference, and calf weaning weight in angus and simmental cow-calf pairs. J. Anim. Sci. 70:3304-3310.

Martin, T. E., D. M. Henricks, J. R. J. Hill, and N. C.

Rawlings. 1978. Active immunization of the cow against oestradiol-17β. J. Reprod. Fertil. 53:173-178.

Mathew, S. R., W. P. McCaughey, A. D. Kennedy, N. J. Lewis, and G. H. Crow. 1999. Electronic monitoring of mounting behavior in beef cattle on pasture. Can. Vet. J. 40:796-798.

McClure, T. J., C. D. Nancarrow, and H. M. Radford. 1978. The effect of 2-deoxy-D-glucose on ovarian function of cattle. Aust. J. Biol. Sci. 31:183-186.

McCracken, J. A., E. E. Custer, and J. C. Lamsa. 1999. Luteolysis: a neuroendocrine-mediated event. Physiol. Rev. 79:263-323.

McCutcheon, S. N., and D. E. Bauman. 1986. Effect of chronic growth hormone treatment on responses to epinephrine and thyroprotein-releasing hormone in lactating cows. J. Dairy Sci. 69:44-51.

McDougall, S., C. R. Burke, K. L. MacMillan, and N. B. Williamson. 1992. The effect of pretreatment with progesterone on the oestrous response to oestradiol-17B benzoate in the post-partum dairy cow. In: Proc. N. Z. Soc. Anim. Prod. 52:157-160.

McGuire, M. A., J. L. Vicini, D. E. Bauman, and J. J. Veenheizen. 1992. Insulin-like growth factors and binding proteins in ruminants and their nutritional regulation. J. Anim. Sci. 70:2901-2910.

McNeilly, A. S. 1997. Lactation and fertility. J. Mammary Gland Biol. Neoplasia 2:291-298.

Minegishi, T., T. Hirakawa, H. Kishi, K. Abe, Y. Abe, T. Mizutani, and K. Miyamoto. 2000. A role of insulin-like growth factor I for follicle-stimulating hormone receptor expression in rat granulosa cells. Biol. Reprod. 62:325-333.

Moenter, S. M., C. J. Woodfill, and F. J. Karsch. 1991. Role of the thyroid gland in seasonal reproduction: thyroidectomy blocks seasonal suppression of neuroendocrine activity in ewes. Endocrinology 128:1337-1344.

Monget, P., and D. Monniaux. 1995. Growth factors and the

control of folliculogenesis. J. Reprod. Fertil.(Suppl. 49):321-333.

Morrison, C. D., J. A. Daniel, B. J. Holmberg, J. Djiane, N. Raver, A. Gertler, and D. H. Keisler. 2001. Central infusion of leptin into well-fed and undernourished ewe lambs: effects on feed intake and serum concentrations of growth hormone and luteinizing hormone. J. Endocrinol. 168:317-324.

Morrison, D. G., J. C. Spitzer, and J. L. Perkins. 1999. Influence of prepartum body condition score change on reproduction in multiparous beef cows calving in moderate body condition. J. Anim. Sci. 77:1048-1054.

Moss, G. E., J. R. Parfet, C. A. Marvin, R. D. Allrich, and M. A. Diekman. 1985. Pituitary concentrations of gonadotropins and receptors for GnRH in suckled beef cows at various intervals after calving. J. Anim. Sci. 60:285-293.

Murphy, B. D. 2000. Models of luteinization. Biol. Reprod. 63:2-11.

Murphy, M. G., M. P. Boland, and J. F. Roche. 1990. Pattern of follicular growth and resumption of ovarian activity in post-partum beef suckler cows. J. Reprod. Fertil. 90:523-533.

Murphy, M. G., W. J. Enright, M. A. Crowe, K. McConnell, L. J. Spicer, M. P. Boland, and J. F. Roche. 1991. Effect of dietary intake on pattern of growth of dominant follicles during the oestrous cycle in beef heifers. J. Reprod. Fert. 92:333-338.

Myers, T. R., D. A. Myers, D. W. Gregg, and G. E. Moss. 1989. Endogenous opioid suppression of release of luteinizing hormone during suckling in postpartum anestrous beef cows. Domest. Anim. Endocrinol. 6:183-190.

Nagatani, S., D. C. Bucholtz, K. Murahashi, M. A. Estacio, H. Tsukamura, D. L. Foster, and K. I. Maeda. 1996. Reduction of glucose availability suppresses pulsatile luteinizing hormone release in female and male rats. Endocrinology 137:1166-1170.

Nagatani, S., P. Guthikonda, R. C. Thompson, H. Tsukamura,

K. I. Maeda, and D. L. Foster. 1998. Evidence for GnRH regulation by leptin: Leptin administration prevents reduced pulsatile LH secretion during fasting. Neuroendocrinology 67:370-376.

Nagatani, S., Y. Zeng, D. H. Keisler, D. L. Foster, and C. A. Jaffe. 2000. Leptin regulates pulsatile luteinizing hormone and growth hormone secretion in the sheep. Endocrinology 141:3965-3975.

Nebel, R. L., M. L. McGillard, and W. L. Walker. 1996. Time of ovulation relative to mounting activity in dairy cattle. J. Dairy Sci. 79:1555-1561.

Nessan, G. K., and G. J. King. 1981. Sexual behaviour in ovariectomized cows treated with oestradiol benzoate and testosterone propionate. J. Reprod. Fertil. 61:171-178.

Nett, T. M. 1987. Function of the hypothalamic-hypophysial axis during the post-partum period in ewes and cows. J. Reprod. Fertil.(Suppl. 34):201-213.

Nett, T. M., D. Cermak, T. Braden, J. Manns, and G. Niswender. 1987. Pituitary receptors for GnRH and estradiol, and pituitary content of gonadotropins in beef cows. I. Changes during the estrous cycle. Domest. Anim. Endocrinol. 4:123-132.

Nett, T. M., D. Cermak, T. Braden, J. Manns, and G. D. Niswender. 1988. Pituitary receptors for GnRH and estradiol, and pituitary content of gonadotropins in beef cows. II. Changes during the postpartum period. Domest. Anim. Endocrinol. 5:81-89.

Nicholls, T. J., B. K. Follett, A. R. Goldsmith, and H. Pearson. 1988. Possible homologies between photorefractoriness in sheep and birds: the effect of thyroidectomy on the length of the ewe's breeding season. Reprod. Nut. Dev. 28:375-385.

Niswender, G. D., J. L. Juengel, P. J. Silva, M. K. Rollyson, and E. W. McIntush. 2000. Mechanisms controlling the function and life span of the corpus luteum. Physiol. Rev. 80:1-29.

Niswender, G. D., R. H. Schwall, T. A. Fitz, C. E. Farin, and H. R. Sawyer. 1985. Regulation of luteal function

in domestic ruminants: new concepts. Recent Prog. Horm. Res. 41:101-151.

Northey, D. L., and L. R. French. 1980. Effect of embryo removal and intrauterine infusion of embryonic homogenates on the lifespan of the bovine corpus luteum. J. Anim. Sci. 50:298-302.

NRC. 1996. Nutrient Requirements of Beef Cattle (7th Ed.). National Academy Press, Washington, DC.

O'Callaghan, D., H. Yaakub, P. Hyttel, L. J. Spicer, and M. P. Boland. 2000. Effect of nutrition and superovulation on oocyte morphology, follicular fluid composition and systemic hormone concentrations in ewes. J. Reprod. Fertil. 118:303-313.

Odde, K. G., H. S. Ward, G. H. Kiracofe, R. M. McKee, and R. J. Kittok. 1980. Short estrous cycles and associated serum progesterone levels in beef cows. Theriogenology 14:105-112.

Osoro, K., and I. A. Wright. 1992. The effect of body condition, live weight, breed, age, calf performance, and calving date on reproductive performance of spring- calving beef cows. J. Anim. Sci. 70:1661-1666.

Ostlund, R. E. J., J. W. Yang, S. Klein, and R. Gingerich. 1996. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. J. Clin. Endocrinol. Metab. 81:3909-3913.

Pennington, J. A., J. L. Albright, and C. J. Callahan. 1986. Relationships of sexual activities in estrous cows to different frequencies of observation and pedometer measurements. J. Dairy Sci. 69:2925-2934.

Perks, C. M., A. R. Peters, and D. C. Wathes. 1999. Follicular and luteal expression of insulin-like growth factors I and II and the type of 1 IGF receptor in the bovine ovary. J. Reprod. Fertil. 116:157-165.

Perry, R. C., L. R. Corah, R. C. Cochran, W. E. Beal, J. S. Stevenson, J. E. Minton, D. D. Simms, and J. R. Brethour. 1991a. Influence of dietary energy on follicular development, serum gonadotropins, and first postpartum ovulation in suckled beef cows. J. Anim. Sci. 69:3762-3773.

Perry, R. C., L. R. Corah, G. H. Kiracofe, J. S. Stevenson, and W. E. Beal. 1991b. Endocrine changes and ultrasonography of ovaries in suckled beef cows during resumption of postpartum estrous cycles. J. Anim. Sci. 69:2548-2555.

Peter, A. T., W. T. K. Bosu, R. M. Liptrap, and E. Cummings. 1989. Temporal changes in serum prostaglandin $F_{2\alpha}$ and oxytocin in dairy cows with short luteal phases after the first postpartum ovulation. Theriogenology 32:277-284.

Peters, A. R., and G. E. Lamming. 1984. Reproductive activity of the cow in the post-partum period. II. Endocrine patterns and induction of ovulation. Br. Vet. J. 140:269-280.

Peters, A. R., G. E. Lamming, and M. W. Fisher. 1981. A comparison of plasma LH concentrations in milked and suckling post- partum cows. J. Reprod. Fertil. 62:567-573.

Pieterse, M. C., K. A. Kappen, T. A. M. Kruip, and M. A. M. Taverne. 1988. Aspiration of bovine oocytes during transvaginal ultrasound scanning of the ovaries. Theriogenology 30:751-762.

Pinto Andrade, L., S. M. Rhind, I. A. Wright, S. R. McMillen, P. J. Goddard, and T. A. Bramley. 1995. Effects of infusion of GnRH pulses and level of body condition on ovarian function in postpartum beef cows. Anim. Reprod. Sci. 40:177-192.

Poretsky, L., and M. F. Kalin. 1987. The gonadotropic function of insulin. Endocr. Rev. 8:132-141.

Prado, R., S. M. Rhind, I. A. Wright, A. J. F. Russel, S. R. McMillen, A. J. Smith, and A. S. McNeilly. 1990. Ovarian follicle populations, steroidogenicity and micromorphology at 5 and 9 weeks post partum in beef cows in two levels of body condition. Anim. Prod. 51:103-108.

Pratt, B. R., J. G. Berardinelli, L. P. Stevens, and E. K. Inskeep. 1982. Induced corpora lutea in the postpartum beef cow. I. Comparison of gonadotropin releasing hormone and human chorionic gonadotropin and effects of progestogen and estrogen. J. Anim. Sci. 54:822-829.

Quesnel, H., A. Pasquier, A. M. Mounier, and A. Prunier. 2000. Feed restriction in cyclic gilts: gonadotrophin-independent effects on follicular growth. Reprod. Nut. Dev. 40:405-14.

Rabiee, A. R. and I. J. Lean. 2000. Uptake of glucose and cholesterol by the ovary of sheep and cattle and the influence of arterial LH concentrations. Anim. Reprod. Sci. 64:199-209.

Rabiee, A. R., I. J. Lean, J. M. Gooden, B. G. Miller, and R. J. Scaramuzzi. 1997. An evaluation of transovarian uptake of metabolites using arterio-venous difference methods in dairy cattle. Anim. Reprod. Sci. 48:9-25.

Rahe, C. H., R. E. Owens, J. L. Fleeger, H. J. Newton, and P. G. Harms. 1980. Pattern of plasma luteinizing hormone in the cyclic cow: dependence upon the period of the cycle. Endocrinology 107:498-503.

Rajamahendran, R., P. C. Lague, and R. D. Baker. 1979. Estrus and LH release in ovariectomized heifers following vaginal devices containing ovarian steroids. J. Anim. Sci. 49:554-559.

Rakestraw, J., K. S. Lusby, R. P. Wettemann, and J. J. Wagner. 1986. Postpartum weight and body condition loss and performance of fall-calving cows. Theriogenology 26:461-473.

Ramirez-Godinez, J. A., G. H. Kiracofe, R. M. McKee, R. R. Schalles, and R. J. Kittok. 1981. Reducing the incidence of short estrous cycles in beef cows with norgestomet. Theriogenology 15:613-623.

Ramirez-Godinez, J. A., G. H. Kiracofe, R. R. Schalles, and G. D. Niswender. 1982. Endocrine patterns in the postpartum beef cow associated with weaning: a comparison of the short and subsequent normal cycles. J. Anim. Sci. 55:153-158.

Randel, R. D. 1981. Effect of once-daily suckling on postpartum interval and cow-calf performance of first-calf Brahman x Hereford heifers. J. Anim. Sci. 53:755-757.

Randel, R. D. 1990. Nutrition and postpartum rebreeding in cattle. J. Anim. Sci. 68:853-862.

Rasby, R. J., R. P. Wettemann, R. D. Geisert, L. E. Rice, and C. R. Wallace. 1990. Nutrition, body condition and reproduction in beef cows: fetal and placental development, and estrogens and progesterone in plasma. J. Anim. Sci. 68:4267-4276.

Rasby, R. J., R. P. Wettemann, P. G. Harms, K. S. Lusby, and J. J. Wagner. 1992. GnRH in the infundibular stalk-median eminence is related to percentage body fat in carcasses of beef cows. Domest. Anim. Endocrinol. 9:71-76.

Rawlings, N. C., L. Weir, B. Todd, J. Manns, and J. H. Hyland. 1980. Some endocrine changes associated with the post-partum period of the suckling beef cow. J. Reprod. Fertil. 60:301-308.

Renaville, R., A. Devolder, S. Massart, M. Sneyers, A. Burny, and D. Portetelle. 1993. Changes in the hypophysial-gonadal axis during the onset of puberty in young bulls. J. Reprod. Fertil. 99:443-449.

Rhind, S. M., S. McMillen, W. A. McKelvey, F. F. Rodriguez-Herrejon, and A. S. McNeilly. 1989. Effect of the body condition of ewes on the secretion of LH and FSH and the pituitary response to gonadotrophin-releasing hormone. J. Endocrinol. 120:497-502.

Richards, M. W., L. J. Spicer, and R. P. Wettemann. 1995. Influence of diet and ambient temperature on bovine serum insulin-like growth factor-I and thyroxine: relationships with non-sterified fatty acids, glucose, insulin, luteinizing hormone and progesterone. Anim. Reprod. Sci. 37:267-279.

Richards, M. W., J. C. Spitzer, and M. B. Warner. 1986. Effect of varying levels of postpartum nutrition and body condition at calving on subsequent reproductive performance in beef cattle. J. Anim. Sci. 62:300-306.

Richards, M. W., R. P. Wettemann, and H. M. Schoenemann. 1989a. Nutritional anestrus in beef cows: body weight change, body condition, luteinizing hormone and ovarian activity. J. Anim. Sci. 67:1520-1526.

Richards, M. W., R. P. Wettemann, and H. M. Schoenemann. 1989b. Nutritional anestrus in beef cows: concentrations of glucose and nonsterified fatty acids

in plasma and insulin in serum. J. Anim. Sci. 67:2354-2362.

Richards, M. W., R. P. Wettemann, L. J. Spicer, and G. L. Morgan. 1991. Nutritional anestrus in beef cows: effects of body condition and ovariectomy on serum luteinizing hormone and insulin-like growth factor-I. Biol. Reprod. 44:961-966.

Riley, G. M., A. R. Peters, and G. E. Lamming. 1981. Induction of pulsatile LH release, FSH release and ovulation in post- partum acyclic beef cows by repeated small doses of Gn-RH. J. Reprod. Fertil. 63:559-565.

Roberts, A. J., R. N. Funston, and G. E. Moss. 2001. Insulin-like growth factor binding proteins in the bovine anterior pituitary. Endocrine 14:399-406.

Roberts, A. J., R. A. Nugent III, J. Klindt, and T. G. Jenkins. 1997. Circulating insulin-like growth factor I, insulin-like growth factor binding proteins, growth hormone, and resumption of estrus in postpartum cows subjected to dietary energy restriction. J. Anim. Sci. 75:1909-1917.

Roche, J. F., M. A. Crowe, and M. P. Boland. 1992. Postpartum anoestrus in dairy and beef cows. Anim. Reprod. Sci. 28:371-378.

Roche, J. F., D. Mackey, and M. G. Diskin. 2000. Reproductive management of postpartum cows. Anim. Reprod. Sci. 60-61:703-712.

Rund, L. A., L. S. Leshin, F. N. Thompson, G. B. Rampacek, and T. E. Kiser. 1989. Influence of the ovary and suckling on luteinizing hormone response to naloxone in postpartum beef cows. J. Anim. Sci. 67:1527-1531.

Russel, A. J. F., and I. A. Wright. 1983. The use of blood metabolites in the determination of energy status in beef cows. Anim. Prod. 37:335-343.

Rutter, L. M., and J. G. Manns. 1987. Hypoglycemia alters pulsatile luteinizing hormone secretion in the postpartum beef cow. J. Anim. Sci. 64:479-488.

Rutter, L. M., and J. G. Manns. 1988. Follicular phase

gonadotropin secretion in cyclic postpartum beef cows with phlorizin-induced hypoglycemia. J Anim Sci 66:1194-1200.

Rutter, L. M., and J. G. Manns. 1991. Insulin-like growth factor I in follicular development and function in postpartum beef cows. J. Anim. Sci. 69:1140-1146.

Rutter, L. M., and R. D. Randel. 1986. Nonpuberal estrus in beef heifers. J. Anim. Sci. 63:1049-1053.

Rutter, L. M., R. Snopek, and J. G. Manns. 1989. Serum concentrations of IGF-I in postpartum beef cows. J. Anim. Sci. 67:2060-2066.

Ryan, D. P., R. A. Spoon, M. K. Griffith, and G. L. Williams. 1994. Ovarian follicular recruitment, granulosa cell steroidogenic potential and growth hormone/insulin-like growth factor-I relationships in suckled beef cows consuming high lipids diets: effects of graded differences in body condition maintained during the puerperium. Domest. Anim. Endocrinol. 11:161-174.

Salfen, B. E., F. N. Kojima, J. F. Bader, M. F. Smith, and H. A. Garverick. 2001. Effect of short-term calf removal at three stages of a follicular wave on fate of a dominant follicle in postpartum beef cows. J. Anim. Sci. 79:2688-2697.

Savio, J. D., M. P. Boland, N. Hynes, and J. F. Roche. 1990a. Resumption of follicular activity in the early post-partum period of dairy cows. J. Reprod. Fertil. 88:569-579.

Savio, J. D., M. P. Boland, and J. F. Roche. 1990b. Development of dominant follicles and length of ovarian cycles in post-partum dairy cows. J. Reprod. Fertil. 88:581-591.

Savio, J. D., W. W. Thatcher, L. Badinga, R. L. de la Sota, and D. Wolfenson. 1993. Regulation of dominant follicle turnover during the oestrous cycle in cows. J. Reprod. Fertil. 97:197-203.

Scaramuzzi, R. J., J. F. Murray, J. A. Downing, and B. K. Campbell. 1999. The effects of exogenous growth hormone on follicular steroid secretion and ovulation

rate in sheep. Domest. Anim. Endocrinol. 17:269-277.

Schallenberger, E. 1985. Gonadotrophins and ovarian steroids in cattle. III. Pulsatile changes of gonadotrophin concentrations in the jugular vein post partum. Acta Endocrinol. (Copenh) 109:37-43.

Schallenberger, E., U. Oerterer, and G. Hutterer. 1982. Neuroendocrine regulation of postpartum function. In: H. Karg and E. Schallenberger (Eds.). Factors influencing fertility in the postpartum cow. Curr. Topics Vet. Med. Anim. Sci. Vol. 20, pp 123-147. Martinus Nijhoff Publ., The Netherlands.

Schams, D., B. Berisha, M. Kosmann, and W. M. Amselgruber. 2002. Expression and localization of IGF family members in bovine antral follicles during final growth and in luteal tissue during different stages of estrous cycle and pregnancy. Domest. Anim. Endocrinol. 22:51-72.

Schams, D., E. Schallenberger, Ch. Menzer, J. Stangl., K. Zottmeier, B. Hoffmann, and H. Karg. 1978. Profiles of LH, FSH and progesterone in postpartum dairy cows and their relationship to the commencement of cyclic functions. Theriogenology 10:453-467.

Schillo, K. K. 1992. Effects of dietary energy on control of luteinizing hormone secretion in cattle and sheep. J. Anim. Sci. 70:1271-1282.

Schneider, J. E., R. M. Blum, and G. N. Wade. 2000. Metabolic control of food intake and estrous cycles in syrian hamsters. I. Plasma insulin and leptin. Am. J. Physiol. Regul. Integr. Comp. Physiol. 278:R476-R485.

Schneider, J. E., M. D. Goldman, S. Tang, B. Bean, H. Ji, and M. I. Friedman. 1998. Leptin indirectly affects estrous cycles by increasing metabolic fuel oxidation. Horm. Behav. 33:217-228.

Schneider, J. E., and G. N. Wade. 1989. Availability of metabolic fuels controls estrous cyclicity of Syrian hamsters. Science 244:1326-1328.

Schreihofer, D. A., F. Renda, and J. L. Cameron. 1996. Feeding-induced stimulation of luteinizing hormone secretion in male rhesus monkeys is not dependent on a

rise in blood glucose concentration. Endocrinology 137:3770-3776.

Selk, G. E., R. P. Wettemann, K. S. Lusby, J. W. Oltjen, S. L. Mobley, R. J. Rasby, and J. C. Garmendia. 1988. Relationships among weight change, body condition and reproductive performance of range beef cows. J. Anim. Sci. 66:3153-3159.

Sen, K. K., S. Azhar, and K. M. J. Menon. 1979. Evidence for involvement of an energy-dependent process in gonadotropin-releasing hormone-stimulated luteinizing hormone release by rat anterior pituitary. Endocrinology 105:1158-1161.

Senger, P. L. 1997. Pathways to Pregnancy and Parturition. Current Conceptions, Inc., Pullman, WA.

Sharma, B. K., M. J. Vandehaar, and N. K. Ames. 1994. Expression of insulin-like growth factor-I in cows at different stages of lactation and in late lactation cows treated with somatotropin. J. Dairy Sci. 77:2232-2241.

Sheffel, C. E., B. R. Pratt, W. L. Ferrell, and E. K. Inskeep. 1982. Induced corpora lutea in the postpartum beef cow. II. Effects of treatment with progestogen and gonadotropins. J. Anim. Sci. 54:830-836.

Shi, Z. D., and G. K. Barrell. 1992. Requirement of thyroid function for the expression of seasonal reproductive and related changes in red deer (Cervus elaphus) stags. J Reprod Fertil 94:251-259.

Shintani, M., H. Nishimura, S. Yonemitsu, H. Masuzaki, Y. Ogawa, K. Hosoda, G. Inoue, Y. Yoshimasa, and K. Nakao. 2000. Downregulation of leptin by free fatty acids in rat adipocytes: effects of triacsin C, palmitate, and 2-bromopalmitate. Metabolism 49:326-330.

Shively, T. E., and G. L. Williams. 1989. Patterns of tonic luteinizing hormone release and ovulation frequency in suckled anestrous beef cows following varying intervals of temporary weaning. Domest. Anim. Endocrinol. 6:379-387.

Short, R. E., R. A. Bellows, E. L. Moody, and B. E.

Howland. 1972. Effects of suckling and mastectomy on bovine postpartum reproduction. J. Anim. Sci. 34:70-74.

Short, R. E., R. A. Bellows, R. B. Staigmiller, J. G. Berardinelli, and E. E. Custer. 1990. Physiological mechanisms controlling anestrus and infertility in postpartum beef cattle. J. Anim. Sci. 68:799-816.

Short, R. E., R. D. Randel, R. B. Staigmiller, and R. A. Bellows. 1979. Factors affecting estrogen-induced LH release in the cow. Biol. Reprod. 21:683-689.

Silveira, P. A., R. A. Spoon, and G. L. Williams. 1993. Evidence for maternal behavior as a requisite link in suckling-mediated anovulation in cows. Biol. Reprod. 49:1338-1346.

Silvia, W. J., G. S. Lewis, J. A. McCracken, W. W. Thatcher, and L., Wilson. 1991. Hormonal regulation of uterine secretion of prostaglandin $F_{2\alpha}$ during luteolysis in ruminants. Biol. Reprod. 45:655-663.

Simpson, R. B., C. C. Chase Jr, L. J. Spicer, R. K. Vernon, A. C. Hammond, and D. O. Rae. 1994. Effect of exogenous insulin on plasma and follicular insulin-like growth factor I, insulin-like growth factor binding protein activity, follicular oestradiol and progesterone, and follicular growth in superovulated Angus and Brahman cows. J. Reprod. Fertil. 102:483-492.

Sinclair, K. D., P. J. Broadbent, and J. S. M. Hutchinson. 1994. The effect pre- and post-partum energy and protein supply on the blood metabolites and reproductive performance of single- and twin-suckling beef cows. Anim. Prod. 59:391-400.

Sirois, J., and J. E. Fortune. 1990. Lengthening the bovine estrous cycle with low levels of exogenous progesterone: a model for studying ovarian follicular dominance. Endocrinology 127:916-925.

Smidt, D., and E. Farries. 1982. The impact of lactational performance on post-partum fertility in dairy cows. 20:358-383.

Snijders, S. E., P. Dillon, D. O'Callaghan, and M. P.

Boland. 2000. Effect of genetic merit, milk yield, body condition and lactation number on in vitro oocyte development in dairy cows. Theriogenology 53:981-989.

Snyder, J. L., J. A. Clapper, A. J. Roberts, D. W. Sanson, D. L. Hamernik, and G. E. Moss. 1999. Insulin-like growth factor-I, insulin-like growth factor-binding proteins, and gonadotropins in the hypothalamic-pituitary axis and serum of nutrient-restricted ewes. Biol. Reprod. 61:219-224.

Soldani, R., A. Cagnaci, A. Paoletti, S. Yen, and G. Melis. 1995. Modulation of anterior pituitary luteinizing hormone response to gonadotropin-releasing hormone by insulin-like growth factor I in vitro. Fertil. Steril. 64:634-637.

Southee, J. A., M. G. Hunter, A. S. Law, and W. Haresign. 1988. Effect of hysterectomy on the short life-cycle corpus luteum produced after GnRH-induced ovulation in the anoestrous ewe. J. Reprod. Fertil. 84:149-155.

Spicer, L. J. 2001. Leptin: a possible metabolic signal affecting reproduction. Domest. Anim. Endocrinol. 21: 251-270.

Spicer, L. J., J. Alonso, and C. S. Chamberlain. 2001. Effects of thyroid hormones on bovine granulosa and thecal cell function in vitro: dependence on onsulin and gonadotropins. J. Dairy Sci. 84:1069-1076.

Spicer, L. J., E. Alpizar, and S. E. Echternkamp. 1993a. Effects of insulin, insulin-like growth factor-I, and gonadotropins on bovine granulosa cell proliferation, progesterone production, estradiol production, and(or) insulin-like growth factor I production in vitro. J. Anim. Sci. 71:1232-1241.

Spicer, L. J., E. Alpizar, and S. E. Echternkamp. 1993b. Effects of insulin, insulin-like growth factor I, and gonadotropins on bovine granulosa cell proliferation, progesterone production, estradiol production, and(or) insulin-like growth factor I production in vitro. J. Anim. Sci. 71:1232-1241.

Spicer, L. J., E. Alpizar, and R. E. Stewart. 1994. Evidence for an inhibitory effect of insulin-like growth factor-I and -II on insulin-stimulated

steroidogenesis by nontransformed ovarian granulosa cells. Endocrine 2:735-739.

Spicer, L. J., C. C. Chase Jr, and L. M. Rutter. 2002. Relationship between serum insulin-like growth factor-I and genotype during the postpartum interval in beef cows. J. Anim. Sci. 80:716-722.

Spicer, L. J., E. M. Convey, K. Leung, R. E. Short, and H. A. Tucker. 1986a. Anovulation in postpartum suckled beef cows. II. Associations among binding of 125I-labeled gonadotropins to granulosa and thecal cells, and concentrations of steroids in serum and various sized ovarian follicles. J. Anim. Sci. 62:742-750.

Spicer, L. J., E. M. Convey, H. A. Tucker, and S. E. Echternkamp. 1986b. Effects of intermittent injections of LHRH on secretory patterns of LH and FSH and ovarian follicular growth during postpartum anovulation in suckled beef cows. J. Anim. Sci. 62:1317-1323.

Spicer, L. J., M. A. Crowe, D. J. Prendiville, D. Goulding, and W. J. Enright. 1992. Systemic but not intraovarian concentrations of insulin-like growth factor-I are affected by short-term fasting. Biol. Reprod. 46:920-925.

Spicer, L. J. and S. E. Echternkamp. 1986. Ovarian follicular growth, function and turnover in cattle: a review. J. Anim. Sci. 62:428-451.

Spicer, L. J., and S. E. Echternkamp. 1995. The ovarian insulin and insulin-like growth factor system with an emphasis on domestic animals. Domest. Anim. Endocrinol. 12:223-245.

Spicer, L. J., S. E. Echternkamp, S. F. Canning, and J. M. Hammond. 1988. Relationship between concentrations of immunoreactive insulin-like growth factor-I in follicular fluid and various biochemical markers of differentiation in bovine antral follicles. Biol. Reprod. 39:573-580.

Spicer, L. J., and W. J. Enright. 1991. Concentrations of insulin-like growth factor I and steroids in follicular fluid of preovulatory bovine ovarian follicles: effect of daily injections of a growth

hormone-releasing factor analog and(or) thyrotropin-releasing hormone. J. Anim. Sci. 69:1133-1139.

Spicer, L. J., W. J. Enright, M. G. Murphy, and J. F. Roche. 1991. Effect of dietary intake on concentrations of insulin-like growth factor-I in plasma and follicular fluid, and ovarian function in heifers. Domest. Anim. Endocrinol. 8:431-437.

Spicer, L. J., and C. C. Francisco. 1997. The adipose obese gene product, leptin: Evidence of a direct inhibitory role in ovarian function. Endocrinology 138:3374-3379.

Spicer, L. J., and C. C. Francisco. 1998. Adipose obese gen product, leptin, inhibits bovine ovarian thecal cell steroidogenesis. Biol. Reprod. 58:207-212.

Spicer, L. J., K. Leung, E. M. Convey, J. Gunther, R. E. Short, and H. A. Tucker. 1986c. Anovulation in postpartum suckled beef cows. I. Association among size and numbers of ovarian follicles, uterine involution, and hormones in serum and follicular fluid. J. Anim. Sci. 62:734-741.

Spicer, L. J., W. B. Tucker, and G. D. Adams. 1990. Insulin-like growth factor-I in dairy cows: Relationships among energy balance, body condition, ovarian activity, and estrous behavior. J. Dairy Sci. 73:929-937.

Spitzer, J. C., D. G. Morrison, R. P. Wettemann, and L. C. Faulkner. 1995. Reproductive responses and calf birth and weaning weights as affected by body condition at parturation and postpartum weight gain in primiparous beef cows. J. Anim. Sci. 73:1251-1257.

Spitzer, J. C., G. D. Niswender, Jr. G. E. Seidel, and J. N. Wiltbank. 1978. Fertilization and blood levels of progesterone and LH in beef heifers on restricted energy diet. J. Anim. Sci. 46:1071-1077.

Stagg, K., M. G. Diskin, J. M. Sreenan, and J. F. Roche. 1995. Follicular development in long-term anoestrous suckler beef cows fed two levels of energy postpartum. Anim. Reprod. Sci. 38:49-61.

Stagg, K., L. J. Spicer, J. M. Sreenan, J. F. Roche, and M. G. Diskin. 1998. Effect of calf isolation on

follicular wave dynamics, gonadotropin and metabolic hormone changes, and interval to first ovulation in beef cows fed either of two energy levels postpartum. Biol. Reprod. 59:777-783.

Staigmiller, R. B., B. G. England, R. Webb, R. E. Short, and R. A. Bellows. 1982. Estrogen secretion and gonadotropin binding by individual bovine follicles during estrus. J. Anim. Sci. 55:1473-1482.

Staples, C. R., and W. W. Thatcher. 1990. Relationship between ovarian activity and energy status during the early postpartum period of high producing dairy cows. J. Dairy Sci. 73:938-947.

Swanson, L. V., and H. D. Hafs. 1971. LH and prolactin in blood serum from estrus to ovulation in Holstein heifers. J. Anim. Sci. 33:1038-1041.

Steel, R. G. D., J. H. Torrie, and D. A. Dickey. 1997. Principles and Procedures of Statistics: A Biometrical Approach. 3rd ed. McGraw-Hill Inc., New York, NY.

Stevenson, J. S., E. L. Knoppel, J. E. Minton, B. E. Salfen, and H. A. Garverick. 1994. Estrus, ovulation, luteinizing hormone, and suckling-induced hormones in mastectomized cows with and without unrestricted presence of the calf. J. Anim. Sci. 72:690-699.

Stevenson, J. S., and G. C. Lamb. 1997. Interrelationships of lactation and postpartum anovulation in suckled and milked cows. Liv. Prod. Sci. 50:57-74.

Stewart, R. E., L. J. Spicer, T. D. Hamilton, and B. E. Keefer. 1995. Effects of insulin-like growth factor I and insulin on proliferation and on basal and lutenizing hormone-induced steroidogenesis of bovine thecal cells: involvement of glucose and receptors for insulin-like growth factor I and luteinizing hormone. J. Anim. Sci. 73:3719-3731.

Stewart, R. E., L. J. Spicer, T. D. Hamilton, B. E. Keefer, L. J. Dawson, G. L. Morgan, and S. E. Echternkamp. 1996. Levels of insulin-like growth factor (IGF) binding proteins, luteinizing hormone and IGF-I receptors, and steroids in dominant follicles during the first follicular wave in cattle exhibiting regular estrous cycles. Endocrinology 137:2842-2850.

Stewart, R. E., J. S. Stevenson, M. O. Mee, I. Rettmer, and J. E. Schneider. 1993. Induction of estrus after thyroidectomy in nonlactating Holstein cows. J. Dairy Sci. 76:2619-2623.

Stewart, R. E., J. S. Stevenson, and J. E. Minton. 1994. Serum hormones in response to estradiol and (or) progesterone in ovariectomized cows after thyroidectomy. Domest. Anim. Endocrinol. 11:13-24.

Stock, A. E., and J. E. Fortune. 1993. Ovarian follicular dominance in cattle: relationship between prolonged growth of the ovulatory follicle and endocrine parameters. Endocrinology 132:1108-1114.

Terqui, M., D. Chupin, D. Gauthier, N. Perez, J. Pelot, and P. Mauleon. 1982. Influence of management and nutrition on postpartum endocrine function and ovarian activity in cows. In: H. Karg and E. Schallenberger (Eds.). Factors influencing fertility in the postpartum cow. Curr. Topics Vet. Med. Anim. Sci. Vol. 20, pp 384-408.

Thissen, J.-P., J.-M. Ketelslegers, and L. E. Underwood. 1994. Nutritional regulation of the insulin-like growth factors. Endocr. Rev. 15:80-101.

Thomas, G. B., J. E. Mercer, T. Karalis, A. Rao, J. T. Cummins, and I. J. Clarke. 1990. Effect of restricted feeding on the concentrations of growth hormone (GH), gonadotropins, and prolactin (PRL) in plasma, and on the amounts of messenger ribonucleic acid for GH, gonadotropin subunits, and PRL in the pituitary glands of adult ovariectomized ewes. Endocrinology 126:1361-1367.

Totusek, R., D. W. Arnett, G. L. Holland, and J. V. Whiteman. 1973. Relation of estimation method, sampling interval and milk composition to milk yield of beef cows and calf gain. J. Anim. Sci. 37:153-158.

Trout, W. E., and P. V. Malven. 1988. Quantification of naloxone binding sites in brains from suckled beef cows during postpartum anestrus and resumption of estrous cycles. J. Anim. Sci. 66:954-960.

Troxel, T. R., and D. J. Kesler. 1984. Ability of indomethacin to alter prostaglandin metabolite

concentrations and to enhance the function of corpora lutea induced in postpartum suckled beef cows. J. Anim. Sci. 59:177-181.

Tsuchiya, T., Y. Nagao, A. Ozawa, M. Matsumoto, K. Sugahara, T. Kubo, and H. Kato. 1998. Decrease of the obese gene expression in bovine subcutaneous adipose tissue by fasting. Biosci. Biotechnol. Biochem. 62:2068-2069.

Vailes, L. D., S. P. Washburn, and J. H. Britt. 1992. Effects of various steroid milieus or physiological states on sexual behavior of Holstein cows. J. Anim. Sci. 70:2094-2103.

Vallet, J. L., G. E. Lamming, and M. Batten. 1990. Control of endometrial oxytocin receptor and uterine response to oxytocin by progesterone and oestradiol in the ewe. J. Reprod. Fertil. 90:625-634.

Vandehaar, M. J., B. K. Sharma, and R. L. Fogwell. 1995. Effect of dietary energy restriction on the expression of insulin-like growth factor-I in liver and corpus luteum of heifers. J. Dairy Sci. 78:832-841.

Vicini, J. L., F. C. Buonomo, J. J. Veenhuizen, M. A. Miller, D. R. Clemmons, and R. J. Collier. 1991. Nutrient balance and stage of lactation affect responses of insulin, insulin-like growth factors I and II, and insulin-like growth factor- binding protein 2 to somatotropin administration in dairy cows. J. Nutr. 121:1656-1664.

Viker, S. D., R. L. Larson, G. H. Kiracofe, R. E. Stewart, and J. S. Stevenson. 1993. Prolonged postpartum anovulation in mastectomized cows requires tactile stimulation by the calf. J. Anim. Sci. 71:999-1003.

Vizcarra, J. A., R. P. Wettemann, and D. K. Bishop. 1995. Relationship between puberty in heifers and the cessation of luteal activity after nutritional restriction. Animal Science 61:507-510.

Vizcarra, J. A., R. P. Wettemann, T. D. Braden, A. M. Turzillo, and T. M. Nett. 1997. Effect of gonadotropin-releasing hormone (GnRH) pulse frecuency on serum and pituitary concentrations of luteinizing hormone and follicle-stimulating hormone, GnRH

receptors, and messenger ribonucleic acid for gonadotropin subunits in cows. Endocrinology 138:594-601.

Vizcarra, J. A., R. P. Wettemann, and G. L. Morgan. 1999. Influence of dose, frequency, and duration of infused gonadotropin-releasing hormone on secretion of luteinizing hormone in nutritionally anestrous beef cows. Domest. Anim. Endocrinol. 16:171-181.

Vizcarra, J. A., R. P. Wettemann, J. C. Spitzer, and D. G. Morrison. 1998. Body condition at parturition and postpartum weight gain influence luteal activity and concentrations of glucose, insulin, and nonesterified fatty acids in plasma of primiparous beef cows. J. Anim. Sci. 76:927-936.

Voss, A. K., and J. E. Fortune. 1993. Levels of messenger ribonucleic acid for cytochrome P450 17α-hydroxylase and P450 aromatase in preovulatory bovine follicles decrease after the luteinizing hormone surge. Endocrinology 132:2239-2245.

Wagner, J. J., K. S. Lusby, J. W. Oltjen, J. Rakestraw, R. P. Wettemann, and L. E. Walters. 1988. Carcass composition in mature Hereford cows: estimation and effect on daily metabolizable energy requirement during winter. J. Anim. Sci. 66:603-612.

Wagner, W. C., and W. Hansel. 1969. Reproductive physiology of the post partum cow. I. Clinical and histological findings. J. Reprod. Fertil. 18:493-500.

Walters, D. L., C. C. Kaltenbach, T. G. Dunn, and R. E. Short. 1982a. Pituitary and ovarian function in postpartum beef cows. I. Effect of suckling on serum and follicular fluid hormones and follicular gonadotropin receptors. Biol. Reprod. 26:640-646.

Walters, D. L., and E. Schallenberger. 1984. Pulsatile secretion of gonadotrophins, ovarian steroids and ovarian oxytocin during the periovulatory phase of the oestrous cycle in the cow. J. Reprod. Fertil. 71:503-512.

Walters, D. L., R. E. Short, E. M. Convey, R. B. Staigmiller, T. G. Dunn, and C. C. Kaltenbach. 1982c. Pituitary and ovarian function in postpartum beef

cows. III. Induction of estrus, ovulation and luteal function with intermittent small-dose injections of GnRH. Biol. Reprod. 26:655-662.

Walters, D. L., R. E. Short, E. M. Convey, R. B. Staigmiller, T. G. Dunn, and C. C. Kaltenbach. 1982b. Pituitary and ovarian function in postpartum beef cows. II. Endocrine changes prior to ovulation in suckled and nonsuckled postpartum cows compared to cycling cows. Biol. Reprod. 26:647-654.

Webb, R., R. G. Gosden, E. E. Telfer, and R. M. Moor. 1999. Factors affecting folliculogenesis in ruminants. Animal Science 68:257-284.

Webster, J. R., S. M. Moenter, C. J. Woodfill, and F. J. Karsch. 1991. Role of the thyroid gland in seasonal reproduction. II. Thyroxine allows a season-specific suppression of gonadotropin secretion in sheep. Endocrinology 129:176-183.

Wehrman, M. E., T. H. Welsh Jr, and G. L. Williams. 1991. Diet-induced hyperlipidemia in cattle modifies the intrafollicular cholesterol environment, modulates ovarian follicular dynamics, and hastens the onset of postpartum luteal activity. Biol. Reprod. 45:514-522.

Werth, L. A., J. C. Whittier, S. M. Azzam, G. H. Deutscher, and J. E. Kinder. 1996. Relationship between circulating progesterone and conception at the first postpartum estrus in young primiparous beef cows. J. Anim. Sci. 74:616-619.

Wettemann, R. P. 1980. Postpartum endocrine function of cattle, sheep and swine. J. Anim. Sci. 51(Suppl. 2):2-14.

Wettemann, R. P., T. W. Beck, E. J. Turman, and R. L. Hintz. 1982. Endocrine response of postpartum anestrous beef cows to GnRH or PMSG. Theriogenology 18:599-613.

Wettemann, R. P., and I. Bossis. 2000. Energy intake regulates ovarian function in beef cattle. Proc. Am. Soc. Anim. Sci., 1999. Available at: htpp://asas.org/jas/symposia/proceedings/0938.pdf.

Wettemann, R. P., K. S. Lusby, J. C. Garmendia, M. W.

Richards, G. E. Selk, and R. J. Rasby. 1986. Postpartum nutrition, body condition and reproductive performance of first calf heifers. Okla. Agric. Exp. Sta. Res. Rep. MP-118:314-315.

Wettemann, R. P., E. J. Turman, R. D. Wyatt, and R. Totusek. 1978. Influence of suckling intensity on reproductive performance of range cows. J. Anim. Sci. 47:342-346.

Whisnant, C. S., T. E. Kiser, F. N. Thompson, and C. R. Barb. 1986a. Influence of calf removal on the serum luteinizing hormone response to naloxone in the postpartum beef cow. J. Anim. Sci. 63:561-564.

Whisnant, C. S., T. E. Kiser, F. N. Thompson, and C. R. Barb. 1986b. Opioid inhibition of luteinizing hormone secretion during the postpartum period in suckled beef cows. J. Anim. Sci. 63:1445-1448.

Whisnant, C. S., F. N. Thompson, T. E. Kiser, and C. R. Barb. 1986c. Effect of Naloxone on serum Luteinizing hormone, cortisol and prolactin concentrations in anestrous beef cows. J. Anim. Sci. 62:1340-1345.

White, F. J. 2000. Seasonal effects of estrous behavior and time of ovulation in beef cows. M. Sc. Thesis. Oklahoma State University.

White, F. J., C. A. Lents, N. H. Ciccioli, R. P. Wettemann, L. J. Spicer, and D. H. Keisler. 2001. Concentrations of leptin and insulin like growth factor-I (IGF-I) during acute nutritionally induced anovulation and realimentation. J. Anim. Sci. 79(Suppl. 1):34 (Abstr.).

White, F. J., R. P. Wettemann, M. L. Looper, T. M. Prado, and G. L. Morgan. 2002. Seasonal effects on estrous behavior and time of ovulation in nonlactating beef cows. J. Anim. Sci.(In press).

Whittier, J. C., D. C. Clanton, and G. H. Deutscher. 1988a. Effect of varying weight gain during the last trimester of gestation on productivity of beef heifers. Anim. Prod. 47:53-57.

Whittier, J. C., D. C. Clanton, and G. H. Deutscher. 1988b. Effect of varying weight gain during the last

trimester of gestation on productivity of beef heifers. Anim. Prod. 47:53-57.

Wiley, J. S., M. K. Petersen, R. P. Ansotegui, and R. A. Bellows. 1991. Production from first-calf beef heifers fed a maintenance or low level of prepartum nutrition and ruminally undegradable or degradable protein postpartum. J. Anim. Sci. 69:4279-4293.

Williams, G. L. 1989. Modulation of luteal activity in postpartum beef cows through changes in dietary lipids. J. Anim. Sci. 67:785-793.

Williams, G. L. 1990. Suckling as a regulator of postpartum rebreeding in cattle: a review. J. Anim. Sci. 68:831-852.

Williams, G. L., and M. K. Griffith. 1995. Sensory and behavioural control of gonadotrophin secretion during suckling-mediated anovulation in cows. J. Reprod. Fertil.(Suppl. 49):463-475.

Williams, G. L., J. Kotwica, W. D. Slanger, D. K. Olson, J. E. Tilton, and L. J. Johnson. 1982. Effect of suckling on pituitary responsiveness to gonadotropin- releasing hormone throughout the early postpartum period of beef cows. J. Anim. Sci. 54:594-602.

Williams, G. L., M. Koziorowski, R. G. Osborn, J. D. Kirsch, and W. D. Slanger. 1987. The postweaning rise of tonic luteinizing hormone secretion in anestrous cows is not prevented by chronic milking or the physical presence of the calf. Biol. Reprod. 36:1079-1084.

Williams, G. L., Jr. W. R. McVey, and J. F. Hunter. 1993. Mammary somatosensory pathways are not requiered for suckling-mediated inhibition of luteinizing hormone secretion and delay of ovulation in cows. Biol. Reprod. 49:1328-1337.

Williams, G. L., F. Talavera, B. J. Petersen, J. D. Kirsch, and J. E. Tilton. 1983. Coincident secretion of follicle-stimulating hormone and luteinizing hormone in early postpartum beef cows: effects of suckling and low-level increases of systematic progesterone. Biol. Reprod. 29:362-373.

Wilson, M. E. 1995. IGF-I administration advances the decrease in hypersensitivity to oestradiol negative feedback inhibition of serum LH in adolescent female rhesus monkeys. J. Endocrinol. 145:121-130.

Wiltbank, J. N. 1970. Research needs in beef cattle reproduction. J. Anim. Sci. 31:755-762.

Wiltbank, J. N., and A. C. Cook. 1958. The comparative reproductive performance of nursed and milked cows. J. Anim. Sci. 17:640-648.

Wiltbank, J. N., W. W. Rowden, J. E. Ingalls, K. E. Gregory, and R. M. Koch. 1962. Effect of energy level on reproductive performance phenomena on mature Hereford cows. J. Anim. Sci. 21:219-225.

Wiltbank, J. N., W. W. Rowden, J. E. Ingalls, and D. R. Zimmerman. 1964. Influence of post-partum energy level on reproductive performance of Hereford cows restricted in energy intake prior to calving. J. Anim. Sci. 23:1049-1053.

Wiltbank, M. C. 1994. Cell types and hormonal mechanisms associated with mid-cycle corpus luteum function. J. Anim. Sci. 72:1873-1883.

Woad, K. J., G. Baxter, C. O. Hogg, T. A. Bramley, R. Webb, and D. G. Armstrong. 2000. Expression of mRNA encoding insulin-like growth factors I and II and the type 1 IGF receptor in the bovine corpus luteum at defined stages of the oestrous cycle. J. Reprod. Fertil. 120:293-302.

Wright, I. A., S. M. Rhind, A. J. F. Russel, T. K. Whyte, A. J. McBean, and S. R. McMillen. 1987. Effects of body condition, food intake and temporary calf separation on the duration of the post-partum anoestrous period and associated LH, FSH, and prolactin concentrations in beef cows. Anim. Prod. 45:395-402.

Wright, I. A., S. M. Rhind, T. K. Whyte, and A. J. Smith. 1992. Effect of body condition at calving and feeding level after calving on LH profiles and duration of the post-partum anoestrous period in beef cows. Anim. Prod. 55:41-46.

Wright, I. A., S. M. Rhind, T. K. Whyte, A. J. Smith, S. R. McMillen, and R. Prado. 1990. Circulating concentrations of LH and FSH and pituitary responsiveness to GnRH in intact and ovariectomized suckled beef cows in two levels of body condition. Anim. Prod. 51:93-101.

Wright, I. A., and A. J. F. Russel. 1984. Estimation in vivo of the chemical composition of the bodies of mature cows. Anim. Prod. 38:33-44.

Yavas, Y. and J. S. Walton. 2000. Postpartum acyclicity in suckled beef cows: a review. Theriogenology 54:25-55.

Yelich, J. V., R. P. Wettemann, H. G. Dolezal, K. S. Lusby, D. K. Bishop, and L. J. Spicer. 1995. Effects of growth rate on carcass composition and lipid partitioning at puberty and growth hormone, insulin-like growth factor I, insulin, and metabolites before puberty in beef heifers. J. Anim. Sci. 73:2390-2405.

Yelich, J. V., R. P. Wettemann, T. T. Marston, and L. J. Spicer. 1996. Luteinizing hormone, growth hormone, insulin-like growth factor-I, insulin and metabolites before puberty in heifers fed to gain at two rates. Domest. Anim. Endocrinol. 13:325-338.

Yoshioka, K., C. Suzuki, S. Arai, S. Iwamura, and H. Hirose. 2001. Gonadotropin-releasing hormone in third ventricular cerebrospinal fluid of the heifer during the estrous cycle. Biol. Reprod. 64:563-570.

Yu, W. H., M. Kimura, A. Walczewska, S. Karanth, and S. M. McCann. 1997. Role of leptin in hypothalamic-pituitary function. Proc. Natl. Acad. Sci. USA 94:1023-1028.

Yuan, W., B. Bao, H. A. Garverick, R. S. Youngquist, and M. C. Lucy. 1998. Follicular dominance in cattle is associated with divergent patterns of ovarian gene expression for insulin-like growth factor (IGF)-I, IGF-II, and IGF binding protein-2 in dominant and subordinate follicles. Domest. Anim. Endocrinol. 15:55-63.

Zalesky, D. D., D. W. Forrest, N. H. McArthur, J. M. Wilson, D. L. Morris, and P. G. Harms. 1990. Suckling inhibits release of luteinizing hormone-releasing hormone from the bovine median eminence following

ovariectomy. J. Anim. Sci. 68:444-448.

Zamorano, P. L., V. B. Mahesh, L. M. De Sevilla, L. P. Chorich, G. K. Bhat, and D. W. Brann. 1997. Expression and localization of the leptin receptor in endocrine and neuroendocrine tissues of the rat. Neuroendocrinology 65:223-238.

Zhang, Y., R. Proenca, M. Maffei, M. Barone, and J. M. Friedman. 1994. Positional cloning of the mouse obese gene and its human homologue. Nature 372:425-432.

Zollers, W. G. Jr, H. A. Garverick, M. F. Smith, R. J. Moffatt, B. E. Salfen, and R. S. Youngquist. 1993. Concentrations of progesterone and oxytocin receptors in endometrium of postpartum cows expected to have a short or normal oestrous cycle. J. Reprod. Fertil. 97:329-337.

Zollers, W. G. Jr, H. A. Garverick, and M. F. Smith. 1989. Oxytocin-induced release of prostaglandin F2 alpha in postpartum beef cows: comparison of short versus normal luteal phases. Biol. Reprod. 41:262-267.

Zollers, W. G. Jr, H. A. Garverick, R. S. Youngquist, J. S. Ottobre, R. W. Silcox, J. P. Copelin, and M. F. Smith. 1991. In vitro secretion of prostaglandins from endometrium of postpartum beef cows expected to have short or normal luteal phases. Biol. Reprod. 44:522-526.

VITA 2

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