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## **Factors Affecting Calf Survival**

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### **INTRODUCTION**

Survival of the calf at or shortly after birth can be compromised leading to high death losses and a serious impact on net income for the cattle producer. This paper will briefly review some findings related to causes of death of the newborn calf.

Nutrition and Dystocia. Corah et al. (1975) reported effects of energy content of the gestation diet of the dam on birth weight and calf survival. Dams were placed on control diets that met NRC requirements and gestation diets that were isonitrogenous, but which supplied 50 or 65% of the NRC recommended levels of energy. Calf birth weights were affected by gestation diet, but there was no effect on dystocia incidence or severity. The percentage of dams showing estrus by the beginning of the breeding season was reduced as was pregnancy rate of the low-energy fed dams. The striking result of these studies was the diet effect on calf survival. One hundred percent of the calves from dams receiving adequate energy were alive at birth compared to 90% for calves from the low fed dams. At weaning, 100% of the calves from the adequate energy dams were alive compared to 71% from the energy deficient dams. The major cause of death loss from birth to weaning was scours, with a death loss of 19% due to this factor.

Bull et al. (1979) reported that cows maintained on diets deficient in crude protein produced calves that exhibited symptoms typical of Weak Calf Syndrome. Carstens et al. (1987) used heifers pregnant with single demi-embryos assigned to isocaloric gestation diets containing 91 or 55% of the NRC recommended crude protein allowance. Calves were placed in a metabolic chamber at 5 hours of age for 8 hours. Calves born to heifers that received the protein-restricted diets had 11.4% lower heat production than calves born to dams on the adequate protein diet. These findings extend those of Bull et al. (1979) and emphasize that inadequate protein intake during gestation can potentially result in calves more susceptible to cold stress.

Carstens' work was followed with studies of diet energy content and body condition of the dam on calf birth weight and the thermogenic ability of the calf. Ridder et al. (1991) used heifers pregnant with demi-embryos assigned to isonitrogenous gestation diets containing 100% or 70%

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decreasing to 40% NRC recommended energy allowance for the last 90 days of gestation. Calves were removed from the dam at birth and placed in a metabolic chamber to determine heat production. Heifer body weights and condition scores of the restricted heifers were lower as were calf birth weights from the energy-restricted dams. Heat production was lower for calves born to energy-restricted dams whether expressed as total heat produced or on a body size basis. Heifers with body condition scores of 5 or 6 had larger calves and their calves had higher heat production than calves from dams with condition scores of 4. Interval to calf standing, calving ease, and duration of Stage 2 of labor were not affected by ration energy content or body condition of the dam. They concluded that calves born to energy deficient dams may be more susceptible to cold stress.

Effects of gestation diet of the dam or dystocia on colostrum quantity and quality is not as clear. Ridder et al. (1991) reported energy content of the gestation diet had no significant effect on colostrum volume. Perino et al. (1995) found calves born to dams that experienced dystocia had numerically lower average plasma protein and immunoglobulin (IgG) concentrations than did calves born to dams experiencing normal parturition. Numerous studies have shown dystocia results in delay of standing by the dam and calf and suckling by the calf. The effects of diet and dystocia on antibody concentrations in the neonate may be a result of the time elapsing from birth to actual intake of colostrum as reported by Vermorel et al. (1989).

Anoxia. Dufty and Sloss (1977) studied the response of fetuses to anoxia by clamping the umbilical cord for 4, 6, or 8 minutes immediately before delivery. Four of six fetuses subjected to 4 minutes of anoxia survived whereas all others died when anoxia was 6 or 8 minutes duration. Clamping of the umbilicus resulted in fetal movement, release of meconium and changes in blood gases. Death occurred just before or soon after delivery. Calves that experienced anoxia and that survived delivery exhibited symptoms typical of Weak Calf Syndrome and tetanic spasms.

Vermorel et al. (1989) found heat production in dystocial calves was 22% and 13% lower at 2 and 13 hr after birth, respectively. Rectal temperatures of dystocial calves was decreased by an average of 5.2° F and increased slowly.

Recent studies at Colorado State University (Adams et al., 1995) have shown that dystocia, associated with varying degrees of fetal asphyxia resulted in major effects on immediate postnatal well being of the calf. Dystocial calves took 58 minutes longer to stand and 78 minutes longer to begin nursing than no-dystocia calves. Dystocial calves had elevated plasma lactate concentrations, depressed plasma glucose levels, but did not have reduced IgG and IgM concentrations at 48 hrs of age. This latter finding is in contrast to results of studies reported by Odde (1988) and others who found reduced antibody titers in calves that experienced dystocia. This result may have been masked in this study by the close supervision and assistance given the calves immediately following birth.

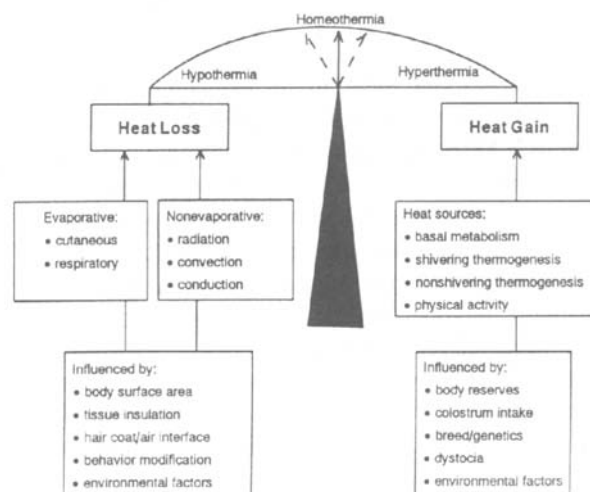
The similarity of these findings emphasize the importance of meeting nutrient needs of the dam during gestation. Deficiencies have a marked effect on calf viability and postpartum reproduction of the dam. This is also true regarding effects of dystocia. Reducing effects of prolonged labor will not only increase calf survival, but will improve subsequent rebreeding of the

dam.

**Thermogenesis:** Young (1983) reviewed the effects of cold stress in ruminants and concluded that failure to produce enough heat can obviously be fatal, but more often cold stress leads to the development of secondary changes and possibly disease. At parturition the calf moves from the controlled, warm uterine environment to the often-times hostile external environment. This transition necessitates many physiological actions to maintain normal body temperature (homeothermy) especially in seasonal environments typical of cattle production in northern regions. Climatic conditions affect neonatal survival and at low environmental temperatures mortality increases (Azzam et al., 1993). Patterson et al. (1987) found total neonatal mortality to range from approximately 4 to 13%. Mortality averaged 7% annually, with cold, wet weather being a major factor causing yearly variation in mortality rate.

Himms-Hagen (1990) and Carstens (1994) have reviewed thermal regulatory physiology in the newborn calf. The ability of the neonate to maintain normal core body temperature is a function of its ability to produce enough heat to balance the loss of heat by evaporative and nonevaporative heat losses. Nonevaporative heat loss involves flow of heat across temperature gradients from the metabolic heat sources in the animal to the environment by radiation, convection, and conduction. Evaporative heat loss occurs as water evaporates from the skin and respiratory tract surfaces. Evaporative heat losses are generally considered minimal except during wet weather and the immediate postnatal period when amniotic fluid is evaporated from the skin and respiratory tract of the neonate. The cold lethal limit is the critical ambient temperature below which the calf is unable to generate sufficient heat to offset heat loss, is no longer able to maintain thermal balance and hypothermia begins (Figure 1). Prolonged periods of exposure below the cold lethal limit will obviously result in death.

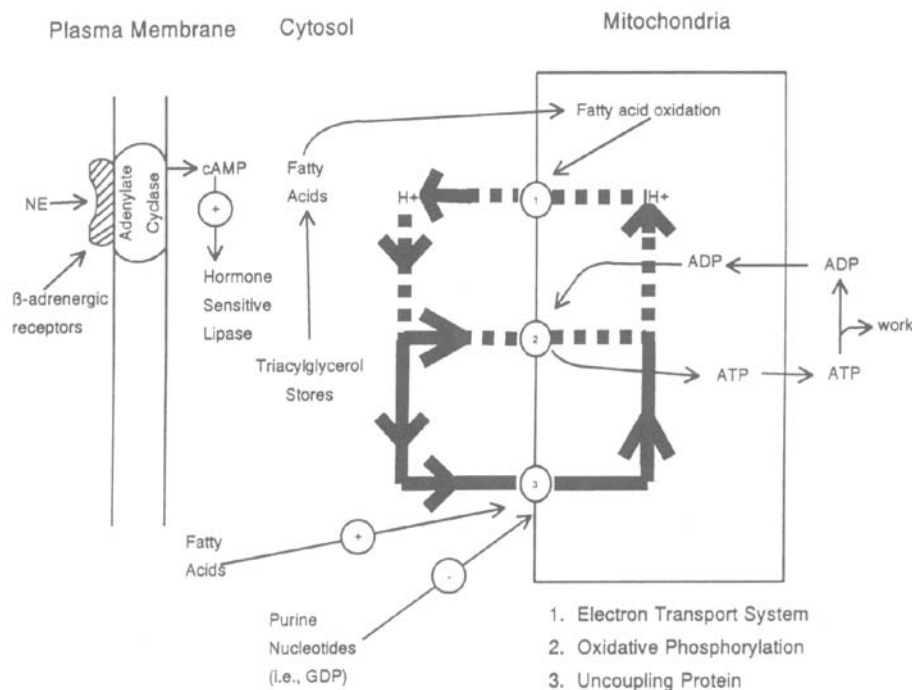
Figure 1. Thermal balance between heat loss and heat gain in neonatal ruminants (from Carstens, 1994).



Production of heat to maintain homeothermy in the neonate is dependent on shivering thermogenesis in the muscle and nonshivering thermogenesis in brown adipose tissue (BAT). BAT is a specialized organ whose thermogenic capacity is attributed to a unique uncoupling

protein (UCP) located in the mitochondria. The UCP in BAT "uncouples" mitochondrial respiration from oxidative phosphorylation (synthesis of adenosine triphosphate, ATP) thereby using energy generated to produce heat (Figure 2). It is estimated approximately 40 to 50% of the thermogenic response during summit metabolism is attributed to nonshivering thermogenesis with the balance (approximately 50 to 60%) attributed to shivering thermogenesis.

Figure 2. Mechanisms for brown adipose tissue thermogenesis (based on Himms-Hagen, 1990 and Carstens, 1994).



The two types of adipose tissue found in the neonatal ruminant are white and BAT. The primary function of white adipose tissue is storage and release of fatty acids for use as an energy source, while that of BAT is generation of heat through nonshivering thermogenesis. The key morphological feature of BAT is the high density of mitochondria, whereas mitochondria from white adipose tissue are relatively few in number. The major anatomic location of BAT is around the kidneys and appears to be similar in lambs, kids, and calves. BAT is extensively vascularized and brown adipocytes and the blood vessels are highly innervated by the sympathetic nervous system. The release of norepinephrine (NE) during cold exposure stimulates increased blood flow and thermogenesis in BAT. Even though BAT accounts for only 1.5-2% of body weight in newborn lambs, it can account for 22% of cardiac output and, as pointed out above, 40 to 50% of maximal thermogenesis during cold exposure. NE stimulation of BAT thermogenesis activates hormone-sensitive lipase which activates lipolysis to provide free fatty acids for mitochondrial respiration. Thus, NE release during sympathetic stimulation plays a critical role in the activation of BAT thermogenesis during cold exposure.

Work in our Laboratory has focused on methodology to increase neonatal calf survival.

Lammoglia et al. (1997) investigated effects of prepartum supplementation of dietary fat on cold tolerance and hormone and metabolite profiles in term and premature cold-exposed calves. In Study 1, dams received prepartum isocaloric-isonitrogenous diets containing 1.7 (basal control) or 4.9% dietary fat (basal plus safflower seeds with 37% oil and 80% of the oil was linoleic acid). Diets were fed from day 230 of gestation until parturition. At 4 hr of age, calves received jugular cannulae and were placed in a controlled temperature room at 32° F for 140 minutes. Rectal temperatures and blood samples were obtained throughout the cold exposure phase.

The procedures for Study 2 were similar to Study 1 with diet supplementation beginning on day 235 of gestation. Diets contained 1.5% (basal control) or 3.4% dietary fat (basal plus high linoleic safflower seeds). On day 260 of gestation, dams received either 0 or 4.4 lb. daily of Ponderosa pine needles. Dams receiving pine needles calved within 5 days after starting the pine needle feeding resulting in premature calves. Dams not receiving pine needles calved normally at an average gestation length of 283 days. Calves were placed in a controlled temperature room at 48° F for 200 minutes and blood samples and rectal temperatures were obtained throughout the time in the cold room. Results are summarized in Figures 3-8.

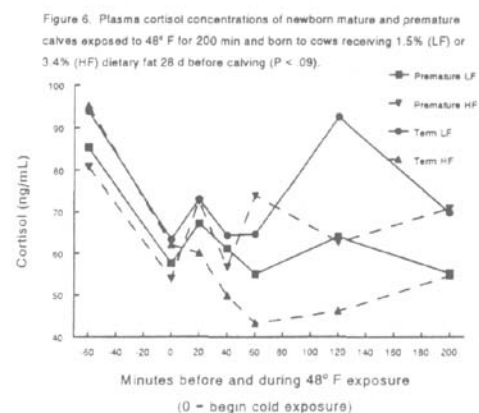
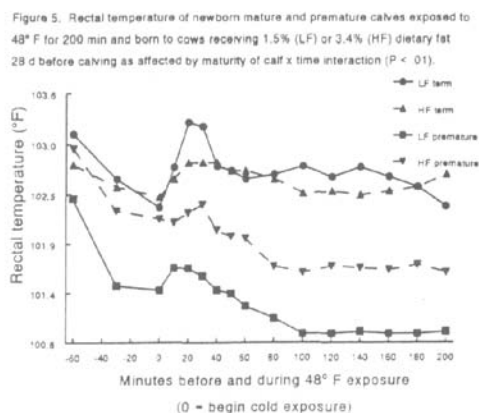
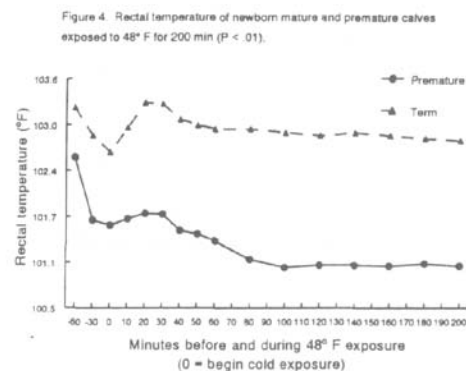
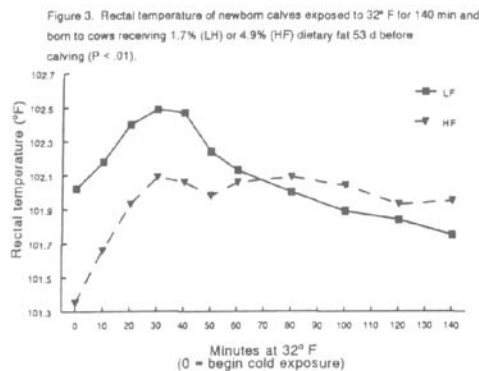


Figure 7. Plasma glucose concentrations of newborn calves exposed to 32° F for 140 min and born to cows receiving 1.7% (LF) or 4.9% (HF) dietary fat 53 d before calving (P < .06).

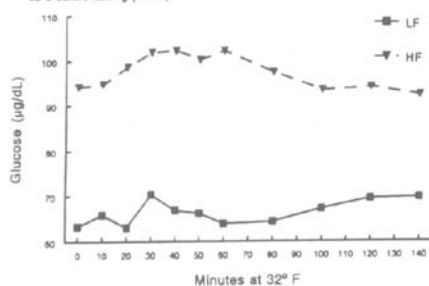
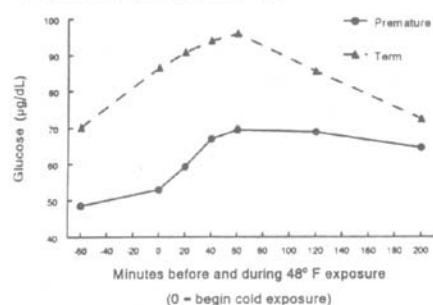


Figure 8. Plasma glucose concentrations of newborn mature and premature calves exposed to 48° F for 200 min and born to cows receiving 1.5% (LF) or 3.4% (HF) dietary fat 28 d before calving (P < .01).



Feeding 4.9% dietary fat during the last 53 days of gestation improved cold tolerance and increased plasma glucose concentrations in newborn calves. The increase in glucose may also be associated with improvement of cold thermogenesis and potentially improved neonatal survival. Feeding fat for the last 28 days of gestation in Study 2 did not influence cold tolerance in the newborn, premature calf, suggesting there may be a latent time period that must be exceeded before effects of supplemental fat can be obtained.

Additional data were obtained in Study 1. Results are summarized in Table 1. Calf birth weights from cows that received the high linoleic safflower diet were increased but calving difficulty scores and calf vigor scores were not affected. Pregnancy rates were greater in dams that received fat supplementation during gestation. This increase in pregnancy rate is of special interest since the dams had not received fat supplementation for an average of 55 days before the beginning of the 53 day breeding season which suggests a carry over effect on subsequent reproduction of supplemental fat during gestation.

Table 1. Effects of gestation dietary fat on calf and dam data

Trait	Diet	
	Control (1.7% fat)	Fat supplemented (4.9% fat, linoleic acid)
Number animal	35	35
Avg. birth wt. (lb)	79.6	83.3**
Dystocia score	1.7	1.8
Calf vigor score	1.1	1.3
Pregnancy rate (%)	57	77 <sup>t</sup>

<sup>t</sup>P = .06.

\*\*P < .01.

These results are similar to those reported by Williams (1989) and Lammoglia et al. (1996) who reported supplementing diets with fat affected hormone profiles, cholesterol concentrations, and ovarian follicular activity. Gambill et al. (1995) reported feeding 10%

supplemental fat (Alifet) to range beef cows before the breeding season resulted in an 18% increase in estrous activity and a 50% increase in pregnancy rate. Alifet contains 27% palmitic, 37% stearic, and 31% oleic fatty acid with a 67%:33% saturated-unsaturated fat composition.

The positive response to fat supplementation may be dependent on the lipid used. Anderson et al. (1992) found that fat supplementation starting 30 days prior to estrus induction by calf removal had no effect on interval to the induced ovulation or on luteal lifespan. This work did report an enhanced progesterone production in supplemented cows. Oss et al. (1993) found that fat supplementation from the third trimester of gestation through the third postpartum estrous cycle had no effect on gestation length, dystocia, calf vigor, or birth weight, but did cause longer postpartum intervals to first estrus (+30 days) and extended luteal life span. The lipid supplement used in these studies was a commercially available rumen escape fat (Megalac, Church and Dwight Co., Inc.) containing 51% palmitic, 35% oleic fatty acid with a 57%:43% saturated-unsaturated fat composition.

There is obviously much work to be done. Can other fat sources be used, how long must fat be fed, what about supplementation before or during the breeding season, what is the most economical level, etc.? But we believe the conclusion is justified that fat content, and possibly the specific fatty acid composition of the cow's diet is important for both the cow and the calf. When rations are formulated we need to be concerned about dietary fat in addition to the other nutrients such as protein, energy, vitamins, and minerals.

Peripartum Temperature Changes. Lammoglia et al. (1996) reviewed literature on temperature changes in the periparturient dam. A prepartum temperature drop in the dam just preceding parturition is common to essentially all mammalian species studied. We are following this physiological change in efforts to establish the mechanisms and relate them to dystocia and calf survival. Body temperature changes were monitored with electronic monitors placed under the muscles of the left flank. Temperatures were transmitted via radio telemetry every 3 minutes for 10 second periods for 144 hours before to 24 hours after parturition. Blood samples were collected every 8 hours throughout the study. Results are summarized in Table 2 and Figures 9 and 10.





Table 2. Effects of calf sex on variables studied

Variables	Sex of calf	
	Male n = 3	Female n = 4
Birth weight (lb)	90.8	93.7
BT <sup>a</sup> (° F)	104.36	103.64*
P4 (ng/mL)	2.71	3.15
E2 (pg/mL)	277.7	235.4**
PGFM (pg/mL)	146.6	110.8*
Cortisol (ng/mL)	6.5	5.6
T3 (ng/mL)	1.33	1.42**
T4 (ng/mL)	51.2	63.6*

<sup>a</sup>BT = Body temperature of the cow, P4 = progesterone, E2 = estradiol-17 $\beta$ , PGFM = prostaglandin F<sub>2a</sub>, T3 = triiodothyronine, T4 = thyroxine.

\*Different (P < .05) from male. \*\*Different (P < .01) from male.

The endocrine control of the prepartum temperature drop appears to be associated with PGFM and T<sub>3</sub> changes, but involvement of progesterone, estrogen, and cortisol cannot be ruled out. It is interesting to speculate what the physiological effects of this temperature change might be. Laburn et al. (1994) reviewed the literature and found that body temperature of the late-term fetus is about 1.1° F higher than that of the dam. The fetomaternal temperature gradient is established before the end of gestation and reflects the balance between rate of heat production by the fetus and fetal heat loss, which occurs mainly via the uteroplacental circulation. Decreased blood flow might be expected to compromise fetal heat loss resulting in a rise in the fetomaternal temperature gradient which could be potentially dangerous to the fetus.

Uterine blood flow declines during labor resulting in an increase in the fetomaternal temperature gradient. In addition, muscular activity during labor increases maternal temperature. All these factors could potentially result in increased fetal temperatures with possible damaging consequences, especially during prolonged parturition.

Is there also a possibility that this fetal temperature rise may also be a mechanism preparing the fetus for the temperature transition from uterine life to the outside world? Higher rectal temperatures in calves at birth have been reported and this heat dissipation could be part of the thermogenic adaption mechanism for the neonate. In addition, it is interesting to speculate on what effects hyperthermia occurring during prolonged parturition might have on calf vigor and survival.

## SUMMARY

Research on some factors affecting survival of the newborn calf have been reviewed.

Gestation diets containing adequate protein and energy have positive effects on calf heat production and rebreeding of the dam. Calves from dams that have received gestation diets low in crude protein show a high incidence of symptoms typical of Weak Calf Syndrome. Dystocia results in various degrees of anoxia in the calf which can result in death or weak, dummy calves. Some studies report gestation diets low in protein or energy result in lowered levels of colostral antibodies. Adequate fat in the gestation diet appears to be of importance in terms of calf thermogenesis and cold tolerance and rebreeding of the dam. Fat supplementation results may be dependent on amount of fat and also fatty acid composition. Cold tolerance in premature calves is poor. There is a drop in body temperature of the dam prior to calving which may be related to heat dissipation or transfer to the fetus during parturition.

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