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Reproductive Diseases of Dairy Cattle

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INTRODUCTION

The efficiency of today's dairy has increased due to the growing number of cows per herd and the production of pounds of milk per cow. Increased milk production however has resulted in a reduction of conception rates causing a loss of income for the dairy producer since the dairy industry relies heavily on milk production, which is caused by good conception rates. Many reproductive disorders like dystocia, metritis, endometritis, and retained placenta affect conception rates, and can lead to metabolic diseases like rumen acidosis, milk fever, and displaced abomasums. Recurrent metabolic diseases can eventually lead to culling of the herd. Many of these metabolic disorders can be the cause of poor nutrition and management during the close-up period and after parturition.

Reproductive and metabolic diseases can be treated and prevented if diagnosed early on such as retained placenta, ketosis, and displaced abomasums. It is crucial for the dairy producer to implement a disease treatment and prevention program with their veterinarian, nutritionist, and herd managers to control and prevent reproductive problems in their herd. It is also the dairy producer's managerial ability to be informed with the age of the cow, stage of her lactation, her reproductive efficiency, milk yield, and her history of past reproductive and metabolic diseases in order to sufficiently maintain a healthy herd.

To facilitate a reduction in reproductive and metabolic disorders the dairy producer should work closely with their nutritionist in order to supply an appropriate feeding program for the transition cow when she is close to calving and after she calves. The body condition of the cow is extremely important to monitor during her pregnancy, especially when she is close to calving. A cow in low body condition of less than 3 (on a scale of 1 through 5 with 1 being the lowest) before calving will give less milk throughout her lactation and will have greater reproductive problems. Cows that are in good health display estrus more frequently, have higher conception and pregnancy rates, have decreased cull rates, and produce more milk when compared with unhealthy herd mates. Therefore, it is important to implement a sufficient management program with a veterinarian, herd manager, and nutritionist in order to reduce metabolic and reproductive diseases on the farm, and sustain a healthy herd.

LITERATURE REVIEW

Increased Production of Dairy Cows

Effects of increased milk production in dairy cows have caused a decrease in reproductive performance and herd health. Milk production per cow has increased tremendously in the last 10 years due to improved nutrition, management, and genetics. However, the greater production per cow has caused reduced reproductive efficiency (14).

> The root cause of the declining fertility is a combination of a variety of psychological and management factors that have an additive effect on reproductive efficiency….A number of recent publications have documented a decline in reproductive efficiency. For example, Butler (1998) presented data showing a decline in first-conception rate from approximately 65% in 1951 to 40% in 1996 (14).

The dairy industry depends on reproductive performance of their dairy cows in order to meet the growing demand of milk products. The lactation cycle is dependent on the cow's ability to become pregnant since the hormones released during and after pregnancy are necessary for the development of the mammary gland and production of alveolar milk producing cells. The crucial hormones needed for lactation; such as prolactin and somatotropin shut down the hormones needed for reproduction and

growth of the follicle. A study was performed on 3 dairies in California to test the effect of peak milk yield on conception rates for 5928 lactation records. Results of the study showed "Cows with peak milk yields greater than the median (38.2 kg/d) were less likely to have conceived in one or two services than cows with peak milk yields 538.2 kg/d" (20). Another study done by L. Badinga of the University of Florida Dairy Science, suggests that cows that are high producers required more artificial insemination services in order to become pregnant, then their lower producing herd mates (1).

> In the American study, a negative association was again recorded between fertility (in this case defined as conception rate to first A.I.) and yield across a range of milk yields from 6,500-10,500 kg. An analysis of variance model using the figures reported by the authors indicated a decline in fertility of 0.34% per 100kg increase in yield (20).

Figure 1 demonstrates the negative relationship between high milk production and decreased conception rates of about 65% with first service AI in low producing cows, versus a conception rate of 40% in higher producing cows.

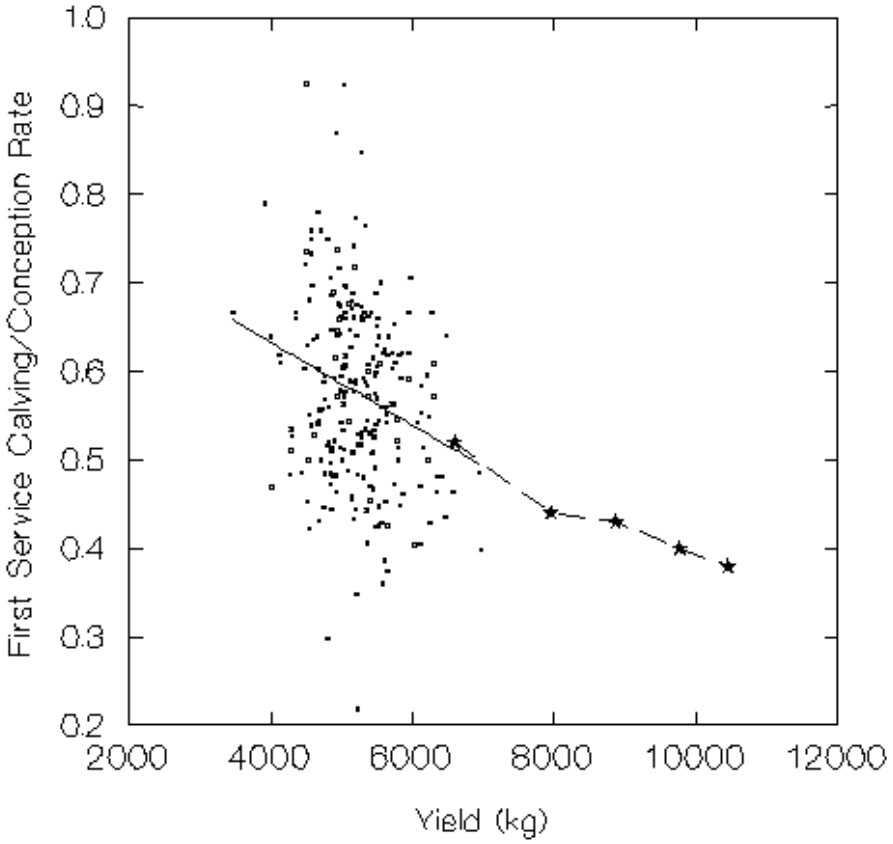

Figure 1. Relationships between milk yield and fertility in Irish and North American herds. DairyMIS data = scatter plot with fitted linear regression line, U.S. data = with dashed line (20).

The higher the milk production the poorer the reproductive efficiency, however reproductive diseases have a more profound negative effect on reproductive efficiency. Conversely, some studies have shown no impact of conception with high producing dairy cows. Therefore it is possible to increase reproductive efficiency by supplying a proper nutritional and management program, one that fulfills the nutrient and energy requirements of high producing lactating cows.

Reproductive Diseases of Dairy Cows

Uterine infections are present in dairy cows most common during the post partum period. Many specific infections have a severe impact on milk production and reproductive performance for example, dystocia, metritis, endometritis, and retained placenta. The vulva along with the vestibular sphincter and cervix protect the uterus from infections caused by bacteria. However, during and after birth or parturition the barriers that protect the uterus are broken allowing many pathogenic and nonpathogenic bacteria to enter into the reproductive tract of the cow. "The organism most commonly associated with uterine disease in cattle is *Actinomyces pyogenes*. In addition, the gram-negative anaerobes *Fusobacterium necrophorum* and *Bacteroides melaninogenicus* frequently associated with *A. pyogenes*" (26). Other microorganisms that cause disease of the uterus are "coliforms, *Pseudomonas aeruginosa*, *staphylococci*, and *Hemolytic streptococci*" (26).

According to Robert Youngquist, author of the book Large Animal Theriogenology, many infections of the uterus occur with: twins, over conditioned body type, under conditioned body type, large herd size, excess urea in dry cow diet, or retained fetal membranes. "Unsanitary calving conditions and traumatic obstetric procedures predispose cows to uterine infections" (26).

Dystocia is defined as difficulty giving birth and occurs when assistance is needed to deliver the calf during the second stage of labor. Dystocia is more common in first-calf heifers than older cows due to the fact that their body has not fully matured before calving, so the heifer must exert more work when giving birth. "Death due to dystocia or as a result of injuries sustained during delivery is the most common case of calf loss during the first 96 hours post partum, with most losses occurring during the first 24 hours after delivery" (26). The dairy producer cannot totally abolish dystocia from his or her herd, but they can help reduce its occurrence by implementing appropriate management of their heifers and cows during gestation and after parturition. According to the United States Department of Agriculture, "calves that survive a dystocia are more susceptible to disease and slower to grow, and dams that experience a dystocia might be culled earlier, produce less milk, and rebreed later than cows that calve unassisted" (13). Dystocia also causes an increase the incidence of metabolic diseases in dairy cattle. "Dystocia is associated with a twofold increase in the risk of milk fever, twofold to threefold increases in retained placenta and metritis, a threefold increase in cystic ovaries, and a greater than twofold increase in the risk for left-displaced abomasum" (24).

There are two stages involved in the birth process. The first stage of labor is when the cow or heifer experiences abdominal discomfort or is restlessness, and will isolate herself from the herd.

Rupture of the chorioallantois and release of the allantoic fluid ("breaking water") may be a more accurate attribute by which to mark the end of the first stage of labor.

The average duration of the first stage of labor is approximately 6 hours, but considerable variation among animals is observed, and the stage may last up to 24 hour in heifers (26).

During the second stage of labor myometrial contractions are stimulated by oxytocin, which forces the fetus into the cervical canal. The head and nose dilate the cervix using mechanical pressure, which stimulates oxytocin to be released, causing uterine contractions from the release of hormones by the pituitary gland. In the third stage of labor the placenta detaches and the "chorionic villi become dislodged from the crypts of the maternal side of the placenta…The time required for the expulsion of the placenta averages 8 hours but can range from a few minutes up to 12 hours without being considered abnormal" (26).

Dystocia can be caused by the dam or by the fetus. The three maternal causes of dystocia are: primary uterine inertia, secondary uterine inertia, and abnormalities of the birth canal. In primary uterine inertia the myometrium experiences failure to contract which is caused by overstretching of the uterus and periparturient hypocalcemia (low blood calcium before and after parturition). The dam is unable to proceed to the second stage of labor; she can only show small contractions in the abdomen. The cervix will be dilated but there will be no fetus in the birth canal. If labor has not been prolonged the calf can be delivered by small traction. (26).

Secondary uterine inertia is caused by collapse of the myometrium due to several failed attempts of delivery of the fetus. To treat, one must remove the obstruction and manually remove the fetus. Retained placenta, uterine prolapse, and slow uterine involution can also arise after uterine inertia. Abnormalities of the birth canal

include: pelvic deformities, deficient size of maternal pelvis or dilation of cervix, hernia of bladder, tumor on vulva or vagina, or uterine torsion (26).

Fetal causes of dystocia include: abnormal fetal presentation or position, fetal monsters, persomus elumbus, and fetal oversize. An abnormal fetal position is described as any position that is not in the "cranial longitudinal presentation and in dorsosacral position, with the head, neck, and forelimbs extended" (26). Fetal monsters are identified by certain fetal phenotypes such as schistosomia reflexus, i.e. extreme curvature of the spine positioning the head near the sacrum with the abdominal and thoracic walls open, leaving exposed intestines. Perosomus elumbus is also another example of a fetal monster. It is identified by flexure and ankylosis of hindlimbs, absent vertebrae in the caudal and thorax portions of spine, and flattened and deformed pelvis. The most common cause of dystocia in dairy cattle is fetopelvic imbalance. This occurs in heifers when the fetus is normal in size, however, the pelvis of the fetus is oversize, or the fetus can be abnormally large and cannot be delivered through the pelvic canal of the dam. (26).

In order to prevent and treat dystocia one must know history of previous occurrences of difficulty calving for each animal, such as any previous zoonotic diseases which caused an early abortion. Current gestation length and the length of time that the animal has been in labor are early predictors of dystocia. If heifer or cow exceeds an adequate amount of time for the first 2 stages of labor, then the dairyman should examine the animal. Since heifers are new to calving, they should be allowed a

longer time period to give birth. "National survey information shows that nearly 20% of all dairy cattle require some form of assistance during the calving process. Dystocia (difficult birth) rates increase to almost 35% for all first calf dairy heifers" (7).

If the amnion sack is ruptured, indicated by a release of allantoic fluid, and the cow or heifer has not given birth within two hours of the sack breaking, then assistance will be needed. (26).

Dystocia causes a huge loss in dairy cattle herds and cannot be predicted but can be reduced by superior management of one's herd. "Replacement heifers should be well developed and fed adequately to reach 65% of their mature weight at breeding… Parturient cows should be observed no less frequently than every 3 hours, and delivery should be assisted if the first or second stage of labor is prolonged" (26). Figure 2 demonstrates the time period that many producers wait to assist a cow or heifer experiencing calving difficulty. Many producers do not allow adequate time for the cow to give birth naturally, which is between 2-8 hours.

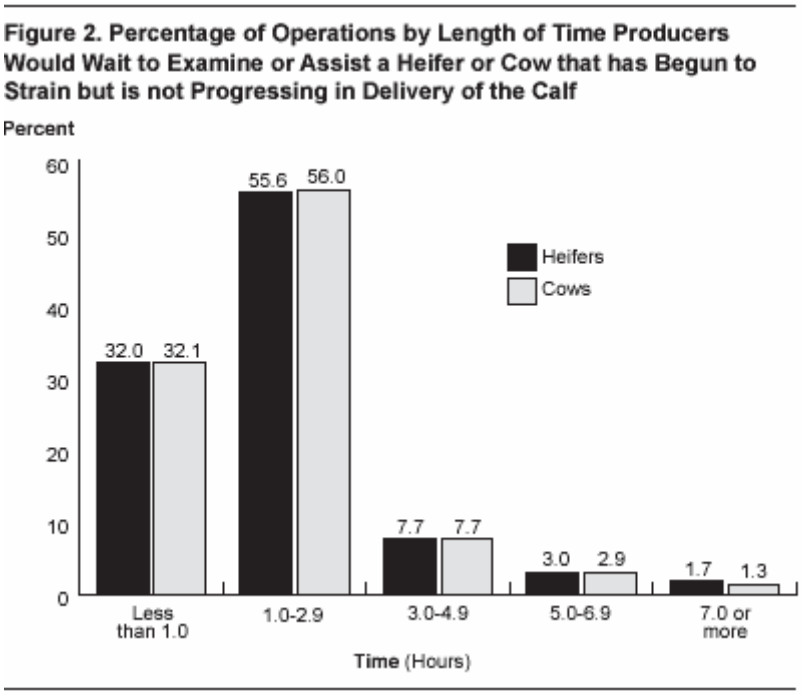

Figure 2. Time that producers wait to assist or examine heifer or cow that shows difficulty during the delivery process (5).

Metritis is defined as inflammation of the uterus which occurs during the first week after calving, and is related to dystocia and calving trauma. Symptoms include: fever, depression, decreased milk production, and anorexia (26). The organisms that cause infections in the uterus of the cow are *Actinomyces pyogenes* and *Fusobacterium necrophorum*, or different gram-negative anaerobic organisms (17).

> According to Veterinary Medicine Textbook Diseases of Dairy Cattle (22), *A. pyogenes* from the uterus during the postpartum period is significantly associated with endometritis, pyometritis and purulent discharge, resulting in an increase in time to first service, more services to conception, an increase in days open, and an increase in cows culled because of infertility.

A. pyogenes also causes abortion at any stage of pregnancy; however, its incidence of causing abortion is 0.28%.

Diagnosis varies from "obvious and persistent purulent exudate from the uterus and vagina, to flakes of exudates in otherwise clear estrous mucus. Changes in uterine consistency may occur, but transrectal palpation alone is an insensitive means of diagnosis" (17). Metritis can be classified as clinical or subclinical. Clinical metritis is a serious rapidly occurring condition that negatively affects the cow's appetite and production. It can be identified with rectal palpation by an enlargement in thickness and size of the uterine wall, and sometimes thick excretion from the vagina is observed (16). According to Pfizer Animal Health (18), clinical signs of metritis are fever of 103ºF or higher, decreased appetite, dehydration, depression, reduced milk production, and a distinct smelling vaginal discharge. Fever occurs in the cow 24-36 hours after calving, before other noticeable clinical signs appear. After calving there will be vaginal discharge for about 2 weeks which is normal indication that the uterus is draining left over fluid and returning back to its normal size.

Subclinical endometritis is not identifiable by rectal palpation and no vaginal discharge is evident. However, it can be diagnosed with a microscopic examination of a uterine biopsy (16). Examination of the cells that line the mucous membrane of the womb along with speculum examination will further assist the veterinarian in making a better diagnosis by knowing the specific location of the infection and the level of sensitivity.

Metritis is caused by an infection in the uterus by bacteria which occurs in 90% of cows that give birth; however, metritis does not always occur. Bacterial contamination that leads to metritis includes: the condition of uterus after calving, the amount and strain of bacteria that are present in the uterus, and the health of the cow to fight infection (18). Other incidences that can cause metritis are twining, improper calving assistance, difficulty calving, or milk fever. Also, inadequate nutrition can affect the rate of the uterus returning back to its normal size, which is necessary to rid the fluid in the uterus to not cause infection (18). Studies have shown that metritis causes cows to be 2 times more likely to get ketosis which leads to left displaced abomasums and cystic ovarian disease (21). Cows affected with metritis decrease feed intake which is harmful to the cow, since the postpartum period requires the highest intake due to the start of lactation.

To treat metritis, one can inject antibiotics into the uterus, however, care must be taken and consultation with a veterinarian is necessary to make sure there are no negative side effects of the drug. Many organisms that cause uterine infections are sensitive to penicillin which is commonly used in countries other than the United States. In the United States, intrauterine administration of antibiotics is not allowed to lactating dairy cows, since it causes contamination of the milk (3). Ceftiofur is another beneficial antibiotic that acts against gram-positive and gram-negative bacteria, which are the cause of most cases of metritis. Ceftiofur can be administer subcutaneously and has the ability to treat all layers of the uterus without residues in

the milk. Ceftiofur is approved in the United States for only systemic administration to affected lactating dairy cows (26).

Some veterinarians prescribe the use of iodine solutions for intrauterine therapy. There, however, has been no evidence that suggests it is beneficial. According to Pfizer Animal Health (18), a new antibiotic, Excenel RTU, was recently approved by the Food and Drug Administration. Excenel RTU is already being used in the dairy industry to treat foot rot and pneumonia. The drug can be administered subcutaneously or intramuscular, without withholding milk production. Excenel concentrates at the site of the infection by binding to plasma, infected tissues, and extracellular fluid where pathogens are present. In figure 3 below, research shows that the active metabolites in Excenel RTU work to reduce the concentration of plasma and fluids associated with metritis (8).

Another attempt to treat or prevent postpartum uterine infections is hormone therapy. However, according to Large Animal Theriogenology, the use of estrogen to cause myometrial contractions is not recommended since it has been known to cause septic contents of the uterus to overflow into the cervix and uterine tubes causing severe bilateral salpingitis (26). Oxytocin combined with estrogen is an effective treatment to cause the myometrium to contract and release fluid, when given 48-72 hours after calving.

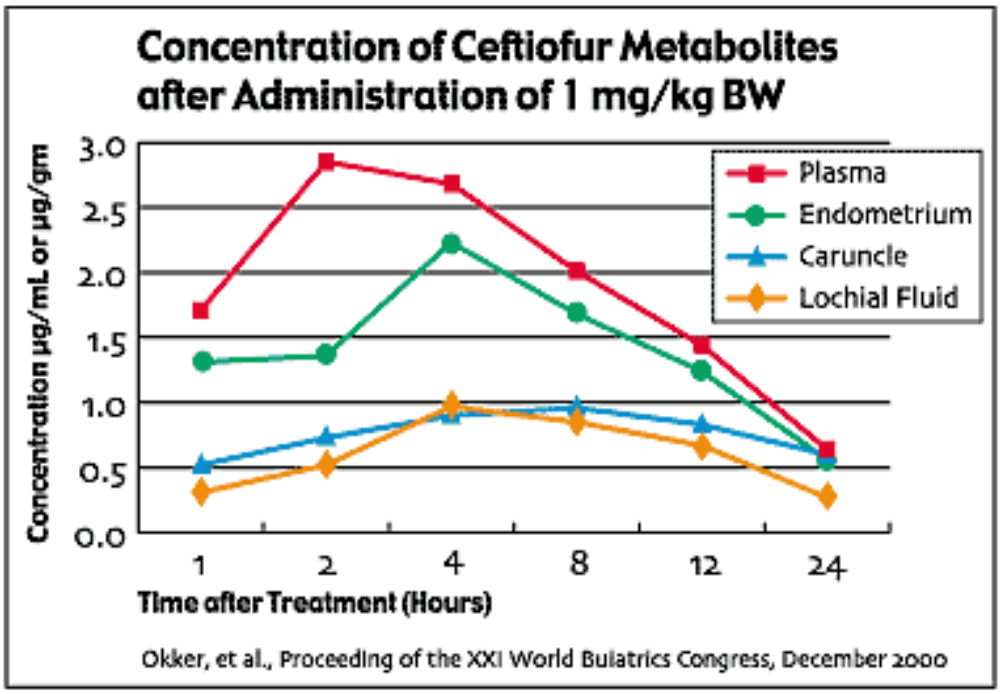

Figure 3. A graph that demonstrates the effects of active metabolites of Excenel RTU concentrating at targeted tissues and fluids of metritis.

In summary, management of sanitation, population density, stress, and nutrition are key factors in the prevention of metritis in dairy cattle. Excessive use of antibiotics after calving has been shown to decrease fertility. However, the use of ceftiofur in dairy cows with dystocia or retained fetal membranes has been shown to reduce the risk of metritis by seventy percent, when compared with cows that were not treated with antibiotics (26).

Endometritis in dairy cattle is defined as inflammation of the endometrium. Endometritis occurs after giving birth, artificial insemination, or infusion of irritants into the endometrium. Symptoms are "purulent exudate" of the vulva and vaginal discharge. Endometritis causes lowered first-service conception rate, and requires overall more services per conception (26).

Subclinical endometritis cannot be detected by rectal palpation and no vaginal secretion is observed; however, it can be detected by microscopic examination of uterine tissue. Metritis and endometritis are common after pregnancy up to 47 days postpartum, when the uterus returns to "its normal nonpregnant size…called involution" (16). To prevent infection that could lead to metritis or endometritis, it is crucial to have proper management and sanitary conditions during this high risk involution period.

Endometritis can be caused by bacteria or fungi that enter the uterus during calving or after calving. Lesions in the vagina can be present during this time which makes the reproductive tract very susceptible to infection, since the animal's natural defense system is compromised (16). Assistance with calving during pregnancy period can cause bacteria or unwanted organisms to enter the uterus. This is why it is important to have clean calving area, which includes clean fresh bedding, free of manure.

Metritis and endometritis can be diagnosed post calving by postpartum examination during early lactation, and by routine follow-up exams of the fresh cow. The veterinarian can diagnose the severity of the condition and suggest further treatment if needed. Treatment involves the knowledge of the health of the animal, nutrition,

temperature, condition of reproductive tract, and systemic involvement (16). Studies have shown that when an animal is in estrus, she is less likely to experience a uterine infection. Prostaglandin hormone is given to cows after parturition to mimic estrus by causing stimulation of uterine contractions and the release of uterine pus. (17). Endometritis should be treated within thirty days after giving birth with the use of prostaglandins 2 times a day for 14 days. This causes the muscles of the uterus to contract, which removes discharge from the endometrium and causes corpus luteum to degenerate. According to Opsomer from the Department of Reproduction, Obstetrics and Herd Health, Faculty of Veterinary Medicine,Ghent University, Belgium (21), "Puerperal metritis should be treated with broadspectrum antibiotics both parenterally and intra-uterine during 1-3 days depending on the severity of the symptoms".

The most important concept in treating endometritis is early diagnosis and close monitoring for the effectiveness of treatment. It is the dairy producer's responsibility to have in place an adequate management program to prevent metritis or endometritis infections in their cows.

> According to Dr. Manspeaker of the University of Maryland (16), if uterine infections are excessive (greater than 20%) in a herd, management of dry and recently calved cows should be thoroughly examined. Adequate housing, nutritionally balanced and palatable rations for the dry and fresh cows, satisfactory calving facilities with optimal ventilation and sanitary conditions, and avoidance of undue stress such as overcrowding and disease are absolute requirements for healthy fresh cows.

Retained placenta in dairy cattle is divided into either primary or secondary retention. By definition, "the primary retention of fetal membranes results from a lack

of detachment from the maternal caruncles, whereas secondary retention is related to a mechanical difficulty in expelling already detached fetal membranes" (26). The placenta is the membrane that transfers nutrients from the dam to the calf during pregnancy. The membrane is composed of blood vessels that supply the calf with blood and where essential nutrients are passed from the mother to the offspring during fetal development. The essential nutrients are oxygen, amino acids, fats, blood sugar, calcium, phosphorus, trace minerals, and vitamins, which are all necessary components of life (15).

All cows that calve have some extent of retained fetal membranes (RFM). However, more then three fourths of cows push out their fetal membranes by 6 hours, and some even after 12 hours after giving birth. The retention of fetal membranes after twelve hours has profound effects on reproductive performance, postpartum disease, milk production, and rate of culling.

Disconnection of fetal membranes normally occurs postpartum and is facilitated by a breakdown of collagen and other proteins. Conversely, retained placenta is caused by a "lack of cotyledon proteolysis is the cause of retained placental membranes" (26). The detachment of the placenta in the cow involves the separation of the cotyledone villa from the caruncle crypts without tearing to the maternal or fetal epithelia. After the placenta has detached, it take the uterus an average of 39 days in normal cows to return back to its normal size. However, in cows with retained placental membranes it takes 50 days for the uterus to return to its normal size. Serotonin, present in the fetal blood, also plays a role in placental separation. It causes the release of

collagenase by uterine cells and causes collagen breakdown to occur in the uterus after giving birth (26). Vitamin deficiencies, such as vitamin A and vitamin E, and mineral deficiencies in Selenium or toxins in feed, such as nitrate can contribute to the retention of fetal membranes (11).

The start of parturition causes the uterus to contract, followed by decrease blood flow to the maternal and fetal segments of the placenta. Attachment of placental membranes will remain in place if a pressure exists on the attachment between the dam and fetus placenta, such as excess fluid or trauma. Metabolic diseases can cause retention of fetal membranes, such as milk fever. Milk fever is characterized by low blood calcium and occurs during or immediately after parturition. It occurs from an imbalance between the input and output of calcium, which is required for muscle function in the dairy cow. The lack of muscle contraction of the smooth muscles in the uterus can cause retained placenta even if the fetal membranes are disconnected from the maternal membranes. Hypocalcaemia increases the risk of milk fever by 5.6 times as compared to healthy cattle.

Commonly, retained placenta is associated with abortion, dystocia, and multiple births. In single calving, the occurrence of retained fetal membranes was 9.5% as compared with twining which increased the rate by 46%. Retained placenta increases metritis in cattle by 55% and is 25 times more likely to occur in cattle with retained placenta vs. healthy cattle (22). The use of glucocorticoid hormones to induce parturition causes 67% retained fetal membranes. Cows that have a history of

retained placenta are more than likely to have retained fetal membranes in future calvings. Retained placenta is higher in herds that have nutritional and management problems, metabolic disease or acute mastitis at calving. Brucellosis, leptospirosis, vibriosis, lsteriosis, and infectious bovine rhinotracheitis can increase the risk of retained placenta by 50% (16).

Retained fetal membrane is a common disease in dairy cattle and there are often no signs of systemic illness. However, when systemic signs are present they are usually due to toxemia and should be treated with the use of non-steroidal anti-inflammatory drugs or antimicrobials (17). Removal of the retained membranes includes abundance of rank smelling dark-brown, red fluid that is combined with small pieces of placenta in the vagina. Physical investigation of the uterus by one's hand will indicate the severity of the fetal membrane involvement. Many clinical examinations of the rectum indicate an overlarge uterus that is loose and lacks ridges. The presence of thick sticky non-foul smelling mucus in the cervix and vagina usually illustrates that the fetal membranes have been released from the uterus. The dead pieces of placenta may take up to 10-14 days to be expelled after treatment has been started (22).

Treatment of retained fetal membranes includes manual removal, use of hormones or antibiotics, collagenase injection, and treatment with hyperosmotic solutions. Manual removal of the retained fetal membranes is risky since it increases the risk of uterine infections versus leaving the animal untreated. If an animal is left untreated it will usually take 2-11 days for the membranes to be released (17).

> According to Dr. Youngquist (26) of the University of Missouri Manual removal has been found to prolong the interval from calving to first functional corpus luteum by 20 days…It is not easy to properly remove a retained placenta; 62% can be removed completely, 27% partially, and 11% are nonremovable…attempts at removal during the first 48 hours after calving are unsuccessful because the placenta is too firmly attached and the apical part of the gravid horn is beyond the reach of the veterinarian.

The uses of hormones have not been shown to prevent metritis in cows with retained fetal membranes and it is not successful in detaching the placenta. When dexamethasone is used with relaxin, the 2 have been shown to prevent retained fetal membranes; however, clinical use for treatment has not been studied. The use of antibiotic injections is controversial because it is hard to obtain the right dosage concentrations of antibiotics due to different tissue absorption. Intrauterine tetracycline has been shown to produce a negative impact on fertility and irritation of the endometrium (26). Inflammation from chemicals can be more harmful on the animal versus non-treatment. More research is needed in the use of antibiotics to treat and prevent retained fetal membranes.

Injection of collagenase into the umbilical arteries of the retained fetal membranes is beneficial since it targets the lack of cotyledon proteolysis. Collagenase is superior with its ability to breakdown different types of collagen tissue, affordability, commercial availability, and causes no blood clotting in the placenta. "Treatment is effective in 85% of affected cows within 36 hours. In the 15% of cows that fail to respond, however, repeat treatment is not recommended because it rarely is effective" (26). This technique must be performed by a veterinarian. Collagenase however is not approved for use in food animals in the United States. Treatment with

hyperosmotic solutions is used to cause a separation of cotyledons from the caruncles by increasing the volume of the cotyledon. This approach however, has not been proven to work during laboratory tests.

Prevention is crucial to avoid retained fetal membranes, by managing your close-up ration 3 weeks before calving. The dairy producer should supply his close-up herd with a negative dietary cation anion difference (DCAD) diet, which will help prevent retained placenta and also increase milk production. To monitor if your herd is receiving an appropriate dietary cation anion difference (DCAD) diet, the dairy producer should check the urine pH on his close-up pen on a weekly basis. A urine pH of 6.0-6.5 indicates the negative DCAD ration is working properly. Adding anionic salts to the close-up ration can prevent milk fever by helping the cow to metabolize calcium (4). However, the dairyman should closely monitor the cows to make sure that dry matter intake is not decreasing due to the addition of anionic salts. Ultimately it is the producer's responsibility to work closely with their veterinarian and herdsman to prevent and treat metabolic and reproductive diseases such as milk fever and retained fetal membranes.

To reduce incidence of retained fetal membranes the dairy producer should breed their heifers to bulls that have a record of good calving ease. The producer should also closely monitor his close-up pen to make sure that the conditions are sanitary, stress free, and to make sure that there are no complications during the calving

process. Assistance should be provided to cattle if labor continues over 30 minutes with no further progress (10).

The economic impact of retained placenta is due to a decrease in milk production, an increase in veterinary services, an increase in calving interval, and an increase in cull rate. A study performed on Holsteins and Ayrshires in the Northeast United States, demonstrated a decrease in milk production by 1.4kg/day due to retained placenta. It was also reported that retained placenta decreased conception rate by 14% when compared with healthy cows (26). The cost of veterinarian treatment is about $244 per affected animal, costing $154 million per year. Table 1 demonstrates the average cost per cow with retained placenta.

TABLE 1. Cost per case of retained placenta (4).

Table 1 Cost per case of milk fever and retained placenta

Milk Fever		Per 100 cows	Per 1,000 Cows
Cost/case	$186		
Previous Incidence Rate	7%		
Total Cost/Year		$1,302.00	$13,020
Cost per cow		$13.02	$13.02
New Incidence Rate	2%		
Total Cost/Year		$372	$3,720
Cost per cow		$3.72	$3.72
Retained Placenta		**Per 100 cows**	**Per 1,000 Cows**
Cost/case	$217		
Previous Incidence Rate	15%		
Total Cost/Year		$3,255	$32,550
Cost per cow		$32.55	$32.55
New Incidence Rate	8%		
Total Cost/Year		$1,736	$17,360
Cost per cow		$17.36	$17.36

Metabolic Diseases of Dairy Cattle Relating to Reproduction

Factors that cause reproductive diseases in cattle have a correlation of causing certain metabolic diseases; such as rumen acidosis, milk fever, and displaced abomasums. Factors associated with reproductive diseases include: decrease in dry matter intake, increase in non-esterified fatty acids (NEFA's), increase in the stress hormone coritsol, and decrease in neutrophil function. Body condition of dairy cattle is based on a scale of 1 to 5, with 1 indicating severely emaciated and 5 indicating obese. Optimal body condition of a dairy cow at time of calving is 3.5 to 3.75.

Decrease in feed intake is associated with an excessive body condition, increased stress from grouping and sorting, increased cortisol levels, and hypocalcaemia. Cows that are fed to much during late lactation enter the transition period overconditioned and are harder to manage due to decrease in feed intake. The higher body conditioned the cow is at calving the greater the occurrence of metabolic disease. Cows that are overconditioned are also at an increase risk of dystocia and retained placenta due to the fat surrounding the reproductive tract, which creates with contractions during calving (6). Figure 4 illustrates the reproductive and metabolic disorders that are caused by overconditioned cows during parturition.

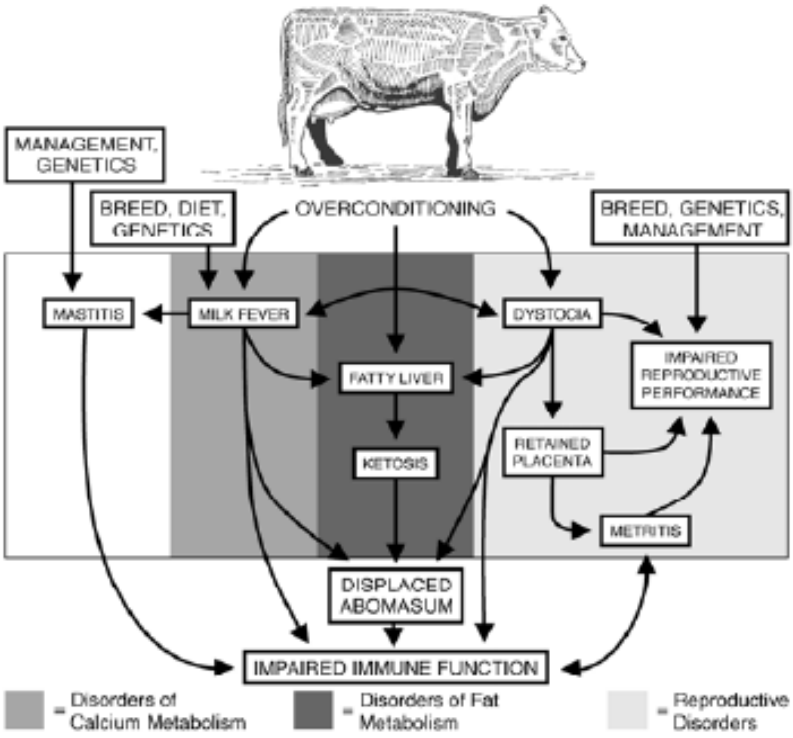

Figure 4. Metabolic and reproductive disorders that occur from overconditioned cows (Courtesy of Dana Boeck, 24).

Cows should be placed in the close-up pen for a minimum of 3 weeks pre-partum. To reduce cortisol levels in cows associated with stress, cows should be placed in individual maternity stalls at least 1 week before calving. The maternity pens should also permit the cow to eat and drink without competition, allowing the producer to monitor the cows closely during this critical time period. Producers should also clean the maternity pens after every calving to reduce health problems to both the calf and the dam. Bacteria and pathogens from manure in the bedding can cause infection in the uterus and eventually lead to metritis, due to the fact that the cow's immune

system is suppressed at calving. "Recent surveys suggest that less than half of all producers clean maternity pens after every calving and less than 10 percent of producers disinfect stalls in addition to stripping bedding" (24).

In larger dairies the transition cows can be moved up to 5 or 6 times. Each move stresses the cow due to repositioning of social dominance, which results in a decrease in dry matter intake as the cow is getting close to calving. The dairy producer should minimize movement of cows during the transition period to increase dry matter intake during this crucial time.

There is a "direct correlation between decreased dry matter intake prior to calving and impaired PMN (neutrophil) function both before and after calving (6). The impaired PMN (polymorphonuclear neutrophil) resulted in an increase in uterine health disorders due to an impaired immune response. "Every 10-minute decrease in average daily feeding time during the week before calving, the animal was 1.72 times more likely to develop a severe case of metritis after calving. For every 1 kg decrease in dry matter intake, the animals were 3 times more likely to develop severe metritis" (6).

When cattle are in negative energy balance, they have to mobilize body fat which leads to fat deposits in the liver, referred to as fatty liver disease. The fat deposits in the liver are converted into glucose by the liver resulting in the production of non-esterified fatty acids (NEFA's). NEFA's are immunosuppressive because they restrict the ability of myeloperoxidase, an enzyme that is necessary for neutrophils to

destroy bacteria. Figure 5 illustrates that as plasma NEFA levels increase, the neutrophils ability to make myeloperoxidase is reduced (6).

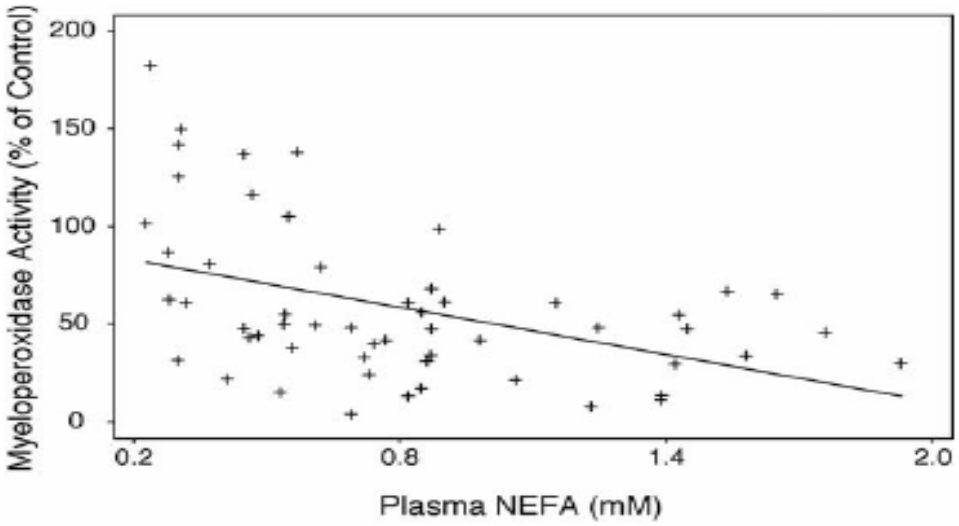

Figure 5. Correlation between plasma NEFA levels and Myeloperoxidase (6).

High NEFA plasma levels indicate decreased dry matter intake of the cows in the close-up pen, which signals the dairy producer to implement a better management program for his transition cows.

Cows in the close-up pen undergo many stressors such as temperature fluctuations, overcrowding, moving from pens, changes in ration, decreased dry matter intake, or metabolic disease which causes cortisol hormone to be released from the adrenal

gland. Cortisol suppresses the immune system making the animal more susceptible to metabolic and reproductive diseases that can overall impact production. This is why it is crucial for the dairy producer to closely monitor the close-up pen and implement a management plan that is stress free to the cow.

During the close-up period the animal has difficulty maintaining constant blood calcium due to the requirements from the fetus and colostrum production.

> A high percentage of metabolic disease problems as well as impaired immune function are directly correlated to low calcium levels…since muscle contraction is impaired, this often results in failure of the uterus to involute normally following parturition, a slowing down of the entire gastro-intestinal tract, and an increase in the incidence of displaced abomasums (6).

Animals with hypocalcaemia have cortisol levels that are three times higher than animals that are healthy at calving. The higher cortisol levels cause the animal to be more vulnerable to infectious diseases at time of calving due to the immunosuppressive effect of cortisol. Hypocalcaemia also puts the animal's immune system at risk, since calcium is involved in the intracellular signaling factors of white blood cells, reducing the animal's ability to fight pathogens in the body (6).

Rumen acidosis in dairy cattle is due to consumption of large amounts of fermented carbohydrates which causes the production of volatile fatty acids (VFA's). The production of vast amounts of VFA's and lactic acid will drop the rumen pH and cause clinical forms of ruminal acidosis that can lead to peracute, acute, or subacute (SARA) diseases. Peracute and acute rumen acidosis are caused by ingestion of too much grain and can cause systemic dehydration which is deadly to the cow. Acidosis

is most commonly caused by operational errors in feed management, and can be prevented by better management.

To maximize milk production the dairy producer must supply the cow with fibrous feed in order to achieve microbial fermentation. Conversely, fibrous feeds do not contain high enough levels of energy to support the growing needs of the cow during lactation. Therefore, grain or fermented feed must be supplemented into the diet. Close-up cows (3 weeks before parturition) and fresh cows (3 weeks after parturition) are referred to as transition cows, since their body is getting ready to give birth and produce milk. During the transition process the cow experiences many changes in diet due to a reduction of feed intake and rapid introduction to large amounts of fermented carbohydrates. The microbial population in the rumen must gradually be introduced to change in the diet to prevent against SARA.

SARA can be diagnosed by performing a rumenocentesis on the animal after she has consumed feed. Table 2 illustrates the when to check ruminal pH after feeding. This procedure is performed by inserting a needle and syringe and puncturing the flank of the cow to access the rumen. The fluid removed is then measured with a pH meter to determine the acidity of the rumen. If the pH is less than 5.5 then the cow is considered to be high risk for subacute ruminal acidosis.

TABLE 2. Recommended Timing of Ruminal pH Measurements (19).

Feeding Program	Rumen Sampling Time to Measure pH
TMR fed once daily	5-8hrs. after feeding
Forage and concentrate fed separately	2-5hrs. after concentrate feeding

The dairy producer should also evaluate the length of fiber to make sure that it is not too small, which can disable the cow from chewing her cud. The cud chewing process creates saliva which acts as a buffer, preventing low rumen pH. Equally as important as effective fiber length is sorting of feed, which tells the farmer what the cow is consuming and if the feeder is doing a good job with the chopping and mixing of the total mixed ration diet (TMR).

Clinical signs of subacute ruminal acidosis (SARA) usual appear 12-36 hours after engorgement of grain. The cow can appear uncoordinated, weak, depressed, increased respiratory rate, or have abdominal pain which is shown by grunting (11). Other observable signs of SARA include: lameness, decreased feed intake ("slug" feeding), decreased body condition, a decrease in milk fat and milk production, and loose feces (26). Table 3 demonstrates the common symptoms to look for when identifying if ones herd has subacute ruminal acidosis. According to Dr. Kung of the University of Delaware (12), "a low milk fat test (less than 3.3 to 3.0%) is one of the

best measures of acidosis. Fat tests less than 2.7 to 2.8% will more than likely be accompanied by cows with laminitis".

Treatment options for SARA include tubing the animal and administering antacids such as magnesium carbonate or magnesium hydroxide followed 2-3 gallons of warm water. Activated charcoal or IV electrolyte and bicarbonate can also aide in the recovery of the cow during mild to severe cases of SARA (11).

TABLE 3. Common symptoms of acidosis (12).

- Low milk fat test; < 3.0 to 3.3%
- Sore hooves; laminitis
- Cycling feed intake
- Diarrhea
- Liver abscesses
- Low rumen pH (< 5.8) in 30 to 50% of animals tested
- Limited cud chewing

Prevention of SARA involves appropriate management of feeding practices in dairy cattle, especially in transitioning cows. Caution should be taken in feeding and processing high levels of carbohydrates that produce lactic acid such as sugars and

starches. Production of VFA's and lactic acid production are controlled by particle size and degradation by microbes in the rumen. The smaller the size of starch leads to an increase in ruminal degradation and increase risk for acidosis.

> According to the Tri-State Dairy Contest in Alberta Canada in 2009, feeding a close up diet that is intermediate in fermentability between the far-away dry cow diet and the lactation diet stimulates the growth of the rumen epithelium, which increases the surface area for VFA absorption. It is thought that increasing the absorptive surface of the rumen helps prevent the accumulation of VFA in the rumen, the main driver of ruminal pH depression (2).

The transition diet should contain adequate fiber length for proper rumination and buffer production. Fiber length should be greater than 0.75 inches and comprise 20% of the diet. The dairy producer can also supply ionophore antibiotics which include Monensin, Lasalocid, and sodium bicarbonate buffers, to improve the efficiency of feed (11).

Hypocalcaemia (milk fever) in dairy cattle is a disease that occurs when calcium is lacking in the body, which "causes a progressive loss of skeletal, cardiac, and smooth muscle function" (26). It is the most common metabolic disease of dairy cattle. The disease commonly occurs 48 hours after parturition in high producing dairy cattle. Parturient hypocalcemia is characterized by low blood calcium in the dam due to the extensive loss of calcium for the production of colostrum. The calcium lost from the production of colostrum is replaced by intestinal or bone resorption of calcium, causing a reduction of calcium in the body of the cow. Figure 4 illustrates the demand of calcium in a high producing dairy cow.

Clinical symptoms of hypocalcemia include: sporadic muscle spasms, lack of appetite, uncontrollable defecation and urination, lateral recumbency, coma, and death if untreated. The incident rate in most herds is 8-9%, but it can vary in different herds from 0% to greater than 60% occurrence. Milk fever can reduce the productivity of a dairy cow by 3.4years and cost $334 per clinical case. According to the Journal of Dairy Science, "the incidences of dystocia, retained placenta, displacement of the abomasums (downer cow syndrome), and uterine prolapse are greatly increased as a result of milk fever" (9). Older cows are also more susceptible to milk fever in their third or later lactation. As cows mature they secrete more milk compared to the previous lactation until they are fully matured. The increase in milk causes a higher demand for calcium. Aging results in a reduction of the cow's ability to store Ca in bones and intestinal tract due to a decrease in active transport of Ca in the body.

Hypocalcemia can be treated with intravenous calcium solutions which contain 8-10g of Ca. This allows the cow to adjust to the deficiency of calcium in the intestinal tract and bones, until the level of calcium is sustained at its normal level. Without intravenous calcium treatment the possibility of death is 60-70% (9).

Prevention of milk fever involves proper management of transition diet. Studies have shown that feeding low calcium diets, less than 100g of Ca per day, can reduce the incidence of milk fever (9). When the calcium level of the cow is dropped she is put into a state of negative calcium balance, causing the cow to gradually produce Ca, preparing her for the large demand of calcium at calving. High potassium forages are

known to increase incidence of milk fever and should be decreased during the close-up period. Forages such as alfalfa contain high levels of calcium but also high levels of potassium and should not be feed before calving. The use of calcium gels $CaCl_2$ can also be administered to cow's right before they calve in order to increase the concentration of calcium in the blood.

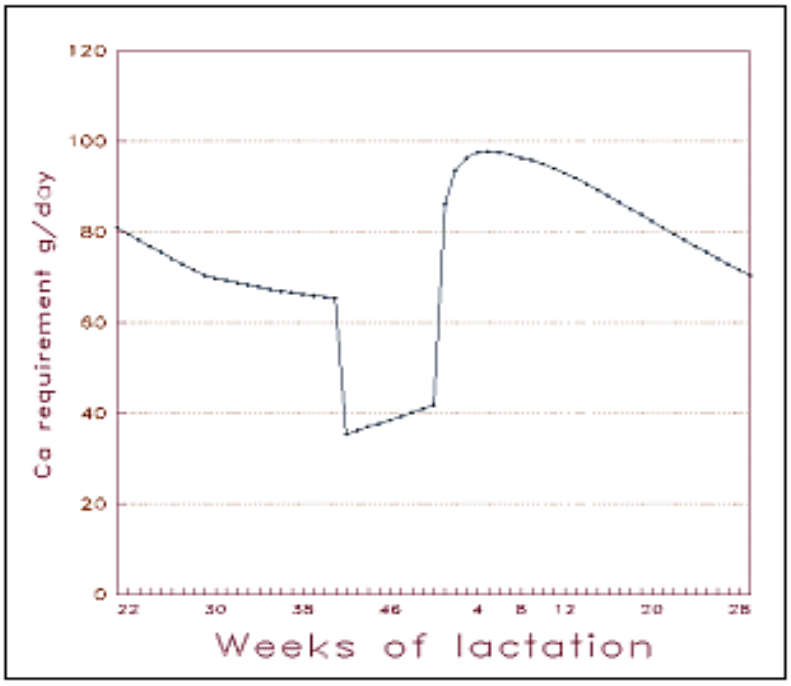

Figure 6. Calcium requirements of a high-yielding cow (23).

Displaced abomasums are associated with a twisting of the abomasal compartment. The "twisting associated with this repositioning effectively slows or stops the flow of digesta through the gastrointestinal tract. The gas buildup leads to a characteristic

bloat appearance. Pinging is detected with a stethoscope by thumping the cow near the last rib and listening on the left flank" (24). The abomasum is held loosely be the omenta, allowing it to move from its normal position on the right ventral side of the abdomen to the left side, or rotate on the right side on its mesenteric axis (17). Right-side displaced abomasums are more harmful to the cow because they are harder to treat and cause a more complete tear of the abomasums and are more likely to require surgery for treatment. Figure 5 shows the normal position of the abomasum, while figure 6 shows a left sided displaced abomasum.

The occurrence of displaced abomasums can be 15% in problem herds. Greater than two-thirds of cases are caused by different metabolic diseases, such as hypocalcaemia. Hypocalcaemia decreases gut function, allowing gas production to occur in the abomasum. According to the Merck Veterinary Manual (17), "about 80% of displacements occur within 1 mo of parturition; however, they can occur any time. LDA is much more common than RDA (8:1)."

Feeding low-roughage and high concentrate diets such as high grain or heavy corn silage before parturition can also increase the risk of displaced abomasums. High grain intake increases the production of volatile fatty acids (VFA's) through the abomasum, causing a loss in contractions in the abomasum. In order for the papillae to absorb the VFAs produced in the rumen, they must be fully developed by gradual introduction of grain of concentrate feeding during early lactation. Feeding high concentrations of grain to transition cows without allowing the papillae to develop

can increase the loss of VFAs from the rumen (24). Cows in high body condition scores before calving are more likely to have displacement than conditioned cows.

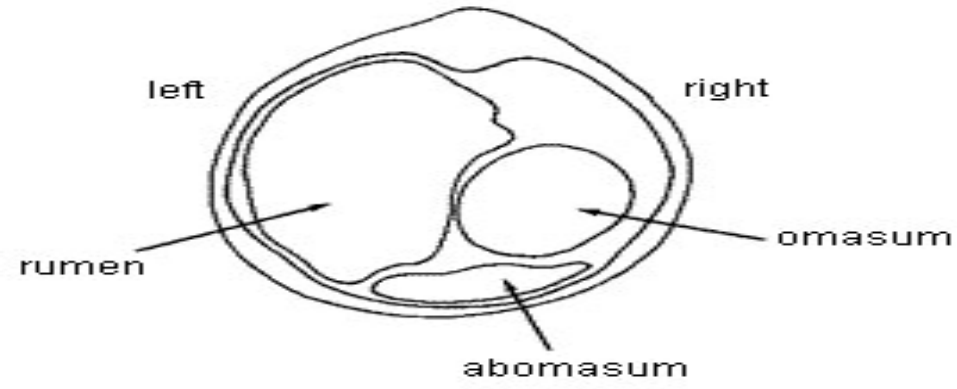

Figure 7. Normal position of abomasum on right side (25).

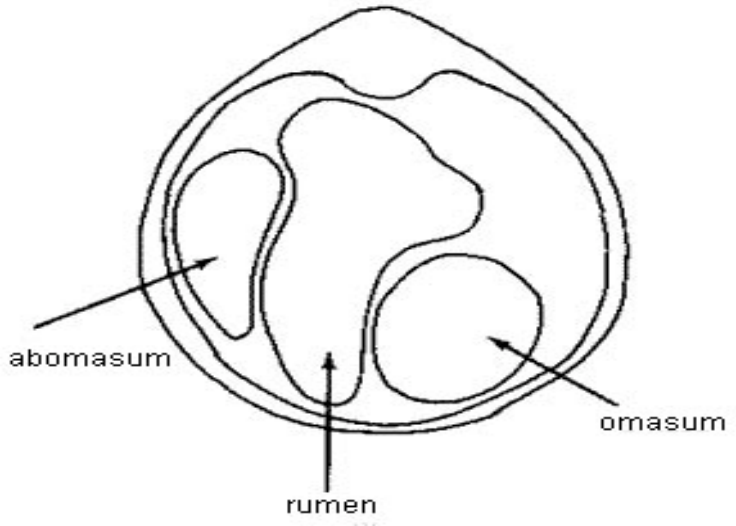

Figure 8. Abomasum displaced to the left of rumen (25).

Clinical findings include a decrease in milk production and a decrease in feed intake, due to the inability of feed to leave the rumen. Cows can also look depressed or in pain and stand with their back arched. Other clinical signs include "sprung" rib cage on the side of the displacement and increased watery feces.

Diagnosis of displaced abomasums occurs 14 days postpartum, but one-fourth of cases arise after 14 days (24). Rectal palpation can be performed to confirm the presence of gas in the rumen, or indicate and empty rumen. Ketosis can develop and ketone bodies can be found in the milk or urine. The ping sound heard with right displaced abomasums is found between the 9^{th} and 11^{th} ribs. Rectal examination performed on a cow with a left displaced abomasum will indicate a medially displaced rumen and left kidney (17).

Treatment of displaced abomasum includes removal of gas from the abomasum. Procedures to remove gas include: rolling the cow over on her right side to treat a left displaced abomasum, however recurrence is common. A surgical procedure can be performed called toggle-pin fixation or blind stitch. Both of these procedures are performed in the right paramedian area and are percutaneous techniques to treat left displaced abomasum (17). Right displaced abomasum can only be corrected with surgery. Prognosis after treatment of left displaced abomasum and right displaced abomasum is 75-95%.

Prevention of displaced abomasums can be achieved by gradually transitioning close-up cows to a higher grain diet, allowing the papillae to adapt and grow. The dairy producer should provide a complete ration with proper roughage and supply of

anionic salts to avoid hypocalcemia. The cow should also be in adequate body condition at calving, body condition of 3.25-3.75 (24).

MATERIALS AND METHODS

The literature review looked at the effects of reproductive and metabolic diseases on the transitioning cow and how these could be reduced with better management. Data was analyzed to determine the cause, the cost, the symptoms, and the prevention of dystocia, metritis, endometritis, retained placenta, acidosis, milk fever, and displaced abomasums on close-up cattle. All research came from professional journals, dairy publications, and dairy books. The materials presented in this paper will assist in determining whether or not better management of one's herd will decrease the occurrence of reproductive and metabolic diseases, while increasing reproductive efficiency and milk production.

CONCLUSION

Dairy cattle have become very efficient in their production of pounds of milk per lactation. However, the increase in production has lead to a rise in metabolic and reproductive problems with the animal due to increased nutritional demands and stress levels. Reproductive and metabolic diseases affect every herd, especially when the animal's immune system is compromised during the transitional period. Post partum diseases like dystocia, metritis, and endometrits can arise from certain metabolic diseases, such as hypocalcaemia, displaced abomasums, and retained placenta. Reproductive diseases are caused by a decrease in dry matter intake which leads to an increase in NEFA levels and a rise in coritsol levels, all of which reduce the ability of the animal's white blood cells to fight pathogens.

Cows in the close-up pen undergo many stressors such as temperature fluxuations, overcrowding, moving from pens, changes in ration, decreased dry matter intake, or metabolic disease which causes cortisol to be released from the adrenal gland. Cortisol suppresses the immune system making the animal more susceptible to metabolic and reproductive diseases that can overall impact production. Cortisol levels are also increased by low blood calcium during the close-up period.

Reduced dry matter intake can be associated with several factors like over-conditioning of animals during the transition period, stress, and hypocalcaemia (milk fever). Low blood calcium associated with hypocalcaemia weakens the animal's immune system by

affecting the receptors on the white blood cells which protect the cow's body from foreign pathogens.

The effect of hypocalcaemia impairs muscle contraction which causing a retention of fetal membranes due to the inability of the uterus to involute normally following parturition. This then can lead to metritis or endometirits, or a slowing of gastro-intestinal tract causing an increase in displaced abomasums.

The transition period is the most crucial time period for the health of the dairy cow and expected production during the 305 day lactation cycle. Management of the transition period should focus on body condition of the animals, dry matter intake, sanitation, movement of pens, overcrowding, handling, and health of the animal. The animal should be in optimal body condition of about 3.5 to 3.75 and should be given an adequate DCAD ration that is supplied by the dairy producer's nutritionist. Management of the feed bunk should be in place to make sure the feed is fresh and accessible to the animal in order to prevent a reduction in dry matter intake. The dairy producer should closely monitor the close-up pen to be able to detect metabolic and infectious diseases and use proper treatment to decrease stress and increase immune response.

Stress levels associated with cortisol can be caused by excessive pen moves, overcrowding, and rough handling of the animal. All of which affect the dry matter intake and ability of the animal's neutrophils to fight infection, allowing the animal to be more susceptible to reproductive and metabolic diseases.

Overall, it is the dairy producer's responsibility to have a sufficient management program in place to treat and prevent the reproductive and metabolic diseases of the herd in order to optimize maximum productivity. The management program should work closely with

a nutritionist in order to supply an appropriate feeding program for the transition cow when she is close to calving and after parturition. It is also crucial for the dairy producer to implement a disease treatment and prevention program with their veterinarian and herd managers. After all, cows are fragile mothers and should be treated with the best care, especially during the transition period.

REFERENCES

1 Badingla L., R.J. Collier, C.J. Wilcox, and W.W. Thatcher. Interrelationships of milk yield, body weight, and reproductive performance. 1985. J. Dairy Sci. 68:1828-1830. http://jds.fass.org/cgi/reprint/68/7/1828. Accessed Nov. 10, 2009.

2 Beauchemin K, Greg P. 2009. Tri-State Dairy and Nutrition Contest: New Developments in Understanding Ruminal Acidosis in Dairy Cattle. Pages 1-5. http://tristatedairy.osu.edu/Proceedings%202009/Beauchemin%20paper.pdf. Accessed Nov. 17, 2009.

3 Bishop, J. R., A. B. Bodine, G. D. O'DelI, and J. J. Janzen. 1984. Retention data for antibiotics commonly used for bovine infections. J. Dairy Sci.67:437. http://jds.fass.org/cgi/reprint/67/2/437.pdf. Accessed Nov. 17, 2009.

4 Block, Elliot. Progressive dairyman: Transitioning your cows to higher profit. Progressive Dairyman Publishing. 2009. http://www.progressivedairy.com/pd/features/2007/0307/0307_transition.html Accessed Nov. 12, 2009.

5 Calving intervention on U.S. dairy operations. 2007. Veterinary services: centers for epidemiology and animal health info sheet. United States Department of Agriculture: Animal and Plant Health Inspection Service. http://nahms.aphis.usda.gov/dairy/dairy07/Dairy2007_Calving_int.pdf. Accessed Nov. 15, 2009.

6 Corbett, Robert. 2009. Minimizing the effects of immunosuppression through management and nutrition. NMC Annual Meeting Proceedings. Dairy Health Consultation. Spring City, Utah. http://nmconline.org/articles/ImmSupp.pdf. Accessed Nov. 19, 2009.

7 Dystocia and dairy calf survival. 1998. Colorado Dairy News. http://www.cvmbs.colostate.edu/ILM/proinfo/cdn/2000articles/Dystocia%20Survivaval%20%20Nov00.pdf. Accessed Nov. 12, 2009.

8 Excenel RTU product detail. Pfizer Animal Health. 2008. Date accessed Nov. 12, 2009. http://www.excenel.com/Excenel.aspx?country=US&drug=RT&species=DA&sec=200

9 Horst, R.L., J.P. Goff, T. A. Reinhardt, and D. R. Buxton. 1997. Strategies for preventing milk fever in dairy cattle. J. Dairy Sci. 80:1269-1280. http://jds.fass.org/cgi/reprint/80/7/1269. Accessed Nov. 18, 2009.

10 Hutchinson, L.J. Trouble shooting infertility problems in dairy cattle. Pennsylvania State University. http://www.wvu.edu/~exten/infores/pubs/livepoul/dirm19.pdf. Accessed Nov. 13, 2009.

11 Jaster, E.H. 2009. Dairy Cattle Management. DSCI 333 Lecture and Activity Notes. El Corral Publications. Poor Richards Press. Cal Polytechnic State University. San Luis Obispo, California.

12 Kung, L. Acidosis in dairy cattle: ruminant nutrition and microbiology laboratory. http://ag.udel.edu/anfs/faculty/kung/articles/acidosis_in_dairy_cattle.htm. Accessed Nov. 15, 2009.

13 Lombard J.E., F.B. Garry, S.M. Tomlinson, and L.P. Garber. 2007. Impacts of dystocia on health and survival of dairy calves. J. Dairy Sci. 90:1751-1760. http://jds.fass.org/cgi/reprint/90/4/1751. Accessed Nov. 10, 2009.

14 Lucy, M.C. 2001. Reproductive loss in high producing dairy cattle: where will it end? J. Dairy Sci. 84:1277-1293. http://www.dairy-science.org/cgi/content/abstract/84/6/1277. Accessed Nov. 11, 2009.

15 Maas, J. 2009. Treating and preventing retained placenta in beef cattle. UCD Vet Views: California Cattlemen's Magazine. http://www.vetmed.ucdavis.edu/vetext/INF-BE_cca/INF-BE_cca08/cca0803-retained-placenta.pdf. Accessed Nov. 13, 2009.

16 Manspeaker, J.E. Retained Placentas: Dairy Integrated Reproductive Management. University of Maryland. 1-2. http://www.wvu.edu/~exten/infores/pubs/livepoul/dirm21.pdf. Accessed Nov.12, 2009.

17 The Merck Veterinary Manual. 1998. Pages 1017-1026. 8th ed. Merck & CO., INC. Whitehouse Station, N.J., U.S.A.

18 Metritis/fresh cow health. Pfizer Animal Health. 2009. Date accessed: Nov. 10, 2009. http://www.pfizerah.com/health.aspx?country=US&species=DA&lang=EN&drug=&t=2070&key=309&type=3

19 Mustvangwa, T. 2008. Sub-acute ruminal acidosis (SARA) in dairy cattle. Ontario Ministry of Agriculture Food and Rural Affairs. Factsheet. Queens Printer for Ontario. http://www.omafra.gov.on.ca/english/livestock/dairy/facts/03-031.htm Accessed Nov. 17, 2009.

20 Nebel and McGilliard. Agriculture and Food Development Authority. 1993. www.teagasc.ie/publications/ndc1999/paper2.asp

21 Opsomer, G., A. de Kuirf. Metritis and endometritis in high yielding dairy cows. 2009;78:83-89. http://vdt.ugent.be/code/showupload.php?id=473. Date accessed Nov. 09, 2009.

22 Radostits, O.M., D.C. Blood. 1994. Veterinary Medicine: A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats, and Horses. Pages 39-46. 8th ed. Bailliere Tindall. Paston Press Ltd., Loddon, Norfolk.

23 Stewart, P.G. 2009. Care and feeding of the dry and post-parturient cow. Cedara Agricultural Development Institute. http://agriculture.kzntl.gov.za/portal/AgricPublications/ProductionGuidelines/DairyinginKwaZuluNatal/TheCareandFeedingoftheDryandPostparturien/tabid/234/Default.aspx. Accessed Nov.18, 2009.

24 Tyler, H.D., Ensminger M.E. 2006. Dairy Cattle Science. 4th ed. Pages 371-381. Pearson Prentice Hall. Columbus, Ohio.

25 Weaver, D., B. Moseley. 1993. Displaced abomasums. University of Missouri Extension. http://extension.missouri.edu/publications/DisplayPub.aspx?P=G7701. Accessed Nov. 18, 2009.

26 Youngquist R.S, Walter R.T, 1997. Current Therapy in Large Animal Theriogenology. Pages 339-373. 2nd ed. Saunders Elsevier Inc. St. Louis, Missouri.