Causes of Embryonic Mortality in Cattle

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In recent years several estimates of embryonic mortality in cattle have been made. Embryonic mortality is generally defined as loss of the conceptus which occurs during the first 42 days of pregnancy, which is the period from conception to completion of differentiation when organ systems develop. Approximately 30 percent of all embryos and fetuses will not survive to birth. About 80 percent of this loss occurs before day 17, 10-15 percent between day 17 and 42 and 5 percent after day 42.

Early research at Penn State showed these losses to be much higher in "repeat breeders" those cows and heifers inseminated three or more times. This has a significant economic impact on dairy herd profitability. Because of the complex interactions among the various processes involved in establishment and maintenance of pregnancy, no single factor can be identified as the primary cause of embryonic death. The following are some major factors contributing to this loss:

It is well documented that short term exposure to heat stress several days before and after insemination results in low conception rate or embryonic death. This is due to elevated temperature of the uterine environment. This has been a serious problem affecting reproductive performance this spring and summer.

Chromosomal abnormalities are known to be a cause of embryo mortality. One research summary estimated an average of 10 percent embryos had gross chromosomal abnormalities. Most of these embryos were lost before day 12. Unfortunately, this problem cannot be controlled through management.

Nutritional factors have been shown to contribute to low conception rates resulting from embryonic mortality. Research studies have shown that cows with severe change in body condition score (greater than 1 point loss on a scale of 1 to 5) during the first five weeks postpartum had an extended interval to first service and a very low conception rate compared to herdmates with only a minor or moderate change in body condition.

Whether this is due to an abnormal hormonal situation such as reduced progesterone secretion or ovulation of a defective oocyte has not been determined. Several reports showed that feeding excess crude protein, excess degradable intake protein or low levels of fermentable carbohydrate and the various combinations of these nutrients can cause low conception rates. Such situations can produce excessive levels of ammonia in the blood and uteri of cows.

Some researchers believe this could be toxic to the gametes and the developing embryo. The excess ammonia that is not utilized by the rumen microbes must be converted to urea at a significant energy cost which can further adversely impact conception rate. Mycotoxins and exposure to toxic substances and toxic plants have also been implicated as a cause of embryonic mortality.

Infectious agents can cause uterine infection or directly affect the embryo causing death. The major organisms adversely affecting reproduction are : Corynebacterium pyogenes, Campylobacter fetus (Vibriosis), Haemophilius somnus, Leptospirosis, Neospora and the viruses bovine virus diarrhea (BVD), infectious bovine rhinotracheitis (IBR) and to a lesser extent Ureaplasma and Mycoplasma.

Uterine defense mechanisms including the immune system are critical to maintaining embryonic development. For example, recent reports show that subclinical mastitis and other systemic infections can substantially increase the incidence of early embryo loss. Furthermore, nutritional deficiencies or an environmental challenge can alter uterine immune function. Currently this is a very active area of research.

Hormonal patterns or imbalances associated with embryonic mortality have been identified. The challenge for researchers is to develop synchronization programs and treatments that provide high progesterone, maintain low estrogen, and lead to a highly functional corpus luteum to maintain pregnancy. Dairy producers should continue to keep informed about new systems that will be developed to minimize embryonic loss. However, these systems will not correct problems associated with nutrition, disease, heat stress or postpartum problems that challenge the uterine defense mechanism.

There are management practices that can be implemented to minimize the incidence of embryonic mortality. These include: proper transition cow management and nutrition, effective vaccination, providing a clean environment, minimize heat stress and proper timing of insemination. There is abundant data to support the fact that between 5 percent and 30 percent of the cows inseminated are not in estrus when bred. Not only will this situation result in a low fertilization rate but most likely such cows are inseminated when the uterus is under the influence of progesterone and the immune system is not geared up to combat an infectious agent introduced during insemination.

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