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The Impact of Heat Stress and Fescue Toxicosis on Beef Cattle Reproduction

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Heat stress is a common problem facing beef-cattle producers throughout the United States, and in 2003 was estimated to cost the U.S. beef industry $369 million dollars annually. Heat stress occurs when an animal is creating and absorbing more heat than they can dissipate, which increases core temperature and physiological stress. Common symptoms of heat stress include increased respiration rates, panting, or standing in water such as ponds or puddles. Heat stress results in negative impacts on growth, health, and reproduction. Cattle can have reduced conception rates, growth rates, and milk production during the summer. During severe heat stress, conception rates can fall to 10%-20% or lower compared to 40%-60% conception rates observed in cooler months.

The severity of heat stress is impacted by both temperature and humidity. The temperature humidity index (THI), which combines temperature and humidity into one value, is used to evaluate the risk of heat stress. THI levels are classified as: normal (<75), alert (75-78), danger (79-83), and emergency (>84), according to the University of Nebraska Lincoln Beef Cattle Temperature Humidity Chart (Figure 1). This means that in Kentucky and surrounding states, heat stress can regularly impact cattle from May through October.

Beef Cattle Temperature Humidity Chart

Temperature (°F) \ Relative Humidity (%)	30	35	40	45	50	55	60	65	70	75	80	85
100	84	85	86	87	88	90	91	92	93	94	95	97
98	83	84	85	86	87	88	89	90	91	93	94	95
96	81	82	83	85	86	87	88	89	90	91	92	93
94	80	81	82	83	84	85	86	87	88	89	90	91
92	79	80	81	82	83	84	85	85	86	87	88	89
90	78	79	79	80	81	82	83	84	85	86	86	87
88	76	77	78	79	80	81	81	82	83	84	85	86
86	75	76	77	78	78	79	80	81	81	82	83	84
84	74	75	75	76	77	78	78	79	80	80	81	82
82	73	73	74	75	75	76	77	77	78	79	79	80
80	72	72	73	73	74	75	75	76	76	77	78	78
78	70	71	71	72	73	73	74	74	75	78	76	76
76	69	70	70	71	71	72	72	73	73	74	72	75

Temperature Humidity Index (THI)

Normal <75 | Alert 75-78 | Danger 79-83 | Emergency >84

Figure 1. Cattle Temperature Humidity Index Chart.
Source: University of Nebraska

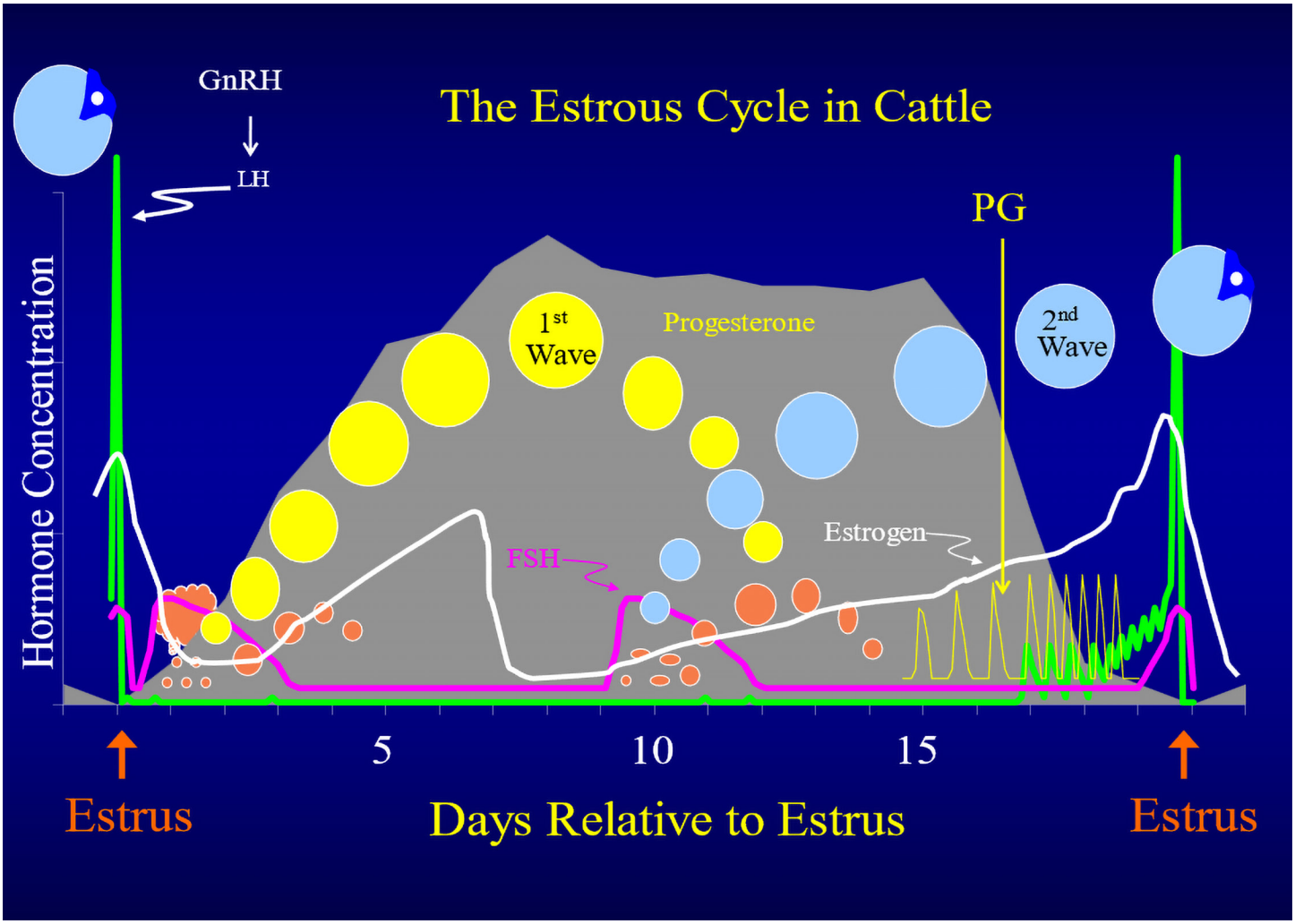

Figure 2. The normal estrous cycle of cattle.

Some animals are more susceptible to heat stress than others. For example, dark-colored cattle and cattle with heavy hair coats are more susceptible to heat stress than light-colored cattle and cattle with slick hair coats. Indicus breeds (Brahman, Brahman-hybrids, Santa Gertrudis, etc.) are less susceptible to heat stress than British and continental breeds (Angus, Hereford, Simmental, Charolais, etc.) due to physiological differences. Indicus cattle have more sweat glands, looser skin, and sleeker builds, resulting in a greater ability to dissipate heat. Even the forage cattle are grazing, such as toxic endophyte infected tall fescue, can make cattle more susceptible to heat stress.

Heat Stress and Female Reproduction

Heat stress negatively impacts each of the phases in the reproductive process. The estrous cycle (number of days between estrus events) is divided into the follicular and luteal phases. Under normal conditions during the follicular phase, a growing follicle on the ovary produces estrogen (Figure 2). Estrogen stimulates estrus behavior causing the female to be receptive to the male. During the follicular phase of heat-stressed animals, the emergence of the preovulatory follicle is hastened which reduces estrogen production. Decreased estrogen can result in poor estrus expression or "weak heats," making estrus detection a challenge for AI technicians and bulls alike. Estrogen not only stimulates estrus behavior, but it also stimulates a surge of hormones resulting in the rupture of the follicle and release of the oocyte. This process is called ovulation.

Once the follicle ruptures, the cells of the follicle collapse and form the corpus luteum (CL). The luteal phase is dominated by the presence of the CL and its production of the hormone progesterone. Progesterone is the "pregnancy hormone" and suppresses the expression of estrus. However, heat stress negatively impacts CL growth and function. This reduces progesterone concentrations, resulting in poor embryonic development and early fetal loss.

The embryo stage of pregnancy is from Day 0 to roughly Day 42 following ovulation. Cattle experiencing heat stress have slower embryo development up to Day 8, which is when the fetus begins to form. Approximately 15-17 days after estrus, the maternal system determines pregnancy and, if pregnant, the CL continues to produce progesterone until calving. Inhibited CL development due to heat stress can result in failure to establish pregnancy. A high THI during days 21-30 of pregnancy significantly increases risk of pregnancy loss. The reproductive process is complex, and possible pregnancies can be lost by failures in fertilization, pregnancy maintenance, and early embryonic growth (up to Day 45 of pregnancy).

Fescue Toxicosis and Female Reproduction

Cattle consuming toxic endophyte-infected tall fescue, such as KY-31, are more susceptible to heat stress at lower THI compared to cattle grazing other forages. The toxic fungal endophytes produce chemicals called alkaloids that cause vasoconstriction and many other biological changes collectively known as fescue toxicosis [refer to *Fescue Toxicosis in Cattle* (ID-221)]. Animals experiencing fescue toxicosis typically have the appearance of the heifer shown in Figure 3. Vasoconstriction is the reduction in diameter of blood vessels resulting in less blood flow to the periphery of the animal. Vasoconstriction does not allow the animal to properly dissipate heat. Fescue toxicosis also decreases serum prolactin, which is associated with hair-coat shedding. Additionally, research has found that alkaloid consumption can result in decreased hair-follicle cycling and sweat-gland productivity. The combination of vasoconstriction and decreased ability to sweat and shed hair significantly increases susceptibility to complications from heat stress.

Impacts of heat stress on beef cattle are amplified when consuming fescue containing toxic endophyte. Cattle experiencing heat stress and consuming endophyte-infected fescue have decreased total cholesterol (a hormone precursor) and further reductions in progesterone, diameter of the preovulatory follicle, and serum estrogen compared to cattle only experiencing heat stress. This results in lower conception rates and poor estrus expression. Fescue toxicosis can decrease conception rates by increasing the susceptibility to heat stress, even when environmental conditions would otherwise be considered thermoneutral.

Figure 3. Heifer experiencing fescue toxicosis. Note the rough, wiry haircoat and mud covering the animal. These are both indicators that the heifer is experiencing fescue toxicosis and is attempting to remain cool by laying/standing in mud or puddles.

Nutritional Deficiencies

One of the hallmarks of fescue toxicosis is decreased feed intake resulting in reduced average daily gain and body condition. Beef cattle breed best at a body condition score of 5 or greater and must be consuming ample feed to maintain their condition. Nutritional status plays an imperative role in the success of a breeding season and can be easily determined by body condition scoring. Heifers may be more susceptible to nutritional deficiencies due to their increased nutrient requirement to support development and growth.

Vasoconstriction of Reproductive Organs

Fescue toxicosis induced vasoconstriction may have more effects on reproduction than heat stress alone. Maternal recognition of pregnancy (MRP) occurs on days 15-17 of the estrous cycle and is a localized event between the uterus and ovary. These organs rely on blood flow from the uterine vein and ovarian artery for hormonal signaling. In one experiment, cycling heifers experienced vasoconstriction of the uterine and ovarian arteries during days 10-17 of the estrus cycle. This reduction of blood flow during a pertinent time of the estrous cycle could influence ovarian function and establishment of pregnancy.

Tissues throughout the body experience varying levels of vasoconstriction induced by alkaloids. Little is known about the effects of vasoconstriction on the placenta but work in sheep suggests that placentas of ewes consuming endophyte-infected fescue seed during mid to late gestation had reduced weight. Subsequently, lambs from these ewes weighed less than lambs of the ewes consuming endophyte-free tall fescue. A follow-up study found that while uterine blood supply was not affected by endophyte toxins, the umbilical arteries were, which may explain the decreased development of the lambs. In some cases, lower birth and weaning weights have been observed in calves born to cows consuming toxic tall fescue.

Alteration of Endocrine Signaling

Like heat stress, animals experiencing fescue toxicosis experience reduction in hormones that support the reproductive cycle including follicle stimulating hormone (FSH), luteinizing hormone (LH), estradiol, and progesterone. Unlike heat stress, animals consuming alkaloids have shown increased levels of prostraglandin-F2α present at abnormal times during the cycle, which can cause untimely CL regression and terminate a pregnancy. Circulating concentrations of prolactin can also be decreased during fescue toxicosis. Prolactin regulates hair coat shedding and supports CL function and lactation. When prolactin levels are decreased following parturition cows may have decreased milk production resulting in a lower rate of gain for offspring.

Heat Stress and Fescue Impacts on Male Reproduction

Reproduction in the male is less affected by heat stress than the female. This is likely because the testes are located outside of the body cavity and the scrotum is designed to reduce the temperature of the testes by 3.6-10.8°F allowing for normal sperm development. Research has demonstrated that if the temperature of the testes rises by two degrees for 48 straight hours, a bull can be rendered infertile for 60 days. Bulls who have been over conditioned and developed fat in the scrotum, which acts as an insulator, are more likely to experienced increased testicular temperatures especially during heat stress events. Even though heat stress may not make the bull infertile, it is likely that libido will be reduced, and pregnancy rates will be lower because the sexual activity of both the male and female is reduced.

Unlike the female, vasoconstriction caused by fescue toxicity does not decrease the fertility of the male by increasing heat stress of the gonads. Instead, the opposite occurs, where the lack of blood flow makes the testis cooler. Fescue toxicity does not affect semen motility, morphology, concentration, or volume, but oocytes fertilized with semen from bulls grazing endophyte-infected fescue have lower cleavage rates and lower survival in the first week of pregnancy. Thus, consumption of endophyte-infected fescue by bulls decreases fertility, even though these bulls may pass a standard breeding soundness exam. It is important to mitigate the effects of heat stress and fescue toxicity in the bull because this can affect the entire herd of females that it will be servicing.

Summary

Fescue toxicosis and heat stress need to be mitigated as much as possible to improve reproductive success. The impact of consumption of endophyte-infected fescue can be mitigated by diluting pastures with warm season grasses and legumes such as red clover [refer to *Fescue Toxicosis in Cattle* (ID-221)]. Pastures can be renovated to introduce novel endophyte varieties of tall fescue which do not result in fescue toxicosis of grazing animals, or other non-fescue forage varieties. Strategic grazing management to keep cattle off infected fescue during breeding may also help. Heat stress is more of a challenge but ensuring that animals have access to shade and water are essential. Heat stress can also be mitigated by moving breeding seasons to cooler times of the year, such as November and December for fall calving.

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