

The Effect of Heat Stress on Reproductive Hormone Secretion

Jennifer A. Spencer, Ph.D.
Extension Dairy Specialist
Department of Animal Sciences
Texas A&M AgriLife Extension Service
The Texas A&M University System

During the summer, the conception rates of dairy cattle decrease by 20% to 30% due to higher temperatures and humidity¹. Heat stress contributes to the increased number of inseminations required in dairy cows during the summer². This is due to the decreased duration and intensity of estrus, increased pregnancy loss, and the negative effects on ovarian, uterine and embryo function³⁻⁵.

Heat stress is measured using the temperature-humidity index (THI). The THI value is influenced by a variety of factors, including solar radiation, air movement and precipitation. But the most important factors influencing this value are the ambient temperature and relative humidity⁶. Literature has indicated that a THI value >68 constitutes heat stress in dairy cattle⁷ (Figure 1). This is equivalent to 80°F with 0% humidity or 72°F and 50% humidity. Nevertheless, the duration and severity of heat stress exposure can intensify the negative effects on dairy cows. Heat stress negatively impacts follicular/oocyte development, embryo development, the uterine environment, hormonal secretions, and overall fertility. This article will focus on discussing the effects of heat stress on reproductive hormones and how these effects in part contribute to reproductive inefficiencies in cattle.

Temperature (°F)	% Relative Humidity					
	0	20	40	50	60	80
72	64	66	67	68	69	70
76	66	68	70	71	72	74
80	68	70	73	74	75	78
84	70	73	75	77	78	81
88	72	75	78	80	81	85
92	73	77	81	83	85	88
96	75	79	83	86	88	92
100	77	82	86	88	91	95
104	79	84	89	91	94	99

Figure 1. Temperature humidity index values. Different colors correspond to degrees of heat stress mild to moderate (yellow); moderate-severe (orange); and severe (red) heat stress.

Hormone Secretion Before Ovulation

Heat stress affects reproductive hormone secretions in many ways. The follicle is a structure on the ovary that produces estrogen and contains the oocyte or “egg,” the female contribution for fertilization. Normally a group of follicles begin to mature on the ovary of a cow, however, through selection a dominant follicle will produce sufficient concentrations of estrogen to cause ovulation.

Research has shown that both the oocyte and follicle are sensitive to increased temperatures⁸. In fact, if cattle experience heat stress before ovulation, the follicle development is delayed. If the follicle development is delayed or slowed, blood estrogen concentrations do not reach the threshold needed to cause ovulation in a timely manner^{5,9-11}. In addition, heat stress impacts oocyte development within the follicle which can reduce the success of fertilization and embryo development^{13,14}.

The slower follicle development is in part due to the degeneration of the estrogen producing cells, which will then impair the development of the corpus luteum (CL)⁹⁻¹². The CL is the structure that develops after ovulation and produces progesterone needed to maintain pregnancy. Collectively, these heat-induced hormonal changes before ovulation can contribute to reduced estrus expression, ovulation rates, fertilization, embryo development and embryo quality^{3-5,9}.

Hormone Secretion After Ovulation

After ovulation, the hormone cells of the follicle transform and become the hormone producing cells of the CL producing progesterone. Progesterone inhibits ovulation and helps the uterus establish and maintain pregnancy. Progesterone secretion also inhibits the secretion of prostaglandin $F_{2\alpha}$, the hormone produced and secreted by the uterus causing CL regression thereby reducing progesterone concentrations for ovulation to occur. The regression of the CL and reduced progesterone concentrations are needed for final maturation of follicles and to increase estrogen concentrations for ovulation¹⁵.

However, the delay of follicle development will result in the prolonged presence of the CL. That, then, extends the time between ovulations and reduces the number of times a cow is eligible to be bred^{4,5}. On the other hand, if the CL were developed from a compromised follicle due to heat stress, the CL would produce less progesterone⁹⁻¹². Lesser concentrations of progesterone would allow for premature secretion of prostaglandin $F_{2\alpha}$ causing the death of the CL and decrease in progesterone concentrations before pregnancy can be established. Therefore, compromised hormone producing cells of the follicle and CL can contribute to embryonic loss.

Heat stress also alters the uterine environment and can make it unsuitable for embryo development. In an *in vitro* study, endometrial cells recovered from heat-stressed cows during the time of embryonic signaling for maternal recognition of pregnancy had increased secretion of prostaglandin $F_{2\alpha}$ compared to endometrial cells that were not exposed to heat stress¹⁶. This is important, as prostaglandin $F_{2\alpha}$ causes the regression of the CL producing progesterone necessary for maintenance of pregnancy. In this scenario, early CL regression would cause a reduction of progesterone concentrations and reduce

embryonic growth and development, delaying embryo signaling for maternal recognition of pregnancy and further increasing the risk of embryonic loss.

Conclusion

Heat stress disrupts the normal reproductive hormone concentrations necessary for cyclicity and establishment of pregnancy. Additionally, heat stress has detrimental effects on the oocyte, follicle, CL, and uterus. Together, these contribute to the reduced conception rates of cattle which are observed in the summer when temperatures and humidity are high. Proper management and cooling of cows can help in reducing the detrimental effects of heat stress, and help to improve reproductive performance.

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