Heat stress and seasonal effects on reproduction in the dairy cow—a review

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Abstract

In dairy cows inseminated during the hot months of the year, there is a decrease in fertility. Different factors contribute to this situation; the most important are a consequence of increased temperature and humidity that result in a decreased expression of overt estrus and a reduction in appetite and dry matter intake. Heat stress reduces the degree of dominance of the selected follicle and this can be seen as reduced steroidogenic capacity of its theca and granulosa cells and a fall in blood estradiol concentrations. Plasma progesterone levels can be increased or decreased depending on whether the heat stress is acute or chronic, and on the metabolic state of the animal. These endocrine changes reduce follicular activity and alter the ovulatory mechanism, leading to a decrease in oocyte and embryo quality. The uterine environment is also modified, reducing the likelihood of embryo implantation. Appetite and dry matter intake are both reduced by heat stress thus prolonging the postpartum period of negative energy balance and increasing the calving-conception interval, particularly in high producing dairy cows. The utilization of cooling systems may have a beneficial effect on fertility but dairy cows cooled in this way are still unable to match the fertility achieved in winter. Recent studies suggest that the use of gonadotropins to induce follicular development and ovulation can decrease the severity of seasonal postpartum infertility in dairy cows.

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1. Introduction

Heat stress is a major contributing factor to the low fertility of dairy cows inseminated in the late summer months [1–4]. The decrease in conception rate during the hot season can
range between 20 and 30% compared to the winter season [5–7]. There are clear seasonal patterns of estrus detection, day to first service and conception rate in dairy cows [5,7–9] and lower conception rates are consistently observed in summer months compared to winter months.

The effects of heat stress on fertility appear to carry into the autumn [6,10–12]. The low fertility generally associated with the warm months of the year (June to September) remains in the autumn (October and November) even though the cows are no longer exposed to heat stress [13]. It has been suggested that this could be a lasting effect of heat stress during the hot months on the antral follicles that will develop into large dominant follicles 40–50 days later [14,15].

1.1. The effect of heat stress on reproductive patterns in dairy cows

In heat stressed cows the duration and intensity of estrus was reduced in some studies [16,17] but was unchanged in others [18]. The balance of these and other studies suggest that heat stress reduces the duration and intensity of estrus in dairy cattle. For example, in summer, motor activity and other manifestations of estrus are reduced [13,19] and the incidence of anestrus and silent ovulation are increased [17]. These effects lead to a reduction in the number of mounts in hot weather compared to cold weather [20], leading to poor detection of estrus. Therefore, in hot climates there is a reduction in the number of inseminations and an increase in the proportion of inseminations that do not result in pregnancy.

1.2. The effect of heat stress on the hypothalamic–hypophyseal–ovarian axis

Since the main factors regulating ovarian activity are gonadotropin-releasing hormone from the hypothalamus and the gonadotropins, luteinizing hormone (LH) and follicle stimulating hormone (FSH) from the anterior pituitary gland, some authors have studied the effect of heat stress on the secretion of these hormones. The effects of heat stress on LH concentrations in peripheral blood are inconsistent. Some studies report unchanged concentrations [17,21] while others report increased concentrations [22] and still others report decreased concentrations [23–26] following heat stress. With regard to the pattern of LH secretion in heat stressed cows, decreases in LH pulse amplitude [25] and LH pulse frequency [24] have been reported. The effect of heat stress on the preovulatory surge of LH is similarly controversial: a reduction of the endogenous LH surge by heat stress was reported in heifers [23] but not in cows [17,21,27]. The reasons for these discrepancies are unclear. It has been suggested that these differences are related to preovulatory estradiol levels because the amplitude of tonic LH pulses and GnRH-induced preovulatory plasma LH surges are decreased in cows with low plasma concentrations of estradiol but not in cows with high plasma concentrations of estradiol [25].

Plasma inhibin concentrations in summer are lower in heat stressed cows [28] and in cyclic buffaloes in India [29], perhaps reflecting reduced folliculogenesis since a significant proportion of plasma inhibin comes from small and medium size follicles. Concentrations of plasma FSH are higher during the preovulatory period in summer; this was associated with lower circulating concentrations of inhibin [4]. However, a reduced
FSH response in heat stressed compared to control cows was observed after administration of a GnRH analogue [25].

Because most studies report that LH levels are decreased by heat stress, we are drawn to conclude that in summer, the dominant follicle develops in a low LH environment and this results in reduced estradiol secretion from the dominant follicle leading to poor expression of estrus, and hence, reduced fertility. The small amount of published information available on the effect of heat stress on blood concentrations of FSH and inhibin in cattle suggests that FSH is increased by heat stress and this may be due to decreased plasma inhibin production by compromised follicles. Further research is required before a conclusion can be reached. However FSH, if increased, appears insufficient to overcome the effect of low LH concentrations and therefore a reduced availability of androgen precursors for estradiol synthesis [30].

Plasma estradiol concentrations are reduced by heat stress in dairy cows [11,28,32]; an effect that is consistent with decreased concentrations of LH and reduced dominance of the selected follicle. However, the effect of heat stress on plasma progesterone concentration is more controversial. Some studies [32] found that heat stress had no effect on the plasma progesterone concentrations but that luteolysis was delayed. Several other studies have reported increased [33–35], decreased [16,18,36–38] or unchanged [30,39] blood concentrations of this hormone during summer heat stress in dairy cows. These differences probably arise because of uncontrolled changes in other factors that affect blood progesterone concentrations. For example, the type of heat stress (i.e. acute or chronic) and differences in dry matter intake will independently affect blood progesterone concentrations, thus confusing the situation. Plasma progesterone concentrations are determined by the differences between the rate of luteal production and the rate of hepatic metabolism and both of these are also affected by changes in dry matter intake.

If the concentration of plasma progesterone is reduced by heat stress this would have consequences for fertility. Low plasma progesterone concentrations during the luteal phase of the pre-conception estrous cycle can compromise follicular development leading to abnormal oocyte maturation and early embryonic death [40]. During the conception cycle low progesterone concentrations can also lead to the failure of implantation [41,42]. In the conception cycle, the effect of progesterone is most probably related to the need for synchronous development of the embryo, and delayed or advanced development of the corpus luteum will lead to higher rates of implantation failure [42]. In fact, it has been reported that the pattern of the post-ovulatory rise in progesterone is related to fertility [43]. However, the use of exogenous progesterone post-insemination to supplement endogenous progesterone has mixed effects on pregnancy rate, with one paper reporting an improvement [44] and another reporting no benefit [45] of this treatment.

The mechanisms by which heat stress alters the concentrations of circulating reproductive hormones are not known. Increased corticosteroid secretion has been suggested [46] because this can inhibit GnRH and thus LH secretion [25]. In a detailed study, heat stress inhibited the secretion of gonadotropins to a greater degree in cows with low plasma concentrations of estradiol compared to those with high concentrations [25]. This study suggests that high concentrations of estradiol can counteract the effect of heat stress, or alternatively, that the neuroendocrine mechanism controlling gonadotropin secretion is more sensitive to heat stress when concentrations of plasma estradiol are low. It has been
suggested that heat stress could also act directly on the ovary to decrease its sensitivity to gonadotropin stimulation [11].

Regardless of the precise mechanism, any alteration in the secretory activity of the follicle and perhaps the corpus luteum caused by heat stress would be important factors in summer infertility.

1.3. The effect of heat stress on gametes and embryos

The formation of gametes is temperature sensitive. Normal spermatogenesis requires a temperature that is below normal body temperature and recent evidence indicates that the development of oocytes is also temperature sensitive [47]. The effect of heat stress on fertility might then be the result of a direct effect of high ovarian temperatures on oocyte quality.

The intrauterine environment is also compromised in cows that are heat stressed; there is a decrease in blood flow to the uterus and an increase in uterine temperature [48,49]. These changes inhibit embryonic development [50], increase early embryonic loss and reduce the proportion of successful inseminations. High ambient temperature will also affect pre-attachment stage embryos [1] but the magnitude of the effect decreases as embryos develop [51]. The production of embryos by superovulation is often reduced [52,53] and embryonic development compromised [54] in hot seasons. Heat stress can affect endometrial prostaglandin secretion [55], leading to premature luteolysis and embryo loss. Most embryo loss occurs before Day 42 in heat stressed cows [56].

1.4. Heat stress effect on follicular development

Heat stress delays follicle selection and lengthens the follicular wave and thus has potentially adverse effects on the quality of oocytes [14,15,57] and follicular steroidogenesis [14,15,18,28]. Summer heat stress reduces the degree of dominance of the dominant follicle and more medium-size subordinate follicles survive [28,30,32,56,57]. Thus, the duration of dominance of the preovulatory follicle is increased in summer, and in beef heifers, duration of dominance is negatively correlated with fertility [59]. When individual follicular dominance is reduced, more than one dominant follicle can develop and this may explain the increase in twinning that may be seen in summer [60]. In this way, heat stress can both decrease follicular steroid secretion and increase twinning rate at the same time.

1.5. Heat stress and energy balance

It is likely that heat stress affects reproductive performance both by direct actions on reproduction and by indirect actions mediated through alterations in energy balance. In the dairy cow, there is an interaction between dry matter intake, stage of lactation, milk production, energy balance and heat stress that results in reduced LH secretion and a decreased diameter of the dominant follicle in the postpartum period [36,38]. Since one of the main causes of anovulation in dairy cows, especially during the early postpartum period, is prolonged negative energy balance [61,62], any worsening of energy balance during summer would further decrease fertility in dairy cows at this time.
In heat stressed dairy cows there is a reduction in dry matter intake [12,13,31,36], which prolongs the period of negative energy balance. Negative energy balance leads to decreased plasma concentrations of insulin, glucose and IGF-I, and increased plasma concentrations of GH and non-esterified fatty acid [62–64]. All of these metabolic hormones can affect reproduction. Metabolic hormones acting on the hypothalamo-pituitary axis and the ovary probably mediate the inhibitory effects of negative energy balance on postpartum fertility.

The plasma concentrations of insulin, IGF-I and glucose are decreased in summer months compared to winter months [7,38,64–67] probably because of low dry matter intake and increased negative energy balance. Insulin is required for the development of follicles and has beneficial effects on oocyte quality [68]. Both IGF-I and glucose are generally stimulatory to follicular growth and implantation and glucose is a primary fuel for the ovary [69]. Glucose availability is also directly involved in modulating LH secretion [70], and severe hypoglycemia inhibits pulsatile LH secretion and prevents ovulation [64]. This is another mechanism by which heat stress and its associated reduction in dry matter intake decrease postpartum fertility of dairy cows. In addition, the metabolic hormone prolactin is temperature sensitive and its levels in summer are increased [34,36,71]. Prolactin can inhibit follicular development and suckling-induced prolactin secretion is a cause of increased postpartum anestrus in suckled cattle [72].

All these data indicate that heat stress alters dry matter intake and as a consequence, postpartum animals may go further into negative energy balance leading to decreased concentrations of plasma insulin and IGF-I and eventually even glucose. Since all of these factors are essential for normal folliculogenesis their lower plasma concentrations lead to impaired follicular development, poor estrus detection and poor quality oocytes.

In conclusion, heat stress has a wide range of effects on the reproductive axis (Fig. 1). Some of these effects directly affect individual reproductive organs such as the hypothalamus, the anterior pituitary gland, the uterus, the follicle and its oocyte and the embryo itself (Fig. 1), while the other effects of heat stress are indirect and probably mediated by changes in the metabolic axis in response to reduced dry matter intake (Fig. 1). We suggest that there is not a single mechanism by which heat stress can reduce postpartum fertility in dairy cows and that this problem is due to the accumulation of the effects of several factors.

1.6. Production systems that can improve fertility during summer months

1.6.1. Temperature and humidity control

One of the factors that increase the calving-conception interval of dairy cow during the hot season of the year is poor detection of estrus. The use of tailhead paint, the HeatWatch system, radio-telemetric pressure transducers and pedometers can improve estrus detection and thus fertility. However, there are no published studies that have evaluated the effects of these aids to estrus detection on summer infertility. Some dairy producers in Italy are turning to the use of natural breeding during the summer in an attempt to overcome poor estrus detection and improve fertility. However, the benefit of improved estrus detection is offset by deterioration in bull fertility caused by heat stress [75].

Various other attempts have been made to overcome the effects of heat stress on fertility, including the use of shade, fans, air-conditioning and sprinkler systems to cool animals
during summer [71,73,76,77]. The most widely used methods are cooling systems that mist the cows with water from overhead sprays and cool the air. The use of these systems has produced some improvement of fertility but they were still unable to match the level of normal winter fertility [13,73–76].

1.6.2. Mineral and vitamin supplements

Heat stress is associated with reduced total antioxidant activity in blood plasma [78] and there is some evidence that the depression in embryo survival following exposure to elevated temperatures involves increased free radical production [79]. However, the short-term administration of the antioxidant vitamin E at the time of AI or at 30 days postpartum had no beneficial effect on pregnancy rate during summer [80], nor did the administration of selenium or β-carotene [81]. In contrast, long-term β-carotene supplements had a beneficial effect on fertility in lactating cows [82,83].

1.6.3. Embryo transfer

Embryo transfer can be used to bypass the harmful effects of heat stress on oocyte quality that limit embryonic development [84,85]. A recent study [86] used timed embryo
transfer to study the effect of heat stress on fertility in lactating dairy cows. These results show that timed embryo transfer improved pregnancy rates under heat stress conditions but only when fresh embryos were transferred [86].

1.6.4. Hormonal therapy

An alternative approach to improving summer fertility is the use of reproductive hormones to stimulate fertility. While not necessarily addressing the fundamental causes of summer infertility, this approach offers the opportunity to overcome its effects on fertility.

In heat stressed cows, the administration of GnRH induces follicular development and a healthy preovulatory follicle [39]. In summer, the administration of GnRH to lactating dairy cows at estrus increased the conception rate from 18 to 29% [87]. However, luteal support from a single administration of hCG (3000 iu) on Day 5 or 6 after insemination did not improve summer fertility [88]. Similar results have been reported following exogenous administration of progesterone with the CIDR intravaginal delivery device [89]. In recent years the effect of timed artificial insemination (i.e. without estrus detection) on fertility in summer has been examined [89,90]. The results of these studies suggest that these techniques can help overcome the effects of heat stress and reduce summer infertility.

The use of fixed time insemination (AI) has the distinct advantage of not requiring the detection of estrus and effective synchronization methods for fixed time AI have been developed. They are based on administration of GnRH or hCG to induce ovulation, followed by a luteolytic dose of prostaglandin F2α 6 to 7 days later and a second treatment with GnRH or hCG 24–60 h after the luteolytic treatment to induce a fertile ovulation [88,92–94]. In the summer these programs did not increase the number of cows pregnant to the fixed time insemination but they did increase the number of cows pregnant by 120 days postpartum and reduced the number of days open [7,9,58,83,90,91]. These results suggest that the principal benefit of these treatments is to induce cyclicity and the development of normal corpora lutea leading to good fertility. These approaches lead to an increase in the number of pregnant cows simply by increasing the number of cows that are inseminated at estrus, and although they limit the effects of summer heat stress on fertility, they do not address the underlying pathology.

2. Conclusions

There is a widely observed decrease in the fertility of postpartum dairy cows inseminated in the summer compared to cows inseminated in winter. The precise mechanism of this effect has not been conclusively identified. However, the plasma levels of LH and estradiol are decreased in heat stressed cows and this is one of the main factors contributing to low fertility during the hot months of the year. With regard to FSH, there is also general agreement that its secretion in summer is increased probably due to decreased inhibin secretion from small follicles. Research into the effects of heat stress on plasma progesterone report an inconsistent picture and the role of progesterone in summer infertility is not very clear. There are many possible reasons for the lack of agreement among the published reports. These include: the type of stress, (chronic versus acute),
Fig. 2. The two main pathways by which heat stress can affect fertility in lactating dairy cow. The numbers in parentheses refer to cited references. Hyperthermia leads to increased lethargy and a compromised uterine environment, both of which can lead to worsening infertility through poor estrus detection and embryo loss. Poor appetite leads to lower dry matter intake, thus exacerbating the effects of negative energy balance in early lactation. Negative energy balance produces lower blood concentrations of insulin and IGF-I, and higher blood concentrations of GH and NEFA, and this altered metabolic profile acting via the hypothalamo-pituitary system reduces GnRH and LH secretion, leading to reduced estradiol secretion by the dominant follicle. The consequences of reduced estradiol secretion from the dominant follicle are poor estrus detection, compromised oocyte quality, and in extreme situations, ovulatory failure. The role of progesterone in summer infertility, if any, remains uncertain and controversial.
genetic factors associated with milk yield (high milk versus low/normal milk), and environmental and management factors.

The principal site of action of heat stress on the reproductive axis appears to be hypothalamic (Fig. 1). However, additional sites of action in the ovary and the uterus also appear to be important (Fig. 1).

There appear to be two distinct and largely independent pathways by which heat stress leads to infertility (Fig. 2). The first is a direct effect of hyperthermia on the reproductive axis. The second is an indirect effect related to the effects of heat stress on appetite and dry matter intake, both of which are reduced by heat stress. The consequence is a worsening energy balance and since the postpartum dairy cow tends to be in negative balance, the consequences of heat stress on fertility are more likely to be severe. There is a reduction in LH secretion leading to reduced estrogen secretion, impaired detection of estrus, reduced oocyte quality, implantation failure and infertility. Heat stress will also change the secretion of thyrotrophic (thyroxine) and adrenocorticotrophic (cortisol) and adrenomedullary hormones (adrenaline). These may potentially impair fertility. However, there is a paucity of data examining the effects of heat stress on these systems in relation to dairy cow fertility.

At least three options are available to reduce or eliminate summer infertility. The use of cooling systems in hot weather has a beneficial effect but these alone do not restore normal fertility. Additionally, summer infertility can be alleviated by the provision of high quality forage and feed to overcome negative energy balance and by the use of hormonal treatments to induce normal cyclicity.

References


