African Journal of Biotechnology Vol. 10(29), pp. 5552-5558, 22 June, 2011
Available online at http://www.academicjournals.org/AJB
DOI: 10.5897/AJB09.033
ISSN 1684–5315 © 2011 Academic Journals

Review

Alterations in reproductive hormones during heat stress in dairy cattle

M. Khodaei-Motlagh[1]*, A. Zare Shahneh[2], R. Masoumi[2] and Fabio Derensis[3]

[1]Department of Animal science, Faculty of Agriculture, Arak university, Arak 38156-8-8349. Iran.
[2]Department of Animal Science, Faculty of Agronomy and Animal Science, College of Agriculture and Natural Resources, University of Tehran, P.O.BOX # 31587-4111 Karaj. Iran.
[3]Dipartimento di Salute Animale, Facoltà di Medicina Veterinaira, Università di Parma

Accepted 10 December, 2010

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. Systems activated by heat stress can influence reproduction at the hypothalamus, pituitary gland or gonads. However, the major impact is thought to be within the brain or at the pituitary gland. Measurement of plasma concentrations of the gonadotrophins provides a good indication of the effects at these higher levels, since the pulsatile secretion of luteinizing hormone (LH) is a reflection of the secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus. Both the secretion and actions of GnRH are influenced by the feedback actions of gonadal sex steroids and inhibin. 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.

Key words: Reproductive hormones, dairy cow and heat stress.

INTRODUCTION

The most comfortable and productive environmental temperature range for dairy cows is between -4 to 8°C (Khodaei Motlagh et al., 2010 and McDowell, 1972). This is the thermal comfort zone. Summer heat stress is a major factor contributing to low fertility in lactating dairy cows. Conception rates drop from about 40 to 60% in cooler seasons to 10 to 20% in warm seasons (Cavestany et al., 1985). The substantial rise in average milk yields in recent years has aggravated the low summer fertility syndrome, because of the concurrent rise in metabolic heat production. Every dairy cow has a natural body heat balance which is optimal for normal physiological processes and productive functions. A normal body heat balance exists only when the net heat gain equals the net heat loss.

Stimuli that challenge homeostasis like heat are commonly called stressors. Heat stress can be defined

*Corresponding author. E-mail: mmotlagh2002@gmail.com.

Abbreviations: FSH, Follicle stimulating hormone; **LH,** luteinizing hormone; **GnRH,** gonadotropin-releasing hormone; **ACTH,** adrenocorticotropic hormone; **CRF,** corticotrophin-releasing factor; **AVP,** arginine vasopressin; **CIDR,** controlled intra-vaginal delivery device.

as the sum of forces external to a homeothermic animal that acts to displace body temperature from the resting state (Yousef, 1984). Such a stress can disrupt the physiology and productive performance of an animal. The increase in body temperature caused by heat stress has direct, adverse consequences on reproductive hormones (Hansen and Arechiga, 1999). Hence, heat stress is a major contributing factor to the low fertility of dairy cows inseminated in the late summer month (Ingraham et al., 1974, Ray et al., 1992; Thompson et al., 1996; Al-Katanani et al., 1999; Khodaei et al., 2006). Effects of heat stress on reproductive hormones and other physiological functions are a direct consequence of the increase in body temperature caused by heat stress or of the physiological changes cows undergo to reduce the magnitude of hyperthermia (De Renesis and Scaramuzzi, 2003; Khodaei et al 2003). High ambient temperature has affected adversely normal reproduction in cattle (Plasse et al., 1970), swine (Edwards et al., 1968) and sheep (Thwaites, 1968), the syndrome being short estrus, abnormal estrus cycle, increased proportion of abnormal ova shed, decreased fertilization rate, and increased embryonic and fetal mort-ality early in gestation (Stott, 1972). These physiological manifestations as influenced by environmental heat have been associated with altered endocrine functions (Stott and Wierama, 1971). A number of experimental condi-tions have been used to evaluate hormonal secretion during heat stress including short-term and long-term temperature modification using environmental chambers, seasonal comparisons of hormonal profiles and the use of microclimatic modification during periods of heat stress. Differences in experimental conditions have pre-sumably contributed to the disparity of results that are found in the literature concerning hormonal secretions during heat stress. Some of the variation in hormonal responses to heat stress probably reflects the fact that ovarian steroid concentrations are dependent not only on rate of secretion from ovarian tissue but also on rate of vascular perfusion of the ovary, on possible adrenal release (at least for progesterone), on metabolism in the liver and other organs, and on the degree of hemodilution or hemoconcentration (Wise et al., 1988). The extent to which heat stress affects these other physiological characteristics could lead to variable changes in steroid hormone concentrations in peripheral blood. For example, heat stress can cause either dilution, concentration, or no effect on blood plasma volume (Richards, 1985; McGuire et al., 1989; Johnson et al., 1991; Elvinger et al., 1992), and the nature of effect of heat stress on blood volume will affect steroid hormone concentrations in blood. Hyperthermia has been shown to decrease ovarian blood flow (Lublin and Wolfenson, 1996) and to inhibit angio-genesis (Fajardo et al., 1988). Blood flow and vascular density determine the follicular perfusion rate, which directly influences the rates of nutrient uptake and hormonal release by the follicle. The relationships among heat stress, vascularity and steroidogenic capacity require further investigation. Although, heat stress impacts on the reproductive axis at the hypothalamus (to affect gonadotropin-releasing hormone (GnRH) secretion) and the pituitary gland (to affect gonadotrophins secretion), with direct effects on the gonads being of less importance.

EFFECTS OF HEAT STRESS ON REPRODUCTIVE HORMONES

Estradiol and progestron

Secretion of the hormones regulating reproductive tract function such as estradiol-17 and progestrone may be altered by heat stress. The effect of heat stress on plasma progesterone concentration is controversial. Wilson et al. (1998) found that heat stress had no effect on the plasma progesterone concentrations but that luteolysis was delayed. Several other studies have reported increased (Trout et al., 1998; Abilay et al., 1975; Vaught et al., 1977), decreased (Rosenberg et al., 1977, Younas et al., 1993; Howell et al., 1994; Jonsson et al., 1997; Ronchi et al., 2001) or unchanged (Roth et al., 2000; Guzeloglu et al., 2001) 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 (that is, acute or chronic) and differences in dry matter intake will independently affect blood progesterone concen-trations, thus confusing the situation. The cells of the corpus luteum differentiate from the cells of the follicle therefore, if heat stress decreases blood progesterone then the decrease could arise from the effects of heat stress on the follicle which ultimately carries over to the corpus luteum (Roth et al., 2001). Plasma progesterone concentrations are determined by the differences bet-ween the rate of luteal production and the rate of hepatic metabolism. Both are 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 (Ahmad et al., 1995). During the conception cycle, low progesterone concentrations can also lead to the failure of implantation (Mann et al., 1999; Lamming and Royal 2001). 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 (Lamming and Royal 2001). In fact, it has been reported that the pattern of the post-ovulatory rise in progesterone is related to fertility (Darwash et al., 1999). However, the use of exogenous progesterone post-insemination to supplement endogenous progesterone has mixed effects on pregnancy rate, with one paper

reporting an improvement (Robinsson et al., 1989) and another reporting no benefit (Breuel et al., 1990) of this treatment.

It has been shown that early atresia of bovine follicles is characterized by a decrease in androgen production by thecal cells (McNatty et al., 1984). Early atresia in medium sized follicles because of heat stress could also be associated with low oestradiol production by granulosa cells and increased progesterone concentrations in the follicular fluid of heat-stressed cows (Roth et al., 2000).

Plasma estradiol concentrations are reduced by heat stress in dairy cows (Wolfenson et al., 1997; Wolfenson et al., 1995; Wilson et al., 1998) an effect that is consistent with decreased concentrations of luteinizing hormone (LH) and reduced dominance of the selected follicle, although, this effect has not always been observed (Rosenberg et al., 1982). Also recent experiments suggest that heat stress can cause an increase in peripheral concentrations of estradiol-17 between day 1 and 4 of the estrous cycle (Wolfenson et al., 1995) and a reduction from day 4 through 8 (Wolfenson et al., 1995) and 11 through 21 of the cycle (Wilson et al., 1998). These results must be compared with earlier reports of no effect of heat stress (Roman-Ponce et al., 1981; Wise et al., 1988ab). Decrease of estradiol concentration in the follicular fluid is more likely to occur after exposure to long-term, chronic (summer) heat stress than to acute heat stress.

The mechanisms by which heat stress alters the concentrations of circulating reproductive hormones are not known. Some effects of heat stress may involve adrenocorticotropic hormone (ACTH). Heat stress can cause increased cortisol secretion (Roman-Ponce et al., 1981; Wise et al., 1988a; Elvinger et al., 1992), and ACTH has been reported to block estradiol- induced sexual behavior (Hein and Allrich, 1992). Increased corticosteroid secretion has been suggested (Roman-Ponce et al., 1977) because this can inhibit GnRH and thus LH secretion (Gilad et al., 1993). 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 (Gilad et al., 1993).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 (Wolfenson et al., 1997). Also the somatic cells within the follicles (theca and granulosa cells) can be damaged by heat stress. In terms of steroid production, the thecal cells and granulosa cells were found to be susceptible to heat stress (Roth et al., 2001).

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.

Follicle stimulating hormone (FSH), luteinizing hormone (LH) and Inhibin

Since the main factors regulating ovarian activity are gonadotrophin-releasing hormone from the hypothalamus and the gonadotrophins, LH and 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 (Gwazdauskas et al., 1981; Howel et al., 1994; Nobel et al., 1997; Pennington et al., 1985; Gauthier, 1986) while others report increased concentrations (Roman-Ponce et al., 1981) and still others report decreased concentrations (Modon and Johnson, 1973; Wise et al., 1988; Gilad et al., 1993; Lee, 1993) following heat stress. With regard to the pattern of LH secretion in heat stressed cows, decrease in LH pulse amplitude (Gilad et al., 1993) and LH pulse frequency (Wise et al., 1988) 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 (Modon and Johnson, 1973) but not in cows (Gwazdauskas et al., 1981; Gauthier, 1986; Rosenburg et al., 1982). 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 (Gilad et al., 1993). 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 these results in reduced estradiol secretion from the dominant follicle leading to poor expression of estrus, and hence, reduced fertility. Plasma inhibin concentrations in summer are lower in heat stressed cows (Wolfenson et al., 1993) and in cyclic buffaloes in India (Palta et al., 1997), perhaps reflecting reduced folliculogenesis since a significant proportion of plasma inhibin comes from small and medium size follicles.

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. However, a reduced FSH response in heat stressed compared to control cows was observed after administration of a GnRH analogue (Gilad et al., 1993). 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 (Roth et al., 2000).

Gonadotrophins and corticostroids

Usually, in long-term a stressor like heat stress activates the hypothalamo pituitary–adrenal axis and the sympathoadrenal system (Tibrook et al., 2000). Stimulation of the hypothalamo-pituitary-adrenal axis is characterized by activation of corticotrophin-releasing factor (CRF) and arginine vasopressin (AVP) neurones in the paraventricular nucleus, and secretion of these neuropeptides into the hypophysial portal system to stimulate the corticotrophs of the anterior pituitary gland (Tibrook et al., 2000). The corticotrophs produce a variety of peptides derived from pro-opiomelanocortin, including ACTH, endorphin and melanocyte-stimulating hormone, all of which are released in response to heat stress (Engler et al., 1989). In terms of the response to heat stress, the physiological significance of the release of -endorphin and melanocyte-stimulating hormone is not clear, but ACTH acts on the cortex of the adrenal glands to stimulate the synthesis and secretion of glucocorticoids like cortisol. Higher concentration of this catabolic hormone may have been produced by heat stressed cows to maintain milk production (Abilay et al., 1975). It is tempting to speculate that the higher concentrations of cortisol in the heat stressed cows influences LH release, because cortisol has been implicated as an inhibitor of anterior pituitary release of LH in the bovine species (Gangwar et al., 1965). Various studies have shown that administration of natural or synthetic glucocorticoids can inhibit the secretion of the gonadotrophins in sheep (Juniewicz et al., 1987), pigs (Turner et al., 1999a, b) and especially in dairy cattle (Thibier and Rolland, 1976). Nevertheless, increased secretion of glucocorticoids is not always associated with decreased secretion of the gonadotrophins, particularly in cases of acute stress. Suppression of reproduction is more likely under conditions of chronic stress and may involve action at the hypothalamus or pituitary. Furthermore, there may be species differences in the extent to which glucocorticoids inhibit the secretion of LH and FSH (Tilbrook et al., 2000). When heat stress is prolonged, it is likely that the secretion of the gonadotrophins will be suppressed and reproduction will be inhibited. However, for acute stress or repeated acute stress, it is unclear whether, or how, reproduction will be affected. It is apparent from various studies that glucocorticoids can inhibit gonadotrophin secretion in some circumstances.

Prostaglandin

It has been shown that heat stress can increase secretion from endometriom and in some cases early activation of luteolysis mechanism, leading to failure in implantation. For instance heat shocks of 42 and 43°C increased output of prostaglandins by cultured endometrium collected at day 17 of the estrous cycle (Putney et al., 1988b; Malayer and Hansen 1990). Furthermore, heat stress on day 17 of pregnancy increased uterine production o in response to oxytocin (Wolfenson et al., 1993).

Androstenedion

Culture of thecal cells at 40.5°C reduced androstenedione production from cultured thecal cells but generally had no effect on estradiol-17 production from cultured granulosa cells (Wolfenson et al., 1997). The mechanism by which heat stress induces a decrease in androstenedione production in thecal cells is not clear. Recent analyses of mRNA content for LH receptor in thecal cells obtained from preovulatory follicles did not provide any evidence for alterations of mRNA content related to previous heat exposure (Roth et al., 2000). However, the significant decrease of LH - stimulated androstenedione production by thecal cells may indicate that heat exposure induces impairment of LH receptor function. Heat stress from day 3 to 5 of the estrous cycle increased androstenedione and reduced estradiol-17 concentrations in follicular fluid of the dominant follicle collected at day 7 (Wolfenson et al., 1997).

USE OF EXOGENOUS HORMONES

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 (Guzeloglu et al., 2001). In summer, the administration of GnRH to lactating dairy cows at estrus increased the conception rate from 18 to 29% (Ullah et al., 1996). However, luteal support from a single administration of human chorionic gonadotropin (hCG) (3000 iu) on day 5 or 6 after insemination did not improve summer fertility (Schmitt et al., 1996). Similar results have been reported following exogenous administration of progesterone with the CIDR intravaginal delivery device (Wolfenson et al., 1994). In recent years the effect of timed artificial insemination (that is, without estrus detection) on fertility in summer has been examined (Wolfenson et al., 1994; De la Sota et al., 1998). 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 (TAI) has the distinct advantage of not requiring the detection of estrus and effective synchronization methods for fixed time insemination have been developed. They are based on

administration of GnRH or hCG to induce ovulation, followed by a luteolytic dose of 6 to 7 days later and a second treatment with GnRH or hCG 24 to 60 h after the luteolytic treatment to induce a fertile ovulation (Schmitt et al., 1996; Pursley et al., 1995; Schmitt et al., 1996). 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 (De Rensis et al., 2002; Almier et al., 2002; Wolfenson et al., 2000; Arechiga et al., 1998; De la Sota et al., 1998; Cartmil et al., 1999). 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.

Injecting cows with GnRH causes a predictable release of LH and a significant increase in serum progesterone. In one study, Ullah et al. (1996) indicated that if cows were injected with 100 ug of GnRH when estrus was first detected, followed by insemination (AI) ten or twelve hours later, the pregnancy rate from the first AI was 28.6% for all treated cows. This can be compared to 17.7% in the control cows. The same ovulation synchronization protocol has also been used by Lee (1999) in Taiwan. The pregnancy rate of lactating cows in summer, when cows were first serviced postpartum using Ovsynch, was 25.0%. This can be compared to the 15% pregnancy rate when cows were first serviced when estrus was detected. The results suggest that timed AI using the Ovsynch protocol in mid-lactation is an effective and economic way of managing the reproduction of lactating cows in a subtropical environment.

CONCLUSION

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 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), genetic factors associated with milk yield (high milk versus low/normal milk), and environmental and management factors.

As discussed above heat stress may affect the secretion of the gonadotrophins through mechanisms that modify the synthesis or the secretion of GnRH, the responsiveness of the gonadotrophs to the actions of GnRH or the feedback actions of gonadal hormones. Prolonged or chronic heat stress results in suppressed gonadotrophins secretion and inhibition of reproduction but, when the duration of the stress response is transient or acute, the effects are less clear. Variable effects of acute stress have been found in dairy cattle, with reports of no effect, stimulatory and inhibitory effects on reproductive hormones secretion. To reduce the adverse effects of heat stress on reproductive hormones during heat stress, it has been suggested to use exogenous hormones like GnRH, progesterone and hCG.

REFERENCES

Abilay TA, Johnson HD, Madam LM (1975). Influence of environmental heath on peripheral plasma progesterone and cortisol during the bovine oestrus cycle. J. Dairy Sci: 58:1836-1840.

Abilay TA, Johnson HD, Madan M (1975). Influence of environmental heat on peripheral plasma progesterone and cortisol during the bovine estrous cycle. J. Anim. Sci. 58: 1836-1840.

Ahmad N, Schrick FN, Butcher RL, Inskeep EK (1995). Effect of persistent follicles on early embryonic losses in beef cows. Biol. Reprod. 52:1129-1135.

Al-Katanani YM, Webb DW, Hansen PJ (1999). Factors affecting seasonal variation in non-return rate to first service in lactating Holstein cow in a hot climate. J Dairy Sci, 82: 2611-2616.

Almier M, De Rosa G, Grasso F, Napolitana F, Bordi A (2002). Effect of climate on the response of three oestrus synchronisation techniques in lactating dairy cows. Anim. Reprod. Sci. 71:157-68.

Arechiga CF, Staples CR, McDowell LR, Hansen PJ (1998). Effects of timed insemination and supplemental beta-carotene on reproduction and milk yield of dairy cows under heat stress. J. Dairy Sci. 81: 390-402

Breuel KF, Spitzer JC, Thompson CE, Breuel J (1990). First-service pregnancy rate in beef heifers as influenced by human chorionic gonadotrophin administration before and/or after breeding. Theriogenology, 34: 139-145.

Cartmill JA, Hensley BA, El-Zarkouny SZ, Rozell TG, Smith JF, Stevenson JS (1999). An alternative AIbreeding protocol during summer heat stress. [abstract]. J Dairy Sci. 82:48.

Cavestany D, El-Whishy AB, Foote RH (1985). Effect of season and high environmental temperature on fertility of Holstein cattle. J. Dairy Sci. 68: 1471-1478.

Darwash AO, Lamming GE, Woolliams JA (1999). The potential for identifying heritable endocrine parameters associated with fertility in post-partum dairy cows. Anim. Reprod. Sci. 68: 333-347.

De la Sota RL, Burke JM, Risco CA, Moreira F, DeLorenzo MA, Thatcher WW (1998). Evaluation of timed insemination during summer heat stress in lactating dairy cattle. Theriogenology, 49: 761-770.

De Rensis F, Marconi P, Capelli T, Gatti F, Facciolongo F, Franzini S., (2002). Fertility in postpartum dairy cows in winter or summer following estrous synchronization and fixed time A.I. after the induction of an LH surge with Gonadotropin releasing hormone (GnRH) or human chorionic gonadotropin (hCG). Theriogenology, 58:1675-1687.

Edwards RI, Omtvedt IT, Turman EJ, Stephens DE, Mahoney GWA (1968). Reproductive performance of gilts following heat stress prior to breeding and in early lactation. J. Anim. Sci. 27: 1634.\

Elvinger F, Natzke RP, Hansen PJ (1992). Interactions of heat stress and bovine somatotropin affecting physiology and immunology of lactating cows. J. Dairy Sci. 75: 449-462.

Engler D, Pham T, Fullerton MJ, Ooi G, Funder JW, Clarke IJ (1989). Studies of the secretion of corticotrophin-releasing factor and arginine vasopressin into the hypophysial-portal circulation of the conscious sheep. Neuroendocrinology, 49: 667-381.

Fajardo LF, Prionas SD, Kowalski J, Kwan HH (1998). Hyperthermia inhibits angiogenesis. Radiation Research. 114: 297-306. Gangwar PC, Branton C, Evans DL (1965). Reproductive and physiological response of Holstein heifers to controlled and natural
climatic conditions. J. Dairy Sci. 48: 222-227.

Gilad E, Meidan R, Berman A, Graber Y, Wolfenson D (1993). Effect of heat stress on tonic and GnRH-induced gonadotrophin secretion in relation to concentration of oestradiol in plasma of cyclic cows. J.
Reprod. Fertil. 99: 315-321.

Guzeloglu A, Ambrose JD, Kassa T, Diaz T, Thatcher MJ, Thatcher WW (2001). Long term follicular dynamicsand biochemical
characteristics of dominant follicles in dairy cows subjected to acute heat stress. Anim. Reprod. Sci. 66:15-34.

Hansen PJ, Arechiga CF (1999). Strategies for managing reproduction in the heat-stressed dairy cows. J. Anim. Sci. 77(2): 36-50.

Hein KG, Allrich RD (1992). Influence of exogenous adrenocorticotropic hormone on estrous behavior in cattle. J. Anim. Sci. 70: 243-247. Howell JL, Fuquay JW, Smith AE (1994). Corpus luteum growth and unction in lactating Holstein cows during spring and summer. J. Dairy Sci. 77: 735-739.

Khodaei Motlagh M (2003). Study of Some Effective Factors in Reproductive Performance of Iranian Holstien Cows Thesis animal
physiology, Tehran University. Iran.

Khodaei Motlagh, Roohani MZ, Zare A, moradi Share Babak M (2006). Study of Some Effective Factors In Reproductive Performance in sharif abad Ghazvin company and Shir-o-dame Boniad. Proceedings of 2end congress animal science. Tehran. Iran.

Khodaei Motlagh M, Zare A, moradi Share Babak M, Deldar H, Zhandi M, Ansari Z., Effect of heat stress on reproductive performance. (2010). Proceeding of the first national conference of environmental stresses in agricultural science. p. 275.

Ingraham RH, Gillette DD, Wagner WD (1974). Relationship of temperature and humidity to conception rate of Holstein cows in
subtropical climate. J. Dairy Sci. 57: 476-481.

Johnson HD, Li R, Manalu W, Spencer-Johnson KJ (1991). Effects of somatotropin on milk yield and physiologicalresponses during
summer farm and hot laboratory conditions. J. Dairy Sci. 74: 1250-1262.

Jonsson NN, McGowan MR, McGuigan K, Davison TM, Hussain AM,

Juniewicz PE, Johnson BH, Bolt DJ (1987). Effect of adrenal steroids on testosterone and luteinizing hormone secretion in the ram. J. Androl. 8: 190-196.

Lamming GE, Royal MD (2001). Ovarian hormone patterns and subfertility in dairy cows. In: Diskin MG, editor. Fertility in the highproducing dairy cow, BSAS Endiburgh: Occasional Publication. 26:105-118.

Lee CN (1993). Environmental stress effect on bovine reproduction. Vet. Clin. North Am. 9:263-273.

Lee SN (1999). Use of GnRH-PGF-GnRH as a method of timed insemination to improve fertility in lactating Holstein cows in southern Taiwan. J. Chin. Soc. Anim. Sci. 28(3): 373-380.

Lublin A, Wolfenson D (1996). Effect of bllod flow to mammary and reproductive systems in heat-stressed rabbits. Comp. Biochem.
Physiol. 115: 277-285.

Malayer JR, Hansen PJ (1990). Differences between Brahman and Holstein cows in heat-shock induced alterations of protein secretion by oviducts and uterine endometrium. J. Anim. Sci. 68: 266-280.

Mann GE, Lamming GE, Robinson RS, Wathes DC (1999). The regulation of interferon-tau production and uterine hormone receptors during early pregnancy. J. Reprod. Fertil. 54: 317-328.

McDowell RE (1972). Improvement of Livestock Production in Warm Climates. Freeman, San Francisco Press, California, USA.

McGuire MA, Beede DK, DeLorenzo MA, Wilcox CJ, Huntington GB, Reynolds CK, Collier RJ (1989). Effects of thermal stress and level of feed intake on portal plasma flow and net fluxes of metabolites in lactating Holstein cows. J. Anim. Sci. 67: 1050-1060.

McNatty KP, Heath DA, Henderson KM, Lun S, Hurst PR, Ellis LM, WMntgomery G, Mrrison L, Hurly DC (1984). Some aspects of thecal and granulosa cell function during follicular development in the bovine ovary. J. Reprod. Fertil. 72: 39-53.

Palta P, Mondal S, Prakas BS, Madan ML (1997). Peripheral inhibin levels in relation to climatic variations and stage of estrous cycle in buffalo (Bubalus bubalis). Theriogenology, 47: 898-995.

Plasse D, Warnick AC, Koger J (1970). Reproductive behavior of Bos indicus females in a subtropical environment. IV. Length of estrous cycle, duration of estrus, time of ovulation, fertilization and embryo survival in grade Brahman heifers. J. Anim. Sci. 30: 63.

Pursley JR, Mee MO, Wiltbank MC (1995). Synchronization of ovulation in dairy cows using PGF2alpha and GnRH. Theriogenology, 44: 915-923.

Putney DJ, JR Malayer TS. Gross WW, Thatcher P J. Hansen, Drost M (1988b). Heat-stress induced alterations in the synthesis and secretion of proteins and prostaglandins by cultured bovine conceptuses and uterine endometrium. Biol. Reprod. 39: 717-728.

Ray DE, Halbach TJ, Armstrong DV (1992). Season and lactation number effects on milk production and reproduction in dairy cattle in Arizona. J. Dairy Sci. 75: 2976-2983.

Renesis FD, Scaramuzzi RJ (2003). Heat stress and sesonal effects on reproduction in the dairy cow. Theriogenology, 60: 1139-1151.

Richards JI (1985). Effect of high daytime temperature on the intake and utilization of water in lactating Friesian cows. Trop. Anim. Health Prod. 17: 209-217.

Robinson NA, Leslie KE, Walton JS (1989). Effect of treatment with progesterone on pregnancy rate and plasma concentrations of
progesterone in Holstein cows. J. Dairy Sci. 72: 202-207.

Roman-Ponce H, Thatcher WW, Wilcox CJ (1981). Hormonal interrelationships and physiological responses of lactating dairy cows
to a shade management system in a subtropical environment. Theriogenology, 16: 139-154.

Roman-Ponce H, Thatcher WW, Buffington DE, Wilcox CJ, Van Horn HH (1977). Physiological and production responses of dairy cattle to a shade structure in a subtropical environment. J. Dairy Sci. 60: 424-430.

Ronchi B, Stradaioli G, Verini Supplizi A, Bernabuci U, Lacetera N, Accorsi PA,(2001). Influence of heat stress or feed restriction on plasma progesterone, oestradiol-17beta, LH, FSH, prolactin and cortisol in Holstein heifers. Livestock Prod Sci. 68: 231-241.

Rosenberg M, Herz Z, Davidson M, Folman J (1977). Seasonal variations in post-partum plasma progesterone levels and
conceptions in primiparous and multiparous dairy cows. J. Reprod. Fertil. 51: 363-367.

Rosenberg M, Folman Y, Herz Z, Flamenbaum I, Berman A, Kaim M (1982). Effect of climatic conditions on peripheralconcentrations of LH, progesterone and oestradiol-17b in highmilk-yielding cows. J. Reprod. Fertil. 66: 13-146.

Roth Z, Meidan R, Braw-Tal R, Wolfenson D (2000). Immediate and delayed effect of heat stress on follicular development and its
association with plasma FSH and inhibin concentration in cows. J. Reprod. Fertil. 120: 83-90.

Roth Z, Meidan R, Shaham-Albalancy A, Braw-Tal R, Wolfenson D (2001). Delayed effect of heat stress on steroid production in
medium-sized and preovulatory bovine follicles. Anim. Reprod. Sci. 121: 745-751.

Schmitt EJP, Diaz T, Barros CM, De-la-Sota RL, Drost M, Fredriksson EW., (1996). Differential response of the luteal phase and fertility in cattle following ovulation of the firstwave follicle with human chorionic gonadotropin or agonist of gonadotropin-releasing hormone. J. Anim. Sci. 74: 1074-1083.

Schmitt EJ-P, Diaz T, Drost M, Fredriksson EW, Thatcher WW (1996). Use of a gonadothropin releasing hormone or human chorionic gonadotrophin for timed insemination in cattle. J. Anim. Sci. 74: 1084-1091.

Stott GH (1972). Climatic thermal stress. A cause of hormone depression and low fertility in bovine. Biometeor. (Abstr.) 5: 113.

Stott GH, Wiersma F (1971). Plasma corticoids as an index of acute and chronic environmental stress. Presental at First Conference on Biometeorology and Tenth Conference on Agricultural Meteorology, Columbia, MO.

Thibier M, Rolland O (1976). The effect of dexamethasone (DXM) on circulating testosterone (T) and luteinizing hormone (LH) in young

postpubertal bulls Theriogenology, 5: 53-60
Thibier M, Rolland O (1976). The effect of dexamethasone on circulating testosterone and luteinizing hormone in young post pubertal bulls. Theriogenology, 5: 53-60.
Thompson JA, Magee DD, Tomaszewski MA, Wilks DL, Fourdraine RH (1996). Management of summer infertility in Texas Holstein dairy cattle. Theriogenology, 46: 547-558.
Thwaites CJ (1968). The influence of age of ewe on embryo mortality under heat stress conditions. Int. J. Biometeor. 12: 29.
Tilbrook AJ, Turner AI, Clarke IJ (2000). Effects of heat stress on reproduction in non-rodent mammals: the role of glucocorticoids and sex differences. J. Reprod. Fertil. 5: 105-113.
Trout JP, McDowell LR, Hansen PJ (1998). Characteristics of the oestrous cycle and antioxidant status of lactating Holstein cows
exposed to stress. J. Dairy Sci. 81: 1244-1250.
Turner AI, Hemsworth PH, Canny BJ, Tilbrook AJ (1999a). Inhibition of the secretion of LH by sustained but not repeated acute elevation of cortisol in the absence but not the presence of stradiol. J. Endocri. 163: 477-486.
Turner AI, Hemsworth PH, Canny BJ, AJ Tilbrook (1999b). sustained but not repeated acute elevation of cortisol impaired the LH surge, estrus and ovulation in gilts. Biol. Reprod. 61: 614-620.
Ullah G, Fuquay JW, Keawhoong T, Clark BL, Pogue DE, Murphy EJ (1996). Effect of gonadotrophin-releasing hormone at estrus on subsequent luteal function and fertility in lactating Holstein during heat stress. J. Dairy Sci. 79: 191950-191953.
Vaught LW, Monty DW, Foote WC (1977). Effect of summer heat stress on serum LH and progesterone values in Holstein-Frisian cows in Arizona. Am. J. Vet. Res.38: 1027-1032.
Wilson SJ, Marion RS, Spain JN, Spiers DE, Keisler DH, Lucy MC (1998). Effect of controlled heat stress on ovarian function in dairy cattle: I. Lactating cows. J. Dairy Sci. 1:2124-2131.
Wilson SJ, Marion RS, Spain JN, Spiers DE, Keisler DH, Lucy MC (1998). Effect of controlled heat stress on ovarian function in dairy cattle: I. Lactating cows. J. Dairy Sci. 1: 2124-2131.
Wise ME, Armstrong DV, Huber JT, Hunter R, Wiersma F (1988). Hormonal alterations in the lactating dairy cow. J. Dairy Sci. 71: 2480-2484.
Wise ME, Armstrong DV, Huber JT, Hunter R, Wiersma F (1988a). Hormonal alterations in the lactating dairy cow in response to thermal stress. J. Dairy Sci. 71: 2480-2485.
Wise ME, Rodriguez RE, Armstrong DV, Huber JT, Wiersma F, Hunter R (1988b). Fertility and hormonal responses to temporary relief of heat stress in lactating dairy cows. Theriogenology, 29: 1027-1035.
Wolfenson D, Kaim M, Rosemberg M (1994). Conception rate of cows supplemented with progesterone postinsemination in the summer. J. Anim. Sci. 72(1): 280.
Wolfenson D, Lew BJ, Thatcher WW, Graber Y, Meidan R (1997). Seasonal and acute heat stress effects on steroid production by
dominant follicles in cow. Anim. Reprod. Sci. 47: 9-19.
Wolfenson D, Roth Z, Meidan R (2000). Impaired reproduction in heatstressed cattle: basic and applied aspects. Anim. Reprod. Sci. 60/61: 535-547.
Wolfenson D, Thatcher WW, Badinga L, Savio JD, Meidan R, Lew BJ., (1995). The effect of heat stress on follicular development during the estrous cycle dairy cattle. Biol. Reprod. 52: 1106-1113.
Wolfenson D, Bartol FF, Badinga L, Barros CM, Marple DN, Cummings K, Wolfe D, Lucy MC, Spencer TE, Thatcher WW (1993). Secretion of PGF2a and oxytocin during hyperthermia in cyclic and pregnant heifers. Theriogenology, 39: 1129-1141.
Younas M, Fuquay JW, Smith AE, Moore AB (1993). Estrus and endocrine responses of lactating Holsteins to forced ventilation during summer. J. Dairy Sci. 76: 430-434.
Yousef MK (1984). Stress physiology: definition and terminology. In: Yousef, M.K. (Ed.) Stress Physiology in Livestock. CRC Press, Boca Raton, FL. pp. 3-7.