Chapter

Adverse Impact of Heat Stress on Bovine Development: Causes and Strategies for Mitigation

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Abstract

Heat stress induces the richness and reproductive domesticated animal's performance by settling the physiology conceptive steps, through hormonal irregularity, diminished oocyte quality and feeble semen quality, and diminished undeveloped organism advancement and endurance. It depends on principally milk production, nutrition, disease management, sexual activities, and heat stress tolerance capacity in livestock farming. The decreases infertility caused by elevated blood heat influences sex gland regulation, oestrus regulation, and gametocyte disturbance and also affects embryonic development. Heat stress reduces the degree of dominance of the seminal vesicles and this may be observed as reduced steroidogenic capability of its theca and granulose cells as fall in blood oestrogen concentrations. Plasma progestin levels are also diminished counting on whether or not the heat stress is acute and on the metabolic state of the animal. The endocrine changes the cyst activities and alters the ovulatory mechanism leading to a decrease in gametocyte and embryo quality. Summer infertility may be countered through oestrus behaviour can be mitigated by with the help of implementation of ovulation phase treatments to limited period of embryonic transfer and also advanced reproductive technologies involving hormonal treatments, systematic artificial insemination and which may enhance the possibility of establishing pregnancy in domestic animals.

Keywords: Heat stress, livestock, Mitigation, Infertility, Reproductive

1. Introduction

Global warming has multifaceted consequences for livestock today, which exhibits as heat stress, lack of feed and fodder, and alters in epidemiological patterns of vector borne diseases among other things resulting in decline in reproduction performance in production. In Dairy and Beef industries heat stress is the major cause for production loss. Bovines are homoeothermic organisms sustain a balanced body temperature by balancing the quantity of metabolic activities generated heat and also the heat depletion to the surroundings [1]. The heat development and loss keep body temperatures in a narrow range, but illness, inadequate nutrition, and extreme environmental temperatures can disturb the metabolism.

At the time of heat stress, animal productivity and reproduction output reduced dramatically. It also declines the rate of food consumption, milk production, dairy cow health, and reproduction. The upper crucial limit of thermo-neutral zone cattle

is approximately 25°C. When the temperature exceeded above the 25°c denotes that dairy cattle can get affected by heat stress [2]. In Lactating cattle's are further vulnerable to heat stress for that reason lactation causes high metabolic energy production, which can lead to hyperthermia. Heat stress appears to have an impact on fertility in the autumn [3]. The lack of fertility, usually related with the June, September, October and November. Thus, cattle are no longer being affected to heat stress [3]. The antral follicle may get affected by the heat stress for long term which further developed 40th to 50th days later into an enlarged dominant follicle [3]. The oocyte get effected by heat stress at the time of pre-ovulatory cycle with involvement of oxidative stress detected in vivo and vitro studies [4, 5]. The administration of antioxidants reduced heat shock in vitro [5]. The embryo pre-implantation is vulnerable towards heat stress, but this vulnerability show reduction in developing embryo. The state of energy balance in lactating cattle's influenced the pattern of follicular growth as well [6]. The negative energy balance and their factors affect the lactation phase, milk productivity level, intake of calcium salts of long-chain fatty acids in energy-rich nutrients and vaccination of bovine somatrophin under the pressure of follicular dynamics. The post-partum lactating and non-lactating cattle's have different numbers of large follicles and E2 ratios during the pre-ovulatory phase. The Temperature-Humidity Index (THI) is a commonly used environmental assessment index for assessing heat stress in dairy production [7]. The values of THI can be categorised into four groups based on the degree of heat stress faced by dairy cattle's. According to Armstrong [2] normal heat stress (71), middle heat stress up to (72 to 79) moderate heat stress (80 to 90), and harsh heat stress (> 90). In tropical and subtropical climates, the THI 72 level is the threshold for high output in terms of lactating and reproduction. The recent studies on THI in temperate climates, on have found that a THI of less than 68 is appropriate for cattle efficiency and welfare.

Heat stress can described with the help of temperature-humidity index (THI) reading with the purpose of is consistently above the thermo-neutral region and has a negative impact on a cattle's efficiency. Thus, the THI > 72 has been linked to heat stress in beef cattle [8] while THI 75 has been linked to heat stress in bulls [8]. Since THI does not account for exposure to radiation or wind velocity, it may underestimate climatic stress in beef cattle.

The objective of this chapter is to describe what could be known about the mitigated strategies for following to overcome heat stress which impairs embryo development and to address physiological, genetic and environmental problems and to enhance bovine production in hot weather.

2. Heat stress imbalances reproductive hormones

The ovarian functions are controlled by gonadotropin hormone (GnRH) which are secreted from the hypothalamus which help in the activation of pituitary gland which further secrete the luteinizing hormones (LH), follicle stimulating hormone (FSH) and gonadotropin hormones [2]. The impact of heat stress on peripheral blood Luteinizing hormone yet to be determined, as some studies have found an increase, decrease or even no effect [2] of heat stress on LH. The lack of LH levels can also disturbs the secretion of estradiol from dominant follicle which causes greater impact on oestrus cycle, maturation of follicles and also decreases the ovarian functions [2]. However, estradiol is essential for ovarian follicle growth, oocyte maturation, and endometrial proliferation. Furthermore, FSH and LH [9], insulin-like growth factor (IGF), LH [9] and anti-Mullerian hormone (AMH) LH [9] have distinct receptors in granulosa cells (GCs) any disruption in GC quality or proliferation capacity can

have an indirect impact on follicle growth, disrupting oocyte maturation resulting in impaired embryo development and an unsatisfactory pregnancy outcome LH [9]. Heat stress reduces plasma estradiol concentrations in dairy cattle's [10] which is consistent with lower luteinizing hormone (LH) concentrations and reduced follicle dominance, though this outcomes has not always been seen [10]. There is also widespread consensus that FSH secretion increases in the summer, owing to reduced inhibit secretion from small follicles. When a stressor affect the hypothalamic–pituitary–adrenal axis (HPA) which are responsible to stimulate the hormone such as gonadotropin releasing hormone (GnRH), vasopressin, releasing hormone (CRH) and glucocorticoids [11]. while progesterone, gonadotropins, prolactin, and glucagon rises [11]. Furthermore, glucocorticoids minimise the vulnerability of target tissues to sex steroids by inhibiting pituitary development of gonadal steroids. The rapid initial release of LH is induced by arachidonic acid and its metabolites, whereas the prolonged release of LH is mediated by protein kinase C-dependent mechanisms. By inhibiting the hydrolysis of phospholipids and thus, preventing the synthesis of arachidonic acid, glucocorticoids reduces the release of LH. Gonadal steroids also have ability to control pituitary gonadotropin activity is also influenced by glucocorticoids [11]. The amount of gonadal steroid hormones will decline in the presence of glucocorticoids over hours or even days [11] disrupting reproductive physiology, behaviour and lowering feeding and appetite [11].

3. Alteration in the mechanism of female reproduction by heat stress

Cattles reported an elevated occurrence of early embryo development during warm seasons for a variety of reasons. Heat stress leads to may adverse conditions at various phases of female reproduction (**Figure 1**). The direct impact of heat stress on oocyte competence and follicular is one of the major cause [2]. Furthermore, adverse impact of heat stress on cattle's super ovulation response, as well as the number and quality of recovered embryos [2]. The heat stress also reduces weight along with diameter of the corpus luteum, as well as the amount of progesterone it releases and the consistency of the oocytes, both contributes to pregnancy loss. The heat stress changes the endometrial environment by up regulating glycoprotein 2 and neurotensin, which can lead to infertility during the summer [11]. All of these reasons decrease the rate of fertilisation and the quality of any resulting embryos, raising the risk of pregnancy failure and lowering reproductive success. On the other hand, beef cattle [11] and dairy cattle have both shown this behaviour. The rate of conception in lactating cattle's decreases as the strength of the heat stress increases. The heat stress event can also affect conception rates from the month before breeding to two weeks after breeding [12]. In addition to these, heat stress is also linked to a smaller concepts scale, which could affect maternal pregnancy recognition and corpus luteum function [12]. Furthermore, heat stress has been linked to a compromised pregnancy during the pre-implantation phase, with an increased risk of foetal loss between days 21 to 30 of pregnancy [12]. The lack of blood flow in uterine can also show impact on nutrients supplementation to embryo and also lesser the secretion of hormones of uterus [12], can further have complicate things. The lactation of dairy cow is primarily effected by heat stress. During heat stress, non-lactating dairy animals and beef cattle are far less likely to become infertile. The conception rates in Holsteins decreased in the summer in Florida for lactating cattle's but not for non-lactating heifers [13]. However, the large amount of quantity of heat emission can process to lactation in the cattle's but the lactating cow is particularly vulnerable to heat stress. The lactation in cattle's will experience hyperthermia (high body temperature) at temperatures as low as 77-284°F [13].

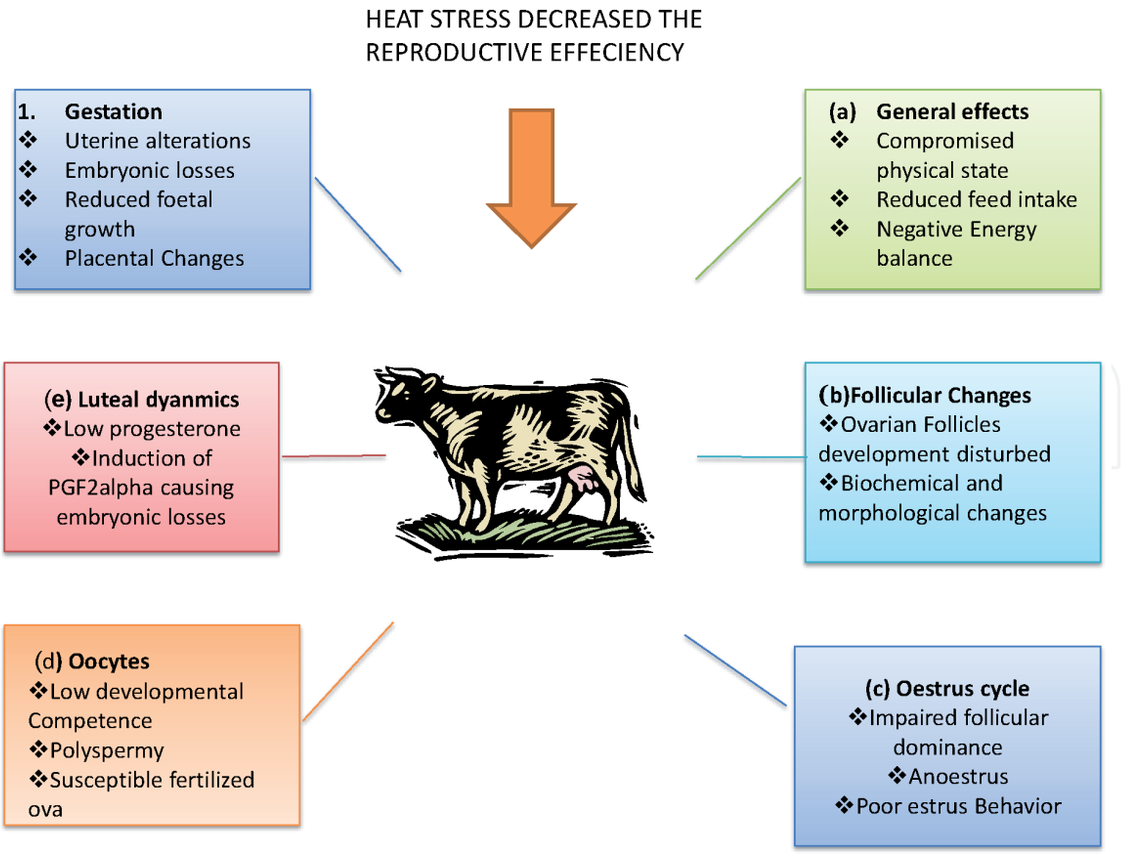

Figure 1.
Impact of heat stress at various phase of female reproduction.

3.1 Heat stress impact on oocyte development at different stages

3.1.1 Oestrus expression

The time interval and potency of oestrus are also abridged by heat stress. Heat stress reduced follicular estradiol, which may have lowered the level of oestrus. The physical inactivity brought in by heat stress may also be a factor in decreased oestrus speech. During oestrus, cattle's become less active hence, less likely be carried out to other cattle's. As a result, dairy cattle's in the summer had about half as many mounts per oestrus as dairy cattle's in the winter. It also diminished the oestrus activity as a result, the Cattles decreased motor activity, which is an attempt to minimise endogenous heat production. In mammalian oestrus also serves as a behavioural predictor, indicating whether or not the female is bred close to ovulation due to a climatic variation shows 80% impact on estruses which are failed to be identified in heat-stressed cattle's [9]. The long period of time towards high temperature decline the rate of pregnancy by shortening the oestrus signs and lowering their severity of pregnancy [9]. Furthermore, during the summer, hot weather triggers ovulation without any symptoms of oestrus [9]. The key cause of imbalanced heat detection represents lower intensity of E2 in blood which alter the steroid genic mechanism of heat stress which is disturbed by granulose cell (GCs) [14]. The increased in rectal temperature seemed to have less effective towards binding protein such as insulin like growth factor, level of progesterone in dominant follicle and in E2 [14]. These various responses must be taken into account in terms of exposure period, oestrus cycle, nutritional quality [14] and other environmental factors including wind and humidity. Since lactation in cattle's produce of high heat can result in milk productivity and ovarian function in lactating cattle's differs from dry cattle's and heifers [14].

3.1.2 Follicular development and oocyte quality

The phase of ovarian folliculogenesis takes about 180 days. At birth, the ovary contains primordial follicles containing an oocyte [5]. The accumulations of primordial follicles in the ovarian pool result in rising of follicles enhance the follicular dynamics and at last increase in the pre-ovulatory follicle. The early changes in inhibit, estradiol, and progesterone have been shown to stifle the growth of primordial follicles. The heat stress imbalance the development of intermediate-size (6-9 m) follicles as result in earlier emergence and sometimes decrease or delayed in dairy cattle's [5]. The possibilities of folliculogenesis are reduced due to which considerable amount of plasma inhibin secreted by small size and intermediate follicles [4]. The earlier development of the pre-ovulatory follicle and a rise in the period of dominance [4] are also correlated with a lower rate of conception [4]. However, follicles produce oestrogen; a hormone that causes cattle's to become overheated. Since smaller follicles contain less oestrogen than larger ones, oestrus activity will be reduced. The oocytes and somatic cells that synthesise estradiol are originate from ovarian follicles whereas estradiol has a number of functions, including inducing of oestrus and cause the LH surge. Heat stress disturbs the follicle range and lengthens follicular waves, lowering oocyte output. It also allows for the development of multiple dominant follicles, which explains why cattle's conceiving in summers has more twins. Heat stress also damages the somatic cells (theca and granulosa cells) within the follicles. The variations in folliculogenesis patterns are likely to accompany changes in oocyte quality caused by heat stress. In cattle's exposed to heat stress, follicular dominance is reduced, ensuing in a boost large number of large follicles on the ovary. Over a longer period of time of ovulatory follicle dominance can enhance the higher secretion follicle stimulating hormone (FSH) and decrease the secretion of estradiol-17 hormone and inhibin hormone [13]. The heat stress in dairy cattle's reduced estradiol production and granulosa cell viability, as well as androstenedione production by thecal cells [15]. The conditions of follicles beneath heat stress is forced by the metabolic markers as result disturbs the level of glucose in blood and also imbalances the levels of non-esterified fatty acid (NEFA). In the cool season, the level of glucose in bovine follicular fluid is around 85% of the level of plasma glucose, and in the hot seasons, the follicular glucose level falls substantially with a corresponding drop in blood glucose level [15]. On the other hand, heat stress does not show any impact on balance of Non-esterified fatty acid despite a substantial increase in plasma levels [15]. The studies suggested that at the time of summer, the conditions of follicles is effected by level of nutrition in blood and level of components of biochemical in body of cattle. However, the concentration of oxygen in fluid of follicular does not get disturbed by heat and non-stressed conditions [15].

3.1.3 Corpus luteum

The progesterone, which is required for embryonic growth, is secreted by the Corpus luteum. The lack of luteal deficiency describes the condition of corpus Luteum that does not secrete sufficient amount of progesterone to maintain the pregnancy as result decrease in the fertility of cattle's. When cattle's are subjected to long-term, persistent, seasonal heat stress, their progesterone levels normally drop significantly [16]. These are may be due to a disturbance in the Corpus luteum formation process, low synthesis of under hyperthermia, or imbalanced pre-ovulatory follicles that shape a Corpus luteum with suboptimal purpose [16]. The lack of progesterone has been observed in at the of summer in luteinized granulose cell and theca cell compared to winter. However, a less significant corpus luteum

and lesser progesterone plasma concentration in the later dioestrus result from a smaller diameter and less steroid concentration in pre-ovulatory follicles, which can compromise embryo implantation and development [13].

However, future studies may require a large number of animals to concluded the comparatively weak consequence of acute heat stress during the delayed follicular processes.

4. Effect of heat stress on oocyte function and development competence

The bovine oocytes are extremely compromised of heat stress impact in cattle's [1]. The high temperature affect germinal vesicle (GV) maturation phase of oocyte which shows vulnerability in bovine oocyte. Heat stress affects the developmental competence of germinal vesicles in oocytes of Holstein cattle's, as evidenced a decrease in consequent embryonic development [1] demonstrated that exposing. The Holstein to environmental chamber at heat stressed are 42°C for 10 hours for done successfully. The number of Normal embryos was reduced during oocyte maturation as compared to control (24°c) [1]. The three successive oestrus cycles can be required for an oocyte to recover from heat stress and then recover its oocyte competency in the following for season [9]. Over a long period of time, heat stress can cause major impact on ovarian pool of oocytes. The impact might also disturbed cow fertility and harmed even in the autumn (when there is no ambient thermal stress). As demonstrated by the oocyte competence stability and pregnancy in warmth weather which follow in winter. The hyperthermia of maternal half of concentration of ovarian follicles but does not affect the follicular pool. The oocyte maturation failure can take a variety of forms when exposed to heat stress. It interferes with the biosynthesis of steroid hormones, which are responsible in oocyte maturation regulation mechanisms [9].

5. Different cellular alteration induced by the heat stress in bovine oocyte

The processes by which impact of high temperature affect physiology of oocyte is still not identified but some studies consider that it has been identified that high temperature can damage the cell structures and organelles depending on the basis of temperature [1]. In biological membranes, cytoplasmic and nuclear compartments, heat induce can cause cellular damage in bovine oocytes had been observed. However, evidence suggests that the cytoplasm of the oocyte is more vulnerable towards negative effects of high temperatures as compared to the nucleus [1]. Heat stress may also prevent oocytes from maturing their nuclei, resulting in a decline in polar body charge. Heat stress perhaps, can cause oocytes cytoskeletal structure to be disrupted [9]. In the cumulus oophorus complex however, heat stress controls the appearance of HSP70, the apoptotic gene caspase-3 and other antioxidant superoxide dismutase (SOD1), catalyse (CAT), and Complexin (CPX4). These changes in gene regulation resemble coefficient of coincidence (COC) self-defence mechanisms when they are exposed to heat stress. The 70 kilo Dalton heat shock proteins (HSP70) is a multiple effect factor that keeps the intracellular surroundings are stable and decline cell death [9]. By controlling Caspase-3 and cytochrome c, HSP70 expression can also defend cells from apoptosis [9]. The higher level of HSP70 appears to aid oocyte survival from heat stress by up regulating SPKH1, BCL-2, SOD1, CAT, and CPX4 while down adaptable p53. The changes can be brought by heat shock in bovine oocytes because of lack of nuclear maturation. The oocytes that entered metaphase II phase (MII) following In vitro maturation (IVM) was decreased germinal vesicle (GV) stage [1] and maturation

of oocytes [1]. By growing the amount of metaphase I (MI) in oocytes. Heat shock stopped meiotic progression. On the other hand, in one study it is demonstrated that heat shock at 41°c after 16 to 18 hours it increases the maturation of nuclear and also increasing amount of MII oocytes [1]. The high temperature can cause cellular harm in oocytes of bovine observed in the cellular compartment along with region of cytoplasmic and in nucleus. The cytoskeleton of oocyte gets affected when it comes in contact of high temperature. The Heat-induced shows interruption of microtubules and microfilaments affects chromosome segregation at the time of fertilisation and cell division, as well as the division of cellular structures like cortical granules and mitochondria. On the other hand, heat shock also causes affect to DNA fragmentation and lowers mitochondrial activity in bovine oocytes, implying that the heat-induced mitochondrial apoptotic pathway is activated for combat the low fertility caused by heat stress, a variety of methods have been used. In bovine oocytes, molecules like IGF-I, caspase inhibitors, and the sphingolipid (S1P) have recently been identified as thermoprotective factors. This factors improved oocyte developmental competence and rescued many cellular functions that had impaired by high temperatures. As a result, identifying and characterisation of cellular thermoprotective molecules may be a viable option for reducing the impact of elevated temperature on reproductive function.

6. Alteration in mechanism of male reproduction by heat stress

The Bulls make up short of the herd, and their reproduction is straightly linked to the fertilisation of oocytes. In order to generate health of bull viability, and genetically modified potential ideas in bulls genes. In mammal species have unique physiological regulation known as thermoregulation that protect the reproductive activities from the climatic circumstances. The testicular temperature for spermatogenesis in bulls cannot go above 33–34°c [14] through spermatogonia to elongated spermatids, spermatogenesis mechanism of bovine is multifaceted which take 61 days to complete their process [17]. However, the timing of spermatozoa exposure to heat stress, as well as the time and withdrawal from heat stress. It is difficult to differentiate that at which phase of spermatogenesis the spermatozoa get affected by heat stress. The collecting ejaculate at the time of spermatogenesis phase denotes that heat stress disturbs the spermatogenesis cycle. Inefficient histone replacement by protamine's, resulting in sperm chromatin conformation shifts, is the cause of cell vulnerability at certain particular periods [17]. Hyperthermia has a detrimental impact on testicular function. The adverse impact of heat stress on reproductive tissue as show impact such as lack of germ cell, low morphology, lack of sperm count. According to requirements of specific cell the DNA of sperm nucleus arranged on basis of requirements. The specific type nuclear protein present in spermatozoon's which sets chromatin in more condensed structure between 6 to 20 times as compared to nucleosome-bound DNA, nucleus [17]. During spermatogenesis, heat stress alter the conformation of chromatin of sperm which further imbalance the conformation of DNA methylation which further represent in reorganisation of zygote [17]. The high compaction is due to the substitution of histone-bounded with chromatin with protamine-bounded with chromatin, which is needed for the secure liberation of DNA of sperm of oocyte in the female reproductive tract under the depletion of oxidative stress. Furthermore, the tremendously condensed sperm nucleus inhibits sperm DNA transcription. Protamine deficiency in the sperm nucleus causes DNA damage, which can result in male sub-fertility or infertility [17]. The heat stress increase the level of thiobarbituric acid reactive substance (TBARs) and also level of oxidative marker vice versa decreasing the level of

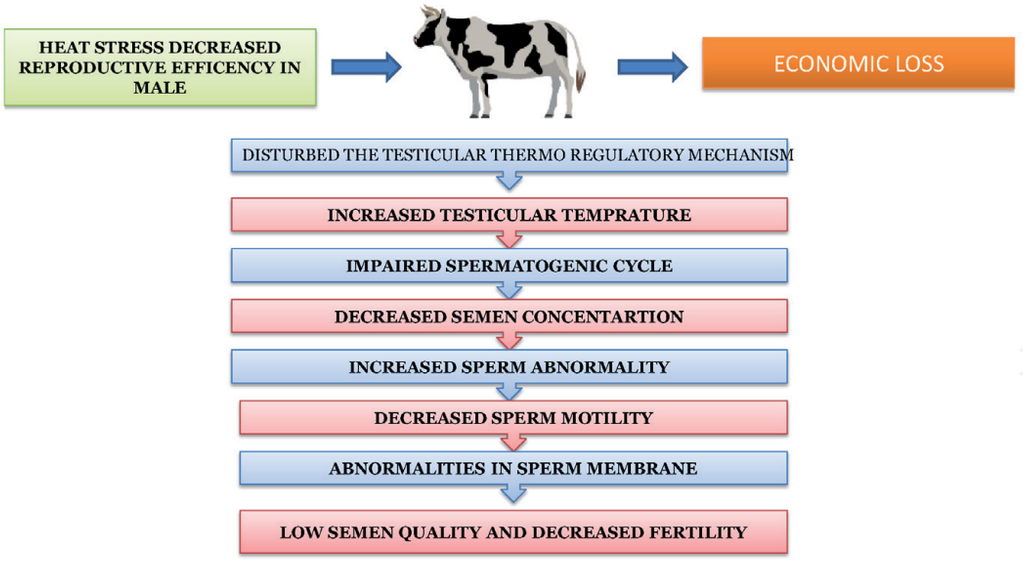

Figure 2.
Impact of heat stress on reproductive efficiency of male reproduction.

glutathione peroxidise (GPx) and enzyme related to antioxidant in seminal plasma of bovines [14]. Bulls plasma luteinizing hormone (LH) levels are reduced by heat stress [14]. The reproductive efficiency of males gets majorly reduced by the impact of heat stress (**Figure 2**).

7. Impact of heatstress on embryo development

The impact of heat stress can cause major impact on maturation of oocyte and development of oocyte competence. The fertilised females under the influence heat stress decline the embryo quality in cattle's [14]. During the fertilisation and implantation of embryo experience towards following phases such as cell proliferation, alteration in patterns of gene expression and cell differentiation. The heat stress significantly inhibits embryo growth 48–72 hours after fertilisation, which correspond to the 8–16 cell stage [14]. After this point, heat stress shows less impact on development and so on cell proliferation [14]. The embryos shows especially vulnerable to maternal heat stress in earliest phase of development as further as development processes the sensitivity towards heat stress. The proportion at which maturation of embryo take place to blastocyst stage after the 8th day of oestrus was decline. When lactating of cattle's were demonstrated on day 1 heat stress impact on oestrus (1-2 cell stage embryo). However, heat stress does not show any impact on blastocyst phase on eighth day [2].

The zygotic genome activation (ZGA) phase occurs at the 4th to 8th cell stage in cattle's [14] is the most vulnerable to heat stress in cow embryos. After the zygotic genome activation heat stress alter the conformation structure of chromatin of embryonic cell [14] potentially disrupting gene expression. Thus, heat stress causes apoptosis in embryonic cells in cattle's [14], and rabbits in addition to maturing oocytes [14].

8. Mitigation strategies for reduction of heat stress

The heat stress impact on cattle's result in considerable financial losses as well as expensive for farmers, but there are following reduction method for heat stress to recoup any of these losses by implementing appropriate heat stress mitigation

strategies. These techniques may be used individually or in combination to improve outcomes by ensuring the best possible atmosphere for farm animals to working farmers are more likely to follow policies that are both cost-effective and incorporate indigenous expertise. Environmental adjustment has traditionally been used to reduce heat load, with the focus on (i) minimising sunlight (ii) increasing air movement [12]. On the other hand wetting of cattle has been subject of research [12] observed that the causes of cooling in day and night, the utilisation of movement of air and water, management of heat load can be measured with help of changes in rectal temperature, respiration time and DMI. There many availabilities of mitigation option for farmers and producers such as (I) oestrus detection (ii) nutritional management (iii) genetic manipulation (iv) antioxidant (v) pharmaceutical treatment (vi) adaptation and acclimation (vii) embryo transfer. In light of antibiotic resistance, nutritional methods are becoming more common. In science also genetics is well known responsible for thermo tolerance capacity, gene identification in cattle's for making them cope up with heat resistance is a new area of research. Individual livestock systems must be assessed for mitigation opportunities to ensure that the mitigation techniques put in place turn into an efficient method for dropping the impact of heat stress in that venture.

8.1 Detection of oestrus and injection at oestrus

Due to the extreme shorter length and lower strength of oestrus, it is difficult to detect. Using a variety of heat detection methods, such as tail-head-paint combined with visual oestrus detection, podometer, pressure enabled patches, and electronic devices put on the tail and head, may boost dairy cow reproductive efficiency. The detection rate for oestrus can be improved by rising the time and several number of visual study [18]. At the time of summer, using an entire male to detect heat at night and early in the morning can improve detection performance [18]. This method suppresses heat stress of oestrus might be hormonal as suggest that indicates heat stress decreases the level of estradiol-17 levels and vice versa increase the secretion level of adrenocorticotrophic which can protect oestrus conduction under the influence of estradiol-induced. The physical lethargy exhibited by heat-stressed cattle's is also likely to reduce oestrus. The other method used for increase fertility in the summer the injection of GnRH are inject during oestrus. According to some studies, when lactating cattle's were vaccinated with GnRH at observed oestrus during the summer, the conception rate increased from 18–29%. The cattle's which are lactating dairy cattle's given GnRH injections at the first indication of status heat during the summer and autumn months had higher conception rates (56%) than untreated (41%) monitors [18]. Heat stress decline the time duration and harshness of oestrus, which leads to an increase in anoestrus and silent ovulation. In order to increase fertility, the timed artificial insemination (TAI) protocol is used for effective oestrus detection and timely insemination. The hormonal therapies have been designed to synchronise ovulation times, allowing for the use of fixed TAI without the need for oestrus detection. The TAI protocol is known as ovary synchronisation content of insemination of gonadotropin-releasing hormone (GnRH) (day 0), prostaglandin F2 (day 7) and GnRH (day 9) and a hormonal therapies, followed by artificial insemination 16-20 hours after the second GnRH hormonal treatment [19]. When coupled with TAI, the ovary synchronisation protocol can successfully synchronise ovulation in cattle's can also enhance conception rate [19]. Under subtropical environmental conditions, the Centre for Inherited Disease Research (CIDR) synchronisation and Pre-synchronisation protocols are also used to increase the rate of conception and rate of pregnancy of Holstein cattle's [19]. This TAI protocol has the potential to minimise reproductive efficiency losses in cattle due to poor oestrus detection in the summer.

8.2 Genetic manipulation

The definite genes that regulate thermoregulation of body and also the cellular responsiveness towards hyperthermia have allelic variants in the mammalian gene pool. Thus, both natural and artificial genetic selection can influence how heat stress affects reproductive function [18]. The coat colour, hair length-controlling genes, and heat shock tolerance in cells are all traits that could be chosen. It may be possibilities to boost up thermal tolerance and also increase fertility in summer by genetically modifying or changing the biochemical properties of the embryo prior to transfer [18]. The recognition of genes plays a vital role in increasing resistivity of cells towards heat shock might led to the transfer of these into heat stress through the breeds sensitivity and transgenic techniques, resulting in cattle's with increasing resistance capacity to defect the heat stress. The selection of breeding animals would need to be given further thought. The performance-based livestock selection and the selection of best breeds based on the phenotypic behaviour with cost effective significant traits like high growth rates, has been practised for decades whereas, farmers can continue to selection of replacement breeds on the basis of individual results cost effective and based on their profits in significant traits in the coming years. According to [13], while genetic improvement initiatives continue to emphasise these economically significant traits, there is a risk that this could lead to a decrease in thermo-tolerance resistivity due to the connection between cattle productivity and also rising metabolism of heat output. The enhancement in metabolic heat output decline the thermo-neutral zone of the animals, which combination with seasonal variation, will make handling cattle in hot weather more difficult.

8.3 Nutritional management

To reduce heat generation through nutrient utilisation inside the animal, choose and feed new, palatable, and high-quality forages as much as possible, feed ingredients with a high digestibility [18]. The animals that are stressed need carbohydrates that can be fermented quickly. The essential ingredients should have buffering capacity such as sodium bicarbonate (NaHCO3), magnesium oxide (MgO) and sodium sesquicarbonate (Na3H (CO3)2) to maintain a natural atmosphere by effectively lower the occurrence of acidosis in the rumen, which is a frequent occurrence in hot weather [18] even if they are not eating as much feed as they need, early lactation cattle's effected to heat stress can go even deeper into increase in negative energy balance. As a result of altered follicle growth and decreased oestrus activity, they are more likely to have poor reproductive efficiency. Any of the symptoms of heat stress can be reduced by feeding high-quality forages and healthy rations. Since potassium is the primary component of sweat gland secretion in cattle, it should also be increased in their diet. As compared to fibre and carbohydrates. The intake of fats in diet is more beneficial because it help to reduction of heat and lowers the metabolic heat. In heat stress conditions, the dry matter easily digestible and also observed decrease in protein-energy ratio. In heat stressed cattle's, feeds on superior quality low-degradable protein has been observed to increase milk productivity as a result, both the amount and type of protein consumed by heat-stressed cattle's and buffaloes are critical. By using supplemental niacin to cattle's diet can also help them cope with heat stress. The Palm oil supplementation increased DMI while lowering heat stress signs [5]. The NEBAL was strengthened by feeding conjugated linoleic acids during heat stress, but milk fat was depleted at the same time. Lipoic acid has been shown to have antioxidant and energetic-metabolism-promoting properties [5]. The Exogenous antioxidant nutrient supplementation such a vitamin C, A, and E, as well as trace minerals including zinc (Zn), manganese (Mn), copper

(Cu), selenium (Se), chromium (Cr) and others, may be used to decline the adverse effect of environment [5]. The elements such as B-complex vitamins, ascorbic acid, tocopherol, rumen-protected by Niacin and Nicotinic acid [5] have all been found to be helpful. Thiazolidinedione's (TZDs) can boost HSP development [5] increase glucose utilisation [5] and boost energetic metabolism, making them a viable heat stress strategy. Dietary betaine, like TZDs, might be a better alternative in heat stressed lactating cattle's [5]. In heat stressed lactating cattle's, chromium supplementation has been shown to increase energy metabolism and performance [19]. There is evidence of the development of reactive oxygen species (ROS) by embryos growing at high body temperatures is one of the causes of embryonic death in heat stressed animals [13]. Efforts to increase the fertility of lactating cattle's subjected to heat stress by administering antioxidants have had mixed results [13].

8.4 Embryo transfer

The heat stress shows adverse impact on embryos greatest sensitivity, however, occurs during the early stages of embryonic development which leads to reduced pregnancy.. The embryos develop some tolerance capacity towards heat stress at time of embryonic development (morula or blastocyst stage). As a result, using embryo transfer of frozen embryos harvested from non-heat stressed cattle's, it might be potential to raise to pregnancy rates in heat stress effected cattle. There are multiple number of adverse effects of heat stress on the pregnancy of cattle (**Figure 3**). Many recent studies have shown that embryo transfer can be used to avoid embryonic death within the first seven days of development, when the embryos are more vulnerable towards heat stress. At the time of the summer, the technique has the potential to increase pregnancy rates dramatically. Furthermore, studies advances in improving embryo heat stress resistance through genotype modification and the accumulation of endurance factors like insulin growth factor-1, which help provide a protection to cells from following types of stresses, can boost pregnancy rates with embryo transfer even more. However, embryos are more responsible towards heat stress early pregnancy and also cooling the inadequate number of instance. At the time of the sensitivity of embryo at peak under heat stress shows enhancement of the pregnancy ratios moderately. As example of the rate of pregnancy towards artificial insemination for cattle's that were chilled for eight days after acceptance of prostaglandin F, (PGF2) was 16% as compared to 6% for control [20]. The limited cooling is not considerably useful for rate of increasing rate of pregnancy due to heat stress. The cooling was started on heat stressed follicles function and stopped at later at the time of pregnancy. The following conditions have hindered widespread trade implementation of embryo transfer for decline the rate of heat stress [21]:

1. The dairy heifers act as donors can origin their initial parturition to be overdue, resulting in lower productivity.

2. The embryo quality and ambient temperature have a negative relationship, the form up good to the superior qualities of embryos viabilities when the most needed.

3. The improvement of embryos is a time-consuming and costly procedure.

4. When it comes to frozen embryos, only developed in vivo have high percentage of pregnant cattle when compared to artificial insemination.

5. The in-vitro production of embryo cost is lesser than compared to in vivo embryos production. However, improved embryos are produced only with help of in-vitro technique. They are transferred fresh in cattle's.

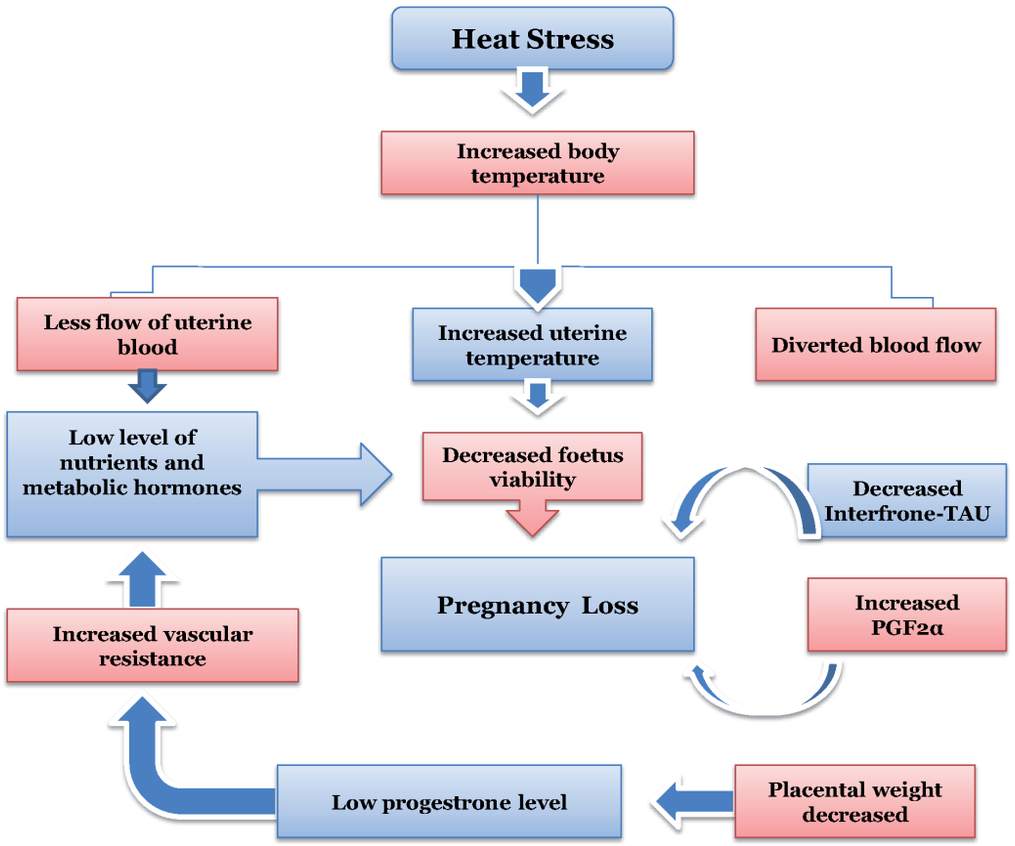

Figure 3.
Impact of heat stress on pregnancy of cattle.

8.5 Environmental strategies

In general, cattle's environmental strategies are a growing field in bovine science that is receiving further become aware of climate change. It aims to create appropriate micro-climate for most favourable production by minimising negative environmental effects on cattle's production organisation. The primary methods of changing the atmosphere can be divided into three categories [22]:

i. Creating a shady environment.

ii. Methods for evaporative cooling.

iii. Fogging systems use fine water droplets that quickly evaporate and disperse into the air stream, cooling the surrounding atmosphere.

8.6 Adaptation and acclimation

The all cattle's have capacities to get adapted in climatic surroundings which very essential to remember. Cattles may change their pattern of behaviour, their physiological, and order of morphological characteristics, in reaction to the temperature or climatic changes [12]. All species have endurance type of strategies to decline the heat stress over the entire body. Adaptation and acclimation are two coping strategies that animals have created. Although the terms adaptation and acclimation have completely dissimilarities in meanings, they can also be interchangeably [12].

8.6.1 Acclimation

The animals phenotypic reaction to an individual stressor in the environment is known as acclimation [23]. However, it is uncommon for only one environmental variable to adjust over time in natural environments. Acclimatisation is the adaptation of an animal to a variety of stressors in its natural environment [12] as a result, acclimation and acclimatisation are not termed as an evolutionary adaptation or natural selection, which are characterised as changes that allow for preferred selection of animal on basis of phenotype and also based on their genetic component that is conceded down to the next generation. If environmental stressors are eliminated, the altered phenotype of acclimated animals will return to normal. On the other hand, Animals are genetically adapted to their climate on the basis of their requirements. In other words, it's a homeostatic system triggered by the endocrine system that cause impact upon the cellular activities, metabolic activities and further alteration in systemic, which permits the animals show adaptability towards heat stress and also overcome to it [12].

At consequence, acclimation can be thought of as a mechanism that occurs over the course of a lifetime, in which constant exposure to a specific stressor, such as extreme hot weather, causes biological changes, improving the fitness of the individual animal to live in climatic conditions [12]. There are three functional distinctions between acclimatory and homeostatic or "reflex" responses, according to Collier and Zimbelman [23].

1. It takes a lot longer for the response to happen (months and years).

2. In the acclimation initial pathway from the central nervous system to the effector cell, acclamatory responses are usually linked to hormones.

3. The acclimatory effect alters an effector cells and organs capacity to respond to environmental change.

As previously mentionesd, these acclamatory responses are typical of homeorhesis processes, and the net result is to synchronise metabolism in order to achieve a recent physiological state. As a result, the metabolism of the seasonally adapted animal differ in the winter from the summer. The Heat stress adaptation is thus, a homeorhesis process involving alter the patterns of hormonal signals that manipulate target tissue sensitivity to environmental stimulus. The improved genetic quality selection of heat stress tolerant genotypes would result from a better understanding of this mechanism.

8.6.2 Adaptation

Adaptation is described as a biological change that occurs over generations as a result, of continuous stressor exposure and favours genetic assortment in an inhabitants to support species endurance [12]. For example Bos- indicus of tropical climates, earlier evolved in tropical climates with elevated temperature along with humidity as a result, they have a range of genetic variations that promote thermo-tolerance [12]. Thus, the ability of Bos-indicus breeds to survive in tropical environments on the basis of their requirements and adaptabilities towards it. They have evolved over generations. The climate conditions has the capability to oblige 'natural' selection for heat tolerant cattle in grazing breeding herds despite

of assortment forces obligatory on the inhabitants. The generations are developing succeeding capacities to adapt them in warmth environment. The progenies developing heat resistivity potential with climatic circumstances. However, it is difficult to reach to conclusion in the case of bovine because of long interval in the productivity. When cattle come in contact of acclimation and adaptation the adapt the degree of resilience. The acclimation and adaptation, when covmbined with the use of mitigation options, which has the ability to improve cattle performance and productivity during the high heat stress.

9. Conclusion

Heat stress as an effect of climate change, would inevitably is the reason heat stress in all farm animals, affecting their reproductive abilities. The impacts of heat stress on both females and males were explored in detailed in this chapter as well as male reproductive. This chapter also discussed mitigation measures that should be considered in order to avoid financial losses caused by environmental pressures on bovine reproductivity. Fortunately, there are managed methods for mitigating the severity of heat stress on bovine reproductivtiy. The involvement of cattle's in climate controlled environments, using the techniques of artificial insemination protocols that conquer the detection of poor oestrus, embryo transfer method and implementation of embryo to avoid damage in oocyte and earlier fertilisation. The heat stress can also causes the embryo to develop abnormally. To aid ruminants cope with harmful conditions, management options such as strategic technique are being use of providing the shades along with wind covering, attachment of sprinklers and providing ventilation at the time period of extreme heat stress should be considered. The strategy of diet intake, in addition to these steps, can be advantageous for ruminants facing environmental challenges also; there are possibilities for manipulating animal genetics to produce a more heat-resistant animal. The animals have genes for body temperature control and cellular tolerance to high temperatures. The temperature, as well as the recognition and genes assimilation in to breeds which are heat-receptive that does not decreases the reproduction, will be a great accomplishment. On the other hand all animals can adapt to their thermal surroundings through adaptation and acclimation. In response to temperature, animals can alter their patterns of behavioural, alter the physiological, and changes the pattern of morphological characteristics, or a combination of these towards heat stress.

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