



ISSN: 2456-2912

NAAS Rating (2026): 4.61

VET 2026; SP-11(8): 24-32

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www.veterinarypaper.com

Received: 20-05-2026

Accepted: 25-06-2026

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affiliations are given below, after
the references.

Early embryonic mortality in bovine with emphasis on heat stress and its amelioration

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DOI: <https://www.doi.org/10.22271/veterinary.2026.v11.i8Sa.3677>

Abstract

Early embryonic mortality in bovines represents a major constraint to reproductive efficiency and economic sustainability in dairy and beef production systems. Despite high fertilization rates (up to 90%), only 40-50% of inseminated cows successfully calve, largely because of embryonic losses during the first few weeks of gestation. The causes of EEM are multifactorial, including genetic and chromosomal defects, endocrine and luteal insufficiency, nutritional imbalances, infectious and immunological factors, and most prominently environmental stress, particularly heat stress. Heat stress disrupts the hypothalamo-pituitary-gonadal and hypothalamo-pituitary-adrenal axes, alters steroidogenesis, impairs gametogenesis, and induces oxidative and immunological imbalances, thereby reducing embryo viability. Biomarkers such as progesterone (P4), interferon-tau, ISG15, and preimplantation factor have been explored for early diagnosis. Ameliorative strategies include managemental modifications, hormonal therapies (GnRH, hCG, P4, melatonin, kisspeptin), recombinant IFN- τ administration, and immunomodulatory or antioxidant interventions. A clearer mechanistic understanding of embryonic loss under thermal stress is essential to design targeted reproductive management and enhance conception rates in cattle and buffaloes.

Keywords: Early embryonic mortality, heat stress, bovine pregnancy, progesterone, interferon-tau, oxidative stress, embryo implantation, reproductive biotechnology

Introduction

The dairy sector of India represents the single largest agricultural commodity in the country, comprising 193.46 million cattle and contributing nearly 5.5% to the national GDP. India is the world's largest milk producer, with a recorded output of 230.6 million tonnes during 2022-23. Sustaining this scale of output has relied on intensive genetic selection for high milk yield, and this selection pressure has progressively been accompanied by a decline in reproductive efficiency (Bello *et al.*, 2012)^[4]. In lactating cows, the heightened metabolic demand further downregulates expression of the *DKK1* gene a regulator of neural development, glucose homeostasis, and embryonic competence (Denicol *et al.*, 2014)^[15]. These reproductive failures manifest most visibly as embryonic mortality, a major source of economic loss to livestock farmers. In cattle, the fertilization rate is approximately 90%, whereas the average calving rate is only about 55%, yielding an overall embryonic/fetal mortality of nearly 35%, with 70-80% of these losses occurring between Days 8 and 16 post-insemination (Diskin *et al.*, 2006)^[17]. Early embryonic loss is consequently more pronounced in high-yielding dairy cattle and remains a primary constraint to milk-production efficiency. Although late embryonic mortality accounts for less than 10% of pregnancy loss, heat stress alone may contribute an additional 15-20% absolute increase in embryonic mortality, making HS a critical determinant of fertility in tropical and subtropical environments.

2. Early embryonic development and pregnancy recognition

In cattle, Day 0 corresponds to estrus, with ovulation occurring approximately 18 h later.

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Ovulation induces luteinization of granulosa and theca cells to form the corpus luteum, the primary source of progesterone (P4), an essential hormone for early embryo survival. P4 synthesis draws on cholesterol from three sources: *de novo* synthesis, hydrolysis of cholesterol esters, and uptake of circulating lipoproteins. Cholesterol is transported intracellularly by sterol carrier protein-2, and its transfer to the inner mitochondrial membrane the rate-limiting step in steroidogenesis is mediated by steroidogenic acute regulatory protein. Cholesterol is then converted to pregnenolone by CYP11A1 and subsequently to P4 by 3 β -hydroxysteroid dehydrogenase (3 β -HSD) (Seedorf *et al.*, 2000) [59]. Around Days 16-17 of pregnancy, maternal recognition of pregnancy is established through IFN- τ secreted by the elongating conceptus. IFN- τ suppresses endometrial estrogen receptor expression, preventing oxytocin receptor upregulation and prostaglandin F $_{2\alpha}$ (PGF $_{2\alpha}$) release, thereby protecting the CL from luteolysis and maintaining P4 secretion. IFN- τ also induces interferon-stimulated genes and downregulates endothelin-1, transforming growth factor- β 1, serpin-1, and thrombospondins 1 and 2, collectively supporting luteal maintenance and pregnancy establishment (Bott *et al.*, 2010) [7].

3. Causes of early embryonic mortality

3.1 Genetic and Chromosomal Factors

Genetic abnormalities account for approximately 10% of embryonic losses during the first two weeks of gestation and may contribute up to 20% of total embryonic and fetal mortality (Vanroose *et al.*, 2000; Diskin & Morris, 2008; Figure 1) [16, 68]. Deficiency of uridine monophosphate synthase results in embryonic death between Days 40 and 50 of gestation (Robinson *et al.*, 1984), and gross chromosomal abnormalities account for about 5% of embryonic mortality (Peters, 1996) [48]. Robertsonian translocation (1/29), which fuses chromosomes 1 and 29 to yield a karyotype of 59 chromosomes (Gustavsson, 1979) [19], is associated with embryonic death during the first or second week of gestation. Dysregulated expression of oxidative stress-related genes upregulation of phospholipase A2 and glutathione peroxidase

along with downregulation of annexin A1 and lactoferrin disrupts oxidative balance and meiotic competence, predisposing embryos to early loss (Puglisi *et al.*, 2013) [51].

3.2 Endocrine factors

Endocrine dysfunction is a leading cause of EEM. Inadequate preovulatory follicular development produces luteal insufficiency via delayed CL development, premature luteolysis, or functional luteal defects, leading to inadequate P4 secretion required for pregnancy maintenance. Suboptimal P4 during the first week after ovulation impairs uterine receptivity by altering MUC1 expression and compromises implantation. Conversely, suprabasal P4 (>1 ng/ml) before ovulation suppresses LH pulsatility, delays the LH surge and ovulation, and promotes follicular overmaturation and embryo-uterine asynchrony, producing aged gametes, repeat breeding, and EEM (Kimura *et al.*, 1987) [31]. High-yielding dairy cows also exhibit reduced peripheral P4 because of increased hepatic metabolism, predisposing them to embryonic loss. Luteal dysfunction contributes to conception failure in approximately 67% of buffaloes and 65% of crossbred cattle (Jisna, 2024; Nivetha, 2024) [28, 45]. Cows with estrous P4 concentrations >1 ng/ml show a lower ovulation rate than those with <0.5 ng/ml (Burns *et al.*, 2008) [8], while prolonged CL lifespan has been associated with EEM, abortion, and fetal mummification (Unuane *et al.*, 2011) [67].

3.3 Nutritional factors

Nutritional imbalance contributes to EEM through endocrine dysfunction, oxidative stress, and impaired metabolic homeostasis. Affected cows exhibit reduced P4 and estradiol-17 β concentrations together with altered protein, urea, creatinine, cholesterol, and liver-enzyme profiles and elevated endogenous intoxication indices, reflecting hepatic and renal dysfunction. Embryonic loss is further associated with deficiencies of zinc, copper, manganese, selenium, magnesium, iodine, and vitamins E and C, and with reduced glutathione peroxidase and catalase activity each undermining antioxidant defence, promoting lipid peroxidation, and amplifying oxidative stress (Nezhdanov *et al.*, 2017) [42].

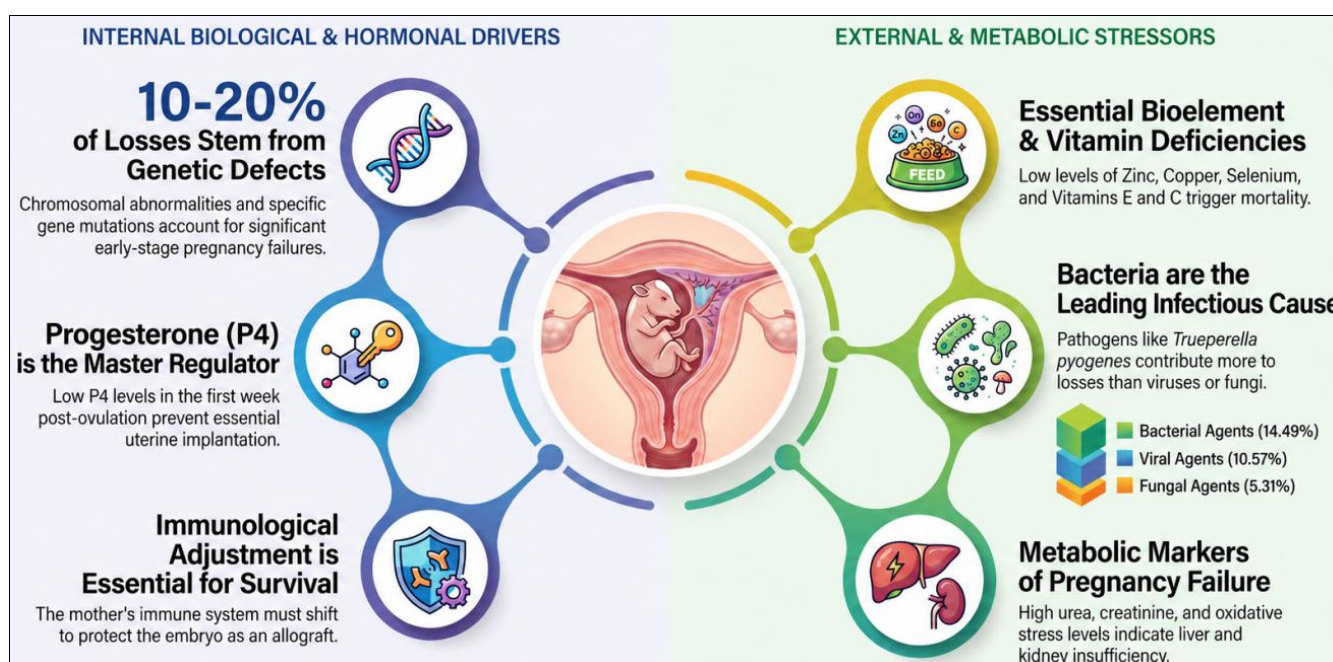


Fig 1: Schematic representation of Intrinsic and Extrinsic Factors Associated with Early Embryonic Mortality in Cattle

3.4 Infectious factors

Infectious agents contribute substantially to EEM; bacterial infections (14.49%) are more prevalent than viral (10.57%) and fungal (5.31%) infections in abortus and stillbirth submissions. Among viral pathogens, bovine herpesvirus-1 and bovine viral diarrhoea virus are most frequently reported, while common bacterial pathogens include *Trueperella pyogenes*, *Bacillus* spp., *Listeria* spp., *Escherichia coli*, and *Leptospira interrogans* (Givens & Marley, 2008; Kumar *et al.*, 2011; Gates *et al.*, 2013) ^[21, 33].

3.5 immunological factors

Successful embryo survival requires maternal immune

tolerance toward the semi-allogeneic conceptus. During early pregnancy, a shift from Th1- to Th2-dominant immunity, together with endometrial and placental expression of insulin-like growth factor-1, IGF-2, and IGF-binding proteins, facilitates nutrient partitioning and conceptus development (Robinson *et al.*, 2000; Sferruzzi-Perri *et al.*, 2007) ^[56, 60]. IFN- τ secreted by the conceptus suppresses lymphocyte proliferation, protecting the embryo from maternal immune rejection (Skopets *et al.*, 1992) ^[61]. Elevated IGFBP-1 and IGFBP-4 concentrations, together with reduced IL-6, IL-13, and IFN- τ levels, have been associated with early embryonic and fetal death (Sobhy *et al.*, 2021) ^[62].

3.6 Schematic Overview of Pathogens Affecting Reproduction

Table 1: Major Infectious Pathogens, Their Routes of Transmission, Reproductive Effects, and Embryonic Mortality in Cattle

Pathogen	Route	Reproductive Effects	Embryonic Mortality
<i>Leptospira</i> spp., <i>L. interrogans</i> , <i>L. borgpetersenii</i>	Haematogenous	Abortion (3rd trimester); stillbirth; infertility	+++
<i>Campylobacter fetus</i> venerealis	Vertical / horizontal	Abortion (4-7 months); infected cow clears infection within 3-6 months	+
<i>Ureaplasma diversum</i> , <i>Mycoplasma</i> spp.	Venereal	Abortion; infertility; endometritis	+
Bovine herpesvirus-1 (infectious bovine rhinotracheitis)	Haematogenous / venereal	Abortion (4-8 months); acts as source of infection	+++
<i>Pestivirus</i>	Haematogenous	Subfertility; acts as source of infection	++
<i>Tritrichomonas foetus</i>	Venereal	Abortion in 1st half of pregnancy; infertility	+
<i>Histophilus somni</i>	Haematogenous	Binds to zona pellucida; infertility	+

(Compiled from Givens & Marley, 2008; Kumar *et al.*, 2011; Gates *et al.*, 2013) ^[21, 33]

4. Predisposing factors for eem

Species. EEM is generally higher in buffaloes than in cattle because of greater susceptibility to heat stress and associated endocrine disturbances (Kumbhar *et al.*, 2021) ^[35].

Breed. High-producing dairy breeds, particularly Holstein cows, exhibit a higher incidence of EEM than dual-purpose breeds such as Simmental and Brown Swiss. Crossbred cattle likewise show a higher prevalence of repeat breeding (17.57%) than indigenous breeds (8.64%) (Verma *et al.*, 2018) ^[69].

Milk yield. High milk production increases metabolic demand, predisposing dairy cows and buffaloes to negative energy balance and a greater risk of early embryonic loss (Kaneda *et al.*, 2024) ^[29].

Parity. Embryonic mortality generally increases with advancing parity. Swedish Red and Swedish Holstein cows in their third or later parity exhibit greater fetal loss (>61 days after AI) than younger cows (Nyman *et al.*, 2018) ^[46]. However, primiparous cows may also experience higher EEM because of reproductive immaturity and incomplete metabolic adaptation.

Age. Maximum conception rates are achieved in heifers bred at 15-16 months of age, whereas first breeding after 26 months reduces pregnancy per insemination by approximately 13% (Martin *et al.*, 2015) ^[38]. Higher EEM has also been reported in cows aged 2.0-2.4 years, while cows aged 3.5-4 years exhibit the lowest losses (Puklová *et al.*, 2014).

Body Condition Score and energy balance. Cows with suboptimal BCS (<2.5 or 2.75-3.5) experience greater

embryonic loss than those with optimal BCS (>3.75) (Senosy *et al.*, 2012) ^[65]. During early lactation, peak milk yield precedes peak feed intake by several weeks, producing NEB, mobilization of body reserves, and approximately a one-unit loss in BCS each compounding the risk of EEM (Parmar *et al.*, 2016) ^[47].

5. Early detection of embryonic mortality

5.1 Preimplantation factor

- Secreted by trophoblast cells and placental tissue (Barnea, 2007; Stamatkin *et al.*, 2011) ^[3, 64].
- Peptide sequence: MVRKPGSANKPSDD.
- Detection method: PIF ELISA.
- Enhances endometrial receptivity, promotes trophoblast invasion, and modulates immunity.
- Detectable in serum at 91.3% by Day 10 and ~100% by Day 20 of pregnancy.

5.2 Progesterone (P4) assay in blood

- Low or declining P4 between Days 10 and 20 post-inseminations is associated with higher embryonic loss in buffalo and cattle (Campanile *et al.*, 2005) ^[50].
- On Day 21, P4 <2 ng/ml indicates non-pregnancy and implantation failure (Roa *et al.*, 1998) ^[54].
- Cutoff serum P4 concentrations of >0.95 ng/mL on Day 0, <1.86 ng/mL on Day 5, and <1.98 ng/mL on Day 18 of the estrous cycle predict luteal phase deficiency in buffaloes owing to suprabasal, delayed rise, and early fall of P4, respectively.
- Milk P4 >5 ng/mL on Days 21-24 is consistent with pregnancy (Humblot, 2001) ^[25].

5.3 Interferon-stimulated genes

- Detected in blood on Days 18-20.

- Expression correlates with MX2 and ISG15 mRNA levels in peripheral blood leucocytes.
- Particularly accurate for nulliparous cows.

5.4 IFN- τ in cervical fluid

- Detected by ELISA with a limit of detection of ~54 pg/mL.

- Optimal detection window: Days 16-19 after AI.
- Detection of IFN- τ on Day 17 followed by ultrasonographic pregnancy loss on Day 32 identifies loss attributable to EEM.
- Sensitivity ~80%; specificity ~75% (Bishop *et al.*, 2025)^[6].

5.5 Comparative Profile of Pregnancy-Associated Biomarkers

Table 2: Biomarkers, Their Sources, Detection Time, Diagnostic Methods, and Applications for Early Pregnancy Detection in Cattle

Biomarker	Source	Matrix	Earliest detection	Method	Notes	References
Early pregnancy factor	Preimplantation trophoblast	Milk, serum	6-48 h post-fertilization Accurate result obtained when samples are collected at 7 to 8 days	Rosette inhibition test	Limited utility because of high losses during first 15 days	Threlfall & Bilderbeck, 1999 ^[75]
Pregnancy-associated glycoprotein	Binucleated trophoblastic ectodermal cells	Milk, serum	>24 days	ELISA	Half-life 3-4 days	Reimers <i>et al.</i> , 1985 ^[71] ; Zoli <i>et al.</i> , 1992b ^[72] ; Sasser & Ruder, 1987 ^[73]
Pregnancy-specific protein B	Fetal membrane (placenta)	Maternal blood	>28 days (90% accuracy on day 30 using RIA)	RIA	Half-life 7-8 days	Humblot <i>et al.</i> , 1988 ^[26, 74]

6. Heat stress

HS is one of the most important environmental factors limiting reproductive efficiency in dairy cattle, particularly under tropical and subtropical conditions. Elevated ambient temperatures reduce feed intake, milk yield, body weight gain, and fertility, producing substantial economic losses. Numerous studies have documented a marked decline in conception and pregnancy rates following AI during periods of HS (García-Isperto *et al.*, 2006; De Rensis *et al.*, 2017)^[13, 20]. The resulting increase in maternal body temperature disrupts ovarian function, uterine receptivity, fertilization, and embryonic development, making HS a major cause of EEM (Berman, 1985; De Rensis & Scaramuzzi, 2003)^[5, 12].

6.1 Neuroendocrine effects of HS

The physiological response to HS is mediated principally through activation of the HPA axis and the sympathetic-adrenal-medullary axis. HS stimulates hypothalamic release of corticotropin-releasing hormone, leading to secretion of adrenocorticotrophic hormone from the anterior pituitary and consequent cortisol release from the adrenal cortex. Although cortisol supports metabolic adaptation by promoting gluconeogenesis and energy mobilization (Ayoob *et al.*, 2018)^[2], sustained elevation suppresses reproductive function by inhibiting KNDy neurons and consequently reducing GnRH, LH, and FSH secretion. This endocrine disruption impairs follicular development, steroidogenesis, ovulation, oocyte competence, and fertilization, collectively increasing EEM (Khan *et al.*, 2020)^[30]. HS also reduces inhibin secretion and alters follicular dynamics, further compromising fertility. Elevated cortisol concentrations in plasma, milk, and faecal 11,17-dioxoandrostane have been proposed as reliable biomarkers of HS in cattle (Möstl *et al.*, 2002)^[41].

6.2 Immunological effects of hs

HS activates the HPA axis, increasing cortisol secretion, which in turn modulates the function of neutrophils, macrophages, monocytes, T cells, and B cells. Prolonged elevation of cortisol suppresses innate and adaptive immune responses, increasing susceptibility to inflammation and

compromising the uterine environment required for successful embryo survival.

Neutrophils: Cortisol inhibits neutrophil activation by suppressing MAPK signalling and reducing expression of adhesion molecules such as L-selectin, impairing neutrophil migration and phagocytic function. HS additionally activates stress-responsive JNK and p38 MAPK pathways, promoting apoptosis and reducing embryonic viability.

Macrophages: HS shifts macrophage polarization by suppressing pro-inflammatory M1 responses and favouring the anti-inflammatory M2 phenotype through modulation of STAT signalling, weakening innate immune defences (Tugal *et al.*, 2013)^[66]. Disruption of NF- κ B signalling further impairs cell survival and embryonic development under thermal stress.

Lymphocytes: HS and cortisol suppress T-cell activation by inhibiting NF- κ B and MAPK signalling, reducing lymphocyte proliferation and adaptive immune function. Although the magnitude of lymphocyte changes varies among breeds, thermotolerant indigenous cattle generally exhibit less immune suppression than high-producing exotic breeds (Elvinger *et al.*, 1991)^[19].

6.3 Transcriptomic response to HS in cattle embryos

HS induces profound transcriptomic and epigenetic alterations in bovine embryos, initially as adaptive responses but ultimately as stress persists impairing embryonic development. Thermal stress activates chromatin remodelling through histone acetylation and demethylation, leading to activation of stress-responsive transcription factors and increased expression of heat-shock protein genes, particularly HSP70. During early embryogenesis, development depends on maternally derived mRNA and protein until embryonic genome activation at the 8-16-cell stage. This transition involves extensive DNA methylation reprogramming mediated by DNA methyltransferases, rendering embryos highly susceptible to heat-induced epigenetic disruption

(Jiang *et al.*, 2014; Dobbs *et al.*, 2013)^[18, 27]. Consequently, HS alters gene expression, compromises embryo quality, and reduces developmental competence (de Barros & Paula-Lopes, 2018)^[14].

Heat stress enhances acetylation of the HSP70 promoter, increasing HSP70 expression, whereas embryos derived from heat-stressed oocytes accumulate the repressive histone mark H3K9me3 and heterochromatin protein-1, producing chromatin condensation and apoptosis (Kisliouk *et al.*, 2017; Camargo *et al.*, 2019b)^[9, 10, 32]. Although HSP70 protects embryos by preventing protein misfolding and inhibiting apoptosis, prolonged or severe HS overwhelms these protective mechanisms (Mosser *et al.*, 2000). Other thermotolerance-associated genes, including HSPA14 and other HSP family members, exhibit stage-dependent responses to heat exposure (Sakatani *et al.*, 2012; Hassan *et al.*, 2019)^[23, 58].

Heat stress also disrupts expression of genes essential for embryonic development, including *DNMT1*, *CDH1*, and *Cx43*, as well as stress-responsive genes (*HSPB11*, *HSPBP1*, *AKR7A2*, *GSTA4*) and components of the ubiquitin-proteasome pathway, reflecting enhanced protein-quality control under thermal stress (Sakatani *et al.*, 2013)^[57]. Reduced expression of developmental genes such as *AQP3* and *ATP1A1* further impairs blastocyst expansion and developmental competence, contributing to increased apoptosis and EEM (Camargo *et al.*, 2019a)^[9, 10]. Overall, HS triggers cytoprotective transcriptomic responses, yet persistent thermal stress disrupts epigenetic programming and developmental gene expression, ultimately compromising embryo viability and pregnancy establishment.

7. Ameliorative strategies for heat stress-associated EEM

7.1 Managemental strategies

- HS should first be minimized through environmental management provision of shade, improved ventilation, evaporative cooling systems (fans, sprinklers, misters), and continuous access to clean drinking water.
- Nutritional management feeding during cooler hours and supplementation with antioxidants and electrolytes reduces metabolic heat load and oxidative stress.
- Precision livestock farming technologies support early detection and mitigation of HS by continuously monitoring physiological and behavioural responses through automated sensing:
- **RFID** for individual animal identification.
- **RTLS** for monitoring movement and activity.
- **GPS** for tracking grazing animals.
- **VF** for controlled grazing and movement management.
- **RR** as an early indicator of thermal stress.
- **CBT** for assessment of internal thermal load.
- **ST** measured by Infrared Thermography.
- **TT, VT, RT** as body temperature indicators.
- **IoT** integration of sensors with automated cooling systems for real-time HS management.

7.2 Hormonal therapy

Gonadotropin-releasing hormone

- 250 µg GnRH at the time of AI increased pregnancy rate by 22.5% in repeat-breeder cattle (Morgan and lean, 1993)^[40].
- 200 µg GnRH on Day 20 post-AI reduced embryonic mortality in buffaloes (Abulaiti *et al.*, 2023)^[1].

- GnRH enhances luteal function and increases P4 secretion, improving embryo survival.

Human chorionic gonadotropin (hCG)

- hCG on Day 6 post-AI increased pregnancy rate to 67.5% (Nishigai *et al.*, 2002)^[43].
- 1000 IU hCG post-AI induces luteinization of the dominant follicle and accessory CL formation in cattle (Cooper *et al.*, 1991)^[11].

Progesterone (P4): P4 supplementation during early pregnancy supports embryo development, although timing is critical. PRID insertion on Day 5 post-AI reduced pregnancy rate and increased EEM because endogenous CL output declined after device removal (Villarroel *et al.*, 2004)^[70].

Kisspeptin: Intravenous administration of Kisspeptin-10 (10 µg/kg) at AI and again on Day 5 post-AI achieved a 50% calving rate in crossbred cattle. Kisspeptin improved reproductive outcomes but did not significantly induce accessory CL formation (Nivetha, 2024 unpublished research)^[45]

Melatonin: Sustained-release melatonin is effective in summer-stressed postpartum anoestrus buffaloes. Melatonin administered at 18 mg/50 kg body weight subcutaneously, followed by a second dose 5 days after AI, improves reproductive performance by reducing oxidative stress and supporting luteal function (Kumar *et al.*, 2016)^[36]

7.3 Other Therapeutic Approaches

Recombinant IFN-τ

- Intrauterine administration of recombinant ovine IFN-τ (100 µg) or recombinant bovine IFN-τ (200 µg) twice daily during Days 14-24 prolongs CL lifespan, enhances luteal lymphangiogenesis, extends the interoestrous interval, suppresses oxytocin receptor-mediated uterine PGF_{2α} release, and improves MRP (Meyer *et al.*, 1995; Spencer & Bazer, 1996; Nitta *et al.*, 2011)^[39, 44, 63].
- Unlike interferon-α, recombinant IFN-τ does not induce hyperthermia (Plante *et al.*, 1991)^[49].

Cyclooxygenase Inhibitors

- Meloxicam (0.5 mg/kg IM) on Days 13-15 post-AI reduced prostaglandin synthesis and increased conception rate by approximately 20% (Rajkumar *et al.*, 2010)^[53].

Immunomodulators

- *Escherichia coli* LPS (100 µg, intrauterine) stimulates the uterine immune response (Dash *et al.*, 2016).
- Carvacrol (0.1%) + Lugol's iodine (0.1%), 50 mL intrauterine, improves uterine health (Lehimcioglu *et al.*, 2019)^[37].
- Neem-oil extract (30-40 mL, intrauterine) yielded a 71.4% conception rate (Kumar *et al.*, 2013)^[34].

7.4 Treatment Strategies for Infectious Causes of EEM

- **Intrauterine antibiotic therapy:** oxytetracycline; levofloxacin + ornidazole/metronidazole suspension.
- **Hormonal therapy:** cloprostenol (500 µg IM); dinoprost (25 mg IM).
- **Combined hormonal + antibiotic therapy:** oxytetracycline (1500 mg intrauterine) + cloprostenol (500 µg IM).

- **Pre- and post-AI preventive therapy:** penicillin G (10 lakh IU in 20-30 mL distilled water), administered 5-6 h before and 3-6 h after AI; streptomycin-penicillin (1 g in 20-30 mL distilled water), on the same schedule.
- **Systemic antibiotic therapy** on Day 4 post-AI (embryo entry into uterus) and Days 9-10 post-AI (zona-pellucida hatching stage) reduces uterine infection and improves embryo survival in high-risk animals.

8. Conclusion

EEM remains one of the major causes of reproductive inefficiency and economic loss in cattle and buffaloes, with most losses occurring during the preimplantation period. Its etiology is multifactorial, involving genetic, endocrine, nutritional, infectious, immunological, and environmental factors among which heat stress has emerged as one of the most important contributors under tropical and subtropical conditions. HS disrupts endocrine homeostasis, impairs oocyte competence, alters maternal immune responses, induces oxidative stress, and causes transcriptomic and epigenetic alterations that collectively compromise embryonic development, MRP, and embryo survival. Recent advances in molecular biomarkers, including IFN- τ , ISGs, PIF, and pregnancy-associated proteins, have improved the prospects for early diagnosis of embryonic loss. Likewise, integrated management strategies environmental cooling, precision livestock technologies, nutritional supplementation, hormonal interventions, antioxidants, and emerging molecular therapies offer promising approaches to mitigate HS-induced EEM. Future research should focus on identifying robust biomarkers of embryo competence, elucidating the molecular mechanisms underlying thermotolerance, and developing precision reproductive-management strategies to enhance fertility and sustain bovine production under changing climatic conditions.

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How to Cite This Article

Agarwal R, Sharma R, Patra MK, Gupta P, Parvin M, Nagar A, *et al*. Early embryonic mortality in bovine with emphasis on heat stress and its amelioration. *International Journal of Veterinary Sciences and Animal Husbandry*. 2026; SP-11(8): 24-32

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