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Review article

Climate change impact on reproduction performances and outbreak of infectious diseases in domestic animals

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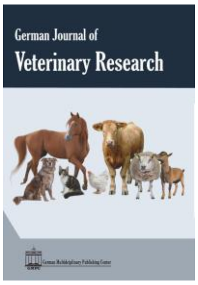

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Abstract

Climate change, driven primarily by rising greenhouse gas emissions, poses a major challenge to global ecosystems, including animal welfare and production systems. While natural processes contribute to climate variability, anthropogenic activities have caused unprecedented shifts, particularly affecting temperature patterns in Mediterranean and temperate regions. Animals display species-specific thermoregulatory responses to temperature fluctuations, which are essential for maintaining homeostasis. Deviations from optimal temperature ranges, influenced by environmental conditions and animal-specific factors, can disrupt physiological functions, including reproductive performance and resistance to infectious diseases. As climate change intensifies, understanding its impacts on animal health and productivity becomes increasingly critical. This review synthesizes recent research on how climate change affects reproductive inefficiency and the emergence of infectious diseases in domestic animals. It further discusses sustainable farming practices aligned with the One-Health framework, aimed at safeguarding environmental, animal, and human well-being. Moreover, the contribution of animal production to climate change, through greenhouse gas emissions and environmental degradation, underscores the urgent need for effective mitigation strategies. By integrating these insights, this work seeks to provide a foundation for addressing the complex challenges posed by climate change within animal agriculture and global sustainability initiatives.

Keywords: Climate change, Fertility, Heat stress, Infectious disease, Oocyte, Spermatozoa

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Introduction

The 20th century witnessed a significant increase in greenhouse gas (GHG) emissions (Pachauri et al., 2014), leading to a rise in the average surface temperature of the Earth, a phenomenon known as global warming (Hansen et al., 2001). Natural processes contribute to climate change (Rojas-Downing et al., 2017); however, anthropogenic temperature increases are much greater than those driven by natural factors. They are among the primary causes of ecosystem alteration, including increased susceptibility to infectious diseases, reduced reproductive efficiency, impaired growth performance, and declines in the quality of life of domestic animals (Rojas-Downing et al., 2017). Even though climate change manifests in various ways, temperature anomalies are among the most tangible effects in Mediterranean (and temperate) regions, with strong implications for animal welfare and production systems (Hansen et al., 2001).

Animal species exhibit different capacities for homeostatic regulation. Each animal species has its own optimal temperature range, known as the thermoneutral zone (TNZ). Exposure to temperatures outside the TNZ is reflected in their physiological and behavioural responses, which are triggered by an array of thermoregulatory mechanisms (Nardone et al., 2010). Domestic mammals spend a considerable portion of their dietary energy on thermoregulation by converting dietary calories into heat (thermoregulatory thermogenesis). In contrast, heat loss is primarily achieved through sweating or panting, which involves the evaporation of moisture from the skin's surface. Modulating the blood flow to the skin is, instead, a suitable strategy for both gaining and losing heat. Vasoconstriction decreases blood flow to the skin, thereby reducing heat loss from the body surface to the environment. Conversely, vasodilation increases heat loss. Furthermore, heat exchange depends on the thickness of the fur layer and/or the subcutaneous fat layer (Collier et al., 2006). Nevertheless, this variety of mechanisms interferes with the fine orchestration of the physiological processes underlying the animal's homeostatic balance, including, although not limited to, the animal's capability to defend itself against infectious diseases and its reproductive efficiency (Boni, 2019).

Given the impact of environmental temperature on animal health and production efficiency, various research groups worldwide are currently predicting and assessing the evolution of climate change across diverse regions of the globe, as well as studying its effects on animal health and livestock production. Although the number of studies focused on elucidating the physiological effects of climate change on domestic animals is constantly increasing (Nardone et al., 2010), the mechanisms by which these changes affect reproductive performance and/or the occurrence of infectious diseases in domestic animals are not yet fully understood, underscoring the need for further research. The present work aims to review the most recent studies assessing the role of climate change in reproductive inefficiencies and in the outbreak of infectious diseases threatening domestic animals. A discussion of the outcomes from the presented literature is provided, along with practical approaches and recommendations that promote sustainable breeding practices and safeguard environmental, animal, and human welfare, in line with the One Health approach.

Effect of climate change on crop production

Global warming alters temperature and rainfall patterns, negatively affecting crop growth. Climate change is making droughts and floods more frequent, altering the availability of seasonal water resources (Ceglar et al., 2019). The prolonged and increasingly arid dry season has reduced water supplies and exacerbated pressure on water resources. In contrast, excessive rainfall during the rainy season can lead to soil waterlogging, suffocating plants. Water shortages caused by fluctuations in rainfall patterns are also expected to intensify conflicts over water use. Extreme weather events, often attributed to climate change, such as floods, droughts, and storms, affect agricultural productivity, damage crops and infrastructure, and lead to soil erosion.

However, the impact of climate change on crop yield per unit area depends on the specific climate change scenario, crop type, planting area, and whether the fertilization effect of elevated carbon dioxide (CO_2) is accounted for. Based on results from various climate change scenarios, researchers in China found that yield per unit area of major grain crops will decrease by 2.6% for every 1 °C increase in temperature (YU et al., 2024). By contrast, a 1% increase in precipitation increased grain crop yield per unit area by about 0.4% (YU et al., 2024). Under elevated CO_2, fertilizer efficiency increased by approximately 16% on average, leading to an overall increase in grain crop yields (YU et al., 2024). The differences in the impact of climate change on the yield of the three major grain crops primarily stem from variations in CO_2 fertilization efficiency, crop varieties, temperature, and precipitation.

Animal production as the cause of climate change

Animal production is often associated with negative environmental impacts, including soil degradation, air and water pollution, and biodiversity loss (Bellarby et al., 2013; Reynolds et al., 2010; Thornton and Gerber, 2010). A study from Michigan State University estimated that livestock farming accounts for approximately 14.5% of total annual emissions of CO_2, nitrous oxide (N_2O), methane (CH_4), and ozone (O_3), collectively representing the primary greenhouse gases (Grasty, FAO, 1999).

Other factors influencing climate change are associated with manure from animal production (Bellarby et al., 2013). Intensive farming, in particular, can have a significant impact due to the large number of animals confined in limited spaces, leading to substantial manure accumulation. This manure releases various greenhouse gases, including methane and nitrous oxide, as highlighted by various studies (Bellarby et al., 2013). Nitrogen emissions from manure are influenced by various environmental factors, including soil infiltration, organic carbon content, pH, soil temperature, rainfall, and plant uptake rates (Mosier et al., 2004). Estimates suggest that global N_2O emissions from stored manure are equivalent to about 10 million tons of nitrogen annually (Bellarby et al., 2013). However, it is important to note that these values can vary considerably depending on farming practices, manure management, and local soil and climate conditions.

Another important effect of animal husbandry on climate change is the conversion of natural habitats into pasture and cultivated land (Thornton and Gerber, 2010). This has altered the natural landscape, disrupted the carbon cycle, and led to a release of large quantities of carbon dioxide into the atmosphere and an increase in greenhouse gas emissions. Similarly, the use of fertilisers and manure for fodder production, feed processing, and feed transport contributes significantly to overall GHG emissions from the livestock sector (Thornton and Gerber, 2010).

Livestock respiration also produces CO_2 emissions, but it is not considered a significant source due to its role in the global biological carbon cycle (Thornton and Gerber, 2010). However, livestock account for approximately 44% of global CH_4 emissions through normal digestive processes (e.g., enteric fermentation by the ruminal microbiota) (Thornton and Gerber, 2010).

Impact of climate change on animals' physiology

Climate change impairs the reproductive efficiency of livestock in both sexes (Nardone et al., 2010), altering not only sexual maturation events (i.e., pre-puberty and post-puberty) but also all stages of reproduction, including gamete production and transport, fertilisation, embryo development, gestation, and parturition (Nardone et al., 2010). Moreover, climate change is linked with abnormal expression of sexual behaviour, pre-term birth, and delayed postpartum recovery (Kumar et al., 2017).

Altered reproductive performances in the male

In the male reproductive system, the major problems attributable to heat stress are hormonal imbalances that affect spermatogenesis. This problem is exacerbated when climate change occurs more rapidly than the animal's ability to adapt (Renaudeau et al., 2012). Heat stress can disrupt the hypothalamic–pituitary–gonadal axis, leading to adverse effects on reproductive function. Seasonal variations influence the characteristics of semen in the ejaculate of *Bos indicus*, with anomalies in spermatozoa proportions being higher in summer than in winter. At the same time, sperm kinematics and viability remain largely unchanged between seasons (Nichi et al., 2006).

De Rensis and Scaramuzzi (2003) observed that bull semen quality was highest in spring, with greater membrane integrity compared to other seasons. The authors attributed this to two main factors: (i) increased testicular blood flow due to cooler spring temperatures, and (ii) improved semen quality resulting from better forage availability during spring (De Rensis and Scaramuzzi, 2003). Spermatogenesis in bulls takes approximately 65–70 days, and the optimal environmental temperature is 15-18 °C (Kastelic, 2013). Possible damage to spermatogenic cells can be detected in the ejaculate two to four weeks after excessive heat stress, and sperm quality may not return to normal until six to twelve weeks later (Kastelic, 2013). Hence, changes in sperm quality induced by environmental factors may be delayed for several weeks, which might explain the differences observed between studies. The testicles in most animals are maintained at a temperature below body temperature through specialised thermoregulatory mechanisms involving the pampiniform plexus of the spermatic cord, the scrotal muscle, and the cremasteric muscle. Increased testicular temperature leads to reduced sperm production, decreased sperm motility, and a higher proportion of morphologically abnormal sperm cells (Waldock et al., 2013). Testicular heating in cattle reduces the proportion of progressively motile and live spermatozoa, while increasing the incidence of morphological abnormalities in sperm heads. During spermatogenesis, germ cells at all stages of maturation are highly sensitive to heat shock damage. In this context, sperm cell damage is primarily due to apoptosis induced by oxidative stress and DNA strand rupture, as well as to indirect effects resulting from the functional impairment of Sertoli and Leydig cells. However, the degree of damage depends on both the extent and duration of temperature exposure (Rahman et al., 2018). In rams, spermatocytes in meiotic prophase are particularly susceptible to heat, whereas more mature spermatozoa typically exhibit metabolic and structural abnormalities (Setchell, 1998). Although sperm morphology can revert to normal, fertilisation rates decrease, with a higher incidence of embryo death, especially in rabbits (Setchell, 1998).

Altered reproductive performances in the female

Studies have shown that heat stress reduces luteinizing hormone (LH) levels (Wise et al., 1988) and inhibin but increases circulating follicle-stimulating hormone (FSH) (Roth et al., 2000). In cows, heat stress is associated with an increased number of small (2–4 mm diameter) and medium-sized (4–8 mm diameter) ovarian follicles. Elevated body temperature reduces both the duration and intensity of oestrus expression in buffaloes, along with lower circulating levels of progesterone, oestradiol, LH, and FSH during summer compared to colder periods (Rao and Pandey, 1983). Similarly, in cows, heat stress disrupts hormonal regulation, leading to reduced LH, lower inhibin, and elevated FSH (Wise et al., 1988; Roth et al., 2000).

A prolonged summer anoestrus in buffaloes has been attributed to the combined effects of poor nutrition and high environmental temperature (Kaur and Arora, 1984). Under severe heat stress, females may become infertile for a temporary period, a condition often termed “summer infertility.” The percentage of inseminated dairy cows becoming pregnant during the hot season is 22.1%, compared to 43.1% during the cold season (López-Gatius, 2003). The magnitude of summer infertility is lower in heifers that do not suckle or in cows producing less milk than in high-yielding cows. This difference is explained by the tendency of high-producing cows to enter a negative energy balance, as they prioritize milk production over reproductive investment (López-Gatius, 2003). In the context of climate change, these effects are expected to intensify, especially in tropical countries.

Following heat stress, the follicular response to gonadotropins is reduced, as demonstrated in goats (Ozawa et al., 2005). The proportion of follicles that ovulated 24 hours after heat exposure was significantly lower than that in animals maintained at a stable temperature. In dominant follicles, high temperatures reduce gonadotropin-induced production of androstenedione and oestradiol, while increasing progesterone production (Ozawa et al., 2005).

Impact on the oocytes’ function

Follicles formed during periods of heat stress produce oocytes of lower quality than those matured under optimal environmental conditions. A study conducted in Israel suggested that fertility recovery in lactating cows can be accelerated by removing heat-stressed follicles formed during the summer (Roth et al., 2000). By monitoring oocyte quality throughout the summer, the authors highlighted a carryover effect of heat stress, with delayed recovery of oocyte quality and embryo development observed in the autumn.

Recent evidence shows that heat stress impairs oocyte functionality by altering cumulus–oocyte complex interactions. Specifically, heat-stressed oocytes exhibit changes in membrane composition and permeability, which hinder normal communication with cumulus cells and compromise subsequent embryo development (Martín-Maestro et al., 2025). The induction of heat stress during ovulation and oocyte maturation in mice and cattle results in slower embryo development or the production of abnormal embryos (Miętkiewska et al., 2022). Moreover, several reports indicate that exposure to high temperatures during the pre-ovulatory period disrupts oocyte maturation (Soto and Smith, 2009).

Impairment of the oviduct, the uterine environment, and embryo development

Along with nutritional deficiency, studies have identified heat stress as one of the most critical factors during the pre-implantation period, when embryos are highly prone to damage, leading to failure of pregnancy recognition (Wolfenson and Roth, 2019). This can result in loss of conception or impaired development of the foetus and placenta, ultimately causing pre-term termination or continuation of pregnancy with deficits of variable severity (Wolfenson and Roth, 2019). Some studies suggest that heat stress reduces circulating progesterone concentrations (Wolfenson and Roth, 2019), likely due to reduced corpus luteum (CL) function arising from heat-compromised dominant follicles (Wolfenson et al., 2002).

Two primary mechanisms explain the negative impact of high temperatures on embryo survival. Firstly, high temperature can directly damage embryonic cells (Kaur and Arora, 1984). Secondly, it can alter the uterine environment by reducing progesterone levels, making it less favourable for embryonic development (Bellarby et al., 2013). Research has also shown that embryos can develop resistance mechanisms to counteract the harmful effects of high temperature. Recent studies, including those by Wang et al. (2024), have identified early signs of immune activation in newly formed embryos. In response to heat stress, embryos can trigger an "innate immune response" that eliminates damaged cells and promotes survival (Wang et al., 2024).

Exposure of lactating cows to heat stress revealed that, on day 1 after oestrus/artificial insemination, the proportion of blastocysts developing was lower compared with cows stressed on days 3 (8–16 cell stage), 5 (morula), and 7 (blastocyst), indicating that embryos at later developmental stages are relatively more resistant to heat shock (Ealy et al., 1993). *In vitro* embryo culture in cattle showed a similar trend, with reduced negative effects of heat stress during the late blastocyst stage (Edwards and Hansen, 1997).

The acquisition of embryo thermotolerance involves the ability to synthesise heat shock protein 70 (HSP70), which stabilises intracellular proteins and subcellular organelles. The synthesis of HSP70 has been observed as early as the two-cell stage in cattle (Edwards and Hansen, 1997) and mice (Christians et al., 1997), suggesting a pivotal role in early embryonic development. Additionally, heat stress reduces intracellular glutathione levels (Ozawa et al., 2005). Glutathione plays a crucial role in protecting cells from oxidative stress, facilitating detoxification, supporting immune regulation, and enabling protein and DNA synthesis. Exposure to heat stress immediately before and/or after the breeding season also has important effects on conception and pregnancy outcomes. An increased occurrence of early pregnancy loss has been reported (Soto and Smith, 2009). Conception rates appear to be most affected when animals are poorly adapted to climatic changes, suggesting that rising minimum daily temperatures are particularly critical for successful conception (Renaudeau et al., 2012).

Impairment of sexual behavior

Male sexual behaviour, including copulation, aggression, scent marking, and ultrasonic vocalisations, is regulated by testosterone, which acts through androgen and oestrogen receptors in specific brain regions. Within the hypothalamus, the medial preoptic area and the amygdala play pivotal roles in the neural control of male sexual behaviour (Hull and Dominguez, 2007). In females, sexual behaviour is primarily mediated through oestrogen receptors located in the hypothalamic nucleus.

The mechanism underlying heat stress–induced reductions in female sexual behaviour remains incompletely understood. However, evidence from studies in humans and other species suggests that oxytocin, in interplay with sex hormones and endogenous opioid peptides, may play a regulatory role. In males, heat stress reduces sexual activity through direct effects on both the hypothalamic–pituitary–adrenal (HPA) and hypothalamic–pituitary–gonadal (HPG) axes (Cunningham et al., 2012). These effects are consistent with the altered hormone profiles described above, ultimately leading to suppressed reproductive performance (Figure 1).

Communicable diseases and climate change

Animals are constantly exposed to a wide and dynamic range of pathogens. Their defence mechanisms, from physical barriers to complex, cell-mediated components, form an integrated network collectively referred to as the immune response. When organisms encounter adverse conditions that threaten homeostasis, and these cannot be avoided or tolerated, body systems act synergistically to activate and coordinate tailored responses (Escobedo-Bonilla et al., 2022).

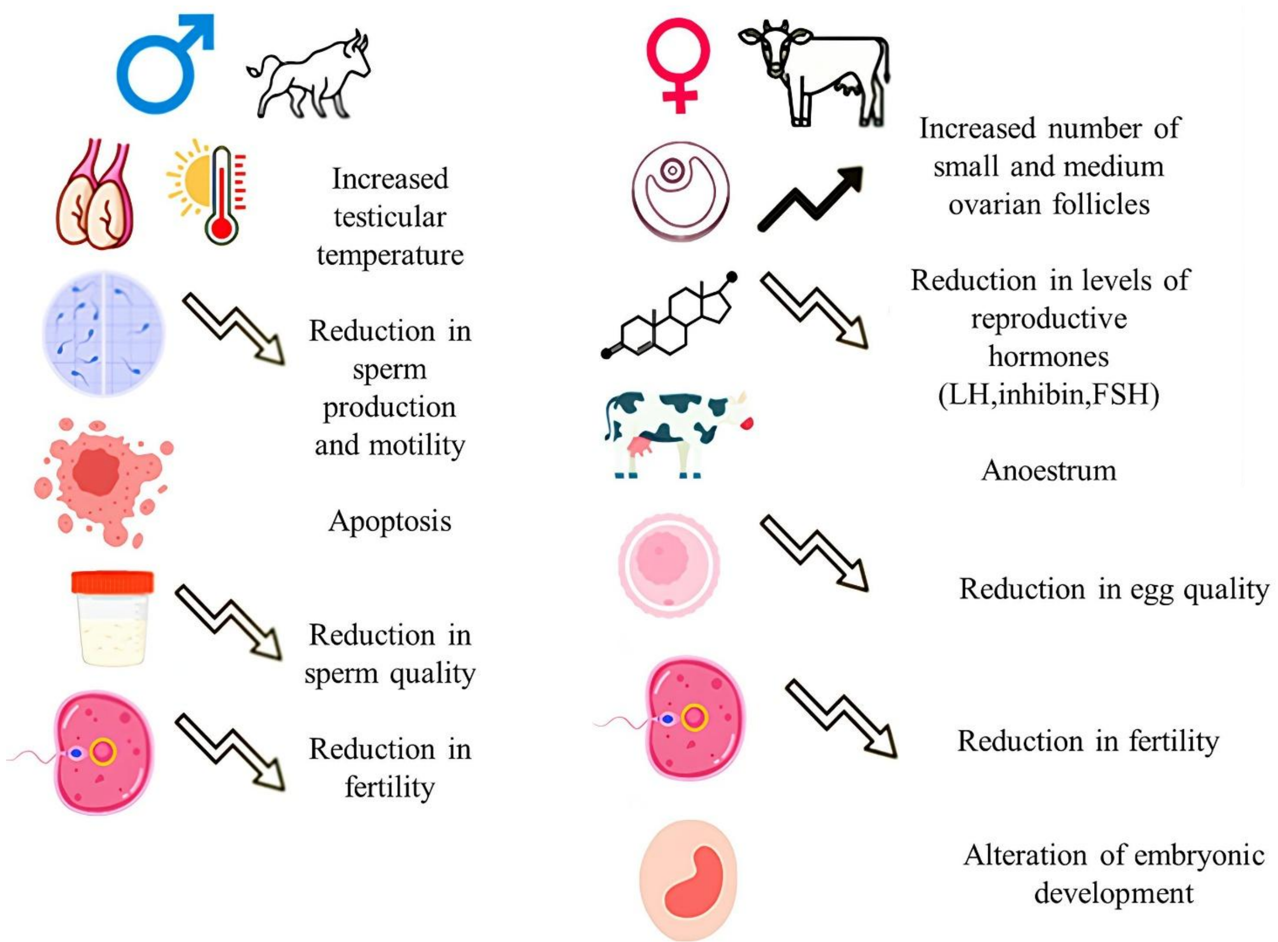

Figure 1: Effect of climate change on the reproductive performance of the male and female.

Clinical and experimental studies have demonstrated that immunodeficient or immunosuppressed organisms are more susceptible not only to severe infectious diseases but also to secondary complications, including impaired wound healing, increased mortality, and reduced lifespan. Over recent decades, environmental changes have contributed to the emergence of more than 40 infectious diseases, including HIV/AIDS, Ebola, Dengue (Procopio et al., 2024), viral haemorrhagic fevers, novel strains of cholera, and chytridiomycosis in amphibians, as well as antibiotic-resistant tuberculosis (Soto and Smith, 2009). Alarmingly, the emergence and re-emergence of such diseases is projected to accelerate in the future (Aguirre and Tabor, 2008), progressing in parallel with the decline of immune system efficiency under climate change (Soto and Smith, 2009).

Livestock diseases

The impact of climate change on infectious diseases in livestock depends on several variables, including primarily the geographical region, soil type and condition, disease characteristics, and its aetiological agent, and animal susceptibility (Thornton and Gerber, 2010). Animal health can be affected both directly and indirectly, particularly by increases in temperature. The direct effects of rising temperatures include increased morbidity and mortality (Thornton and Gerber, 2010). Elevated temperatures may accelerate the growth of pathogens and/or parasites that spend part of their life cycle outside the host, thereby triggering disease spread, facilitating outbreaks, or even introducing new diseases.

Another direct effect of rising temperatures is the expansion of vector-borne diseases, which is among the most concerning consequences. Wittmann et al. (2001) estimated that a 2°C increase in average temperature could promote the widespread dispersal of *Culicoides imicola*, the primary vector of bluetongue virus and other zoonotic viral agents, with serious repercussions for sheep, cattle, goats, and deer. In Australia, climate change is expected to have significant impacts on livestock, including a potential 18% loss in body weight due to increased tick infestations (Wittmann et al., 2001). Moreover, climate change can enhance the survival of insect vectors, including mosquitoes (Banhazi et al., 2008), with important consequences for the spread of mosquito-borne diseases in Europe. The projected changes in the geographical distribution of bluetongue virus in Europe further support this concern (Bellarby et al., 2013).

The effects of heat stress on immune activity are not always direct. They may depend on factors such as species, breed, genotype, age, level of acclimation, and the intensity and duration of exposure to adverse conditions. Sudden environmental changes can compromise the immune system's efficiency, thereby facilitating the onset and spread of infectious diseases (Soto and Smith, 2009). Nardone et al. (2002) reported that severe heat stress reduces colostral immunoglobulins (IgG and IgA) in dairy cows, negatively affecting the immunisation and survival of newborn calves. In addition, a dramatic depression in lymphocyte function has been observed in severely heat-stressed peri-parturient dairy cows, increasing their vulnerability to pathogens and reducing the efficacy of vaccination (Gaughan et al., 2009).

Climate change and zoonoses

Zoonoses, i.e., infectious diseases transmitted between animals and humans, are significantly influenced by climate change. Climate change affects the survival and distribution of both reservoir and vector species, as highlighted by Soto and Smith (2009) and Tazerji et al. (2022). These alterations can affect a pathogen's ability to infect other species, including humans. West Nile Virus (WNV) serves as a prime example. This mosquito-borne flavivirus, primarily transmitted by Culex mosquitoes, has a natural reservoir in wild birds but can also infect various other animals, including horses (Chancey et al., 2015; Shehata et al., 2025). Increasing interactions between animals and humans have put pressure on the virus to adapt, resulting in human infections. Humans are accidental hosts and may experience severe neurological damage, paralysis, coma, or even death (Acevedo-Whitehouse and Duffus, 2009). In this context, understanding vector habitat characteristics is crucial for predicting vector survival and distribution, as well as for designing effective mitigation strategies (Banhazi et al., 2008).

The rapid spread of *Aedes albopictus* in Europe exemplifies another climate-change–related risk. This mosquito is a potential vector for several arboviruses (Liu-Helmersson et al., 2019). Climate change is thought to contribute to the re-emergence of yellow fever and dengue fever in temperate regions, primarily due to rising temperatures (Procopio et al., 2024). Both diseases are caused by viruses of the Flavivirus genus, transmitted by forest mosquitoes, and capable of infecting humans (Al-Katanani et al., 2002). In Europe, indigenous dengue cases have been diagnosed, including one case in Croatia, along with 15 additional cases indicating recent infection (Wettemann et al., 1976). Climate projections for 2050 suggest that dengue transmission may shift geographically due to increases in humidity and temperature, potentially allowing for seasonal dengue outbreaks in southern Europe (Rojas-Downing et al., 2017).

It is also important to note that some insects can transmit multiple pathogens. For example, *Aedes albopictus*, although considered a secondary vector for dengue, can also transmit yellow fever (Rojas-Downing et al., 2017). Table 1 summarizes the major vector-borne zoonotic diseases. The table provides, for each vector-borne disease listed, the most relevant vector, hosts, and how climate change impacts transmission and diffusion over time and space.

Table 1: Summary of the major vector-borne diseases of animal and human concern.

Study	Disease	Vector	Affected Animals	Modes of transmission*
Soto and Smith (2009)	West Nile Virus	Mosquitoes (*Culex* spp.)	Wild birds, horses, humans	Increased vector distribution and seasonality
Banhazi et al. (2008)	Dengue	Mosquitoes (*Aedes* spp.)	Humans	
Bellarby et al. (2013)	Chikungunya	Mosquitoes (*Aedes* spp.)	Primates, rodents, humans	
Reynolds et al. (2010)	Malaria	Mosquitoes (*Anopheles* spp.)	Humans	
Hansen et al. (2001)	Yellow Fever	Mosquitoes (*Aedes* spp.)	Primates, rodents, humans	
Wettemann et al. (1976)	Dengue	Mosquitoes (*Aedes* spp.)	Humans	
Pachauri et al. (2014)	Dengue	Mosquitoes (Aedes spp.)	Humans	

* Facilitated by climate change

Discussion

The effects of climate change on reproductive performance manifest in multiple ways. It alters reproductive timing, gamete production, fertilization, and embryonic development. In addition, it can influence reproductive behaviours such as mate choice, copulation, parental care, and diapause (Kumar et al., 2017). Similarly, climate change affects infectious diseases by modulating the distribution, survival, and transmission of pathogens, as well as host susceptibility and immune competence. Therefore, understanding the influence of climate change on both reproductive performance and disease susceptibility is crucial for the conservation and sustainability of animal and plant populations.

Given the complexity and dynamic nature of this phenomenon, deciphering the mechanisms by which climate change affects reproductive performance and infectious disease susceptibility is essential for developing effective conservation strategies and protecting biodiversity (Acevedo-Whitehouse and Duffus, 2009; Shivanna, 2022). Monitoring increases in disease incidence, both infectious and non-infectious, is particularly important, as it indicates that populations are approaching stress thresholds and negatively impact immune function (Acevedo-Whitehouse and Duffus, 2009).

Monitoring climate adaptation from multiple perspectives is therefore highly desirable. Currently, assessing production traits as indicators of adaptation is relatively straightforward and standardized. Conversely, evaluating physiological adaptation through inherited traits related to heat tolerance remains poorly investigated (Acevedo-Whitehouse and Duffus, 2009; Renaudeau et al., 2012). Further research is needed to identify novel predictive traits of adaptation capacity.

In parallel, livestock management practices must be revised change. A primary intervention involves selecting for heat-tolerant animals. Typically, the high heat tolerance of local tropical breeds is associated with small body size, lower production levels, and specific morphological traits (e.g., skin or hair properties, sweating ability, tissue insulation, and special appendages) compared with those of conventional breeds (Pegorer et al., 2007). However, selection for these traits may conflict with market demands and increase the likelihood of negative recessive traits or genetic drift (Pegorer et al., 2007).

Other sustainable methods to help animals cope with climate stress include providing natural or artificial shade to minimise heat exposure (Collier et al., 2006). Trees and other shading materials are associated with improved milk yield, reduced respiratory rates, and lower rectal temperatures in dairy cows (Collier et al., 2006). However, shade alone does not eliminate all heat-stress effects. Additional strategies promoting acclimatization include water misting systems, which in pig production can improve growth performance by 5–10% (Banhazi et al., 2008). Similarly, sprinklers increased overall egg production by 3% during summer (Gaughan et al., 2009). Maximizing natural ventilation through well-oriented, semi-open buildings with high, insulated roofs further improves thermoregulation (Banhazi et al., 2008; Renaudeau et al., 2012). Optimizing functional nutrition programs to modulate the microbiota and enhance immune and reproductive performance is also being explored, with promising results (Ben Gunawan et al., 2023). Notably, these countermeasures can be applied to both domestic and wild animal populations.

A common perception is that mitigation measures are feasible for domestic animals but not for wild animals (Lacetera, 2019). Wild animals may migrate to favourable climates, but this exposes them to novel photoperiods or climatic conditions with limited food availability (Walker et al., 2023). Scarce resources can compromise reproductive success and immune function, thereby increasing pathogen exposure and threatening the survival of progeny (Lacetera, 2019). Climate change can also disrupt hormonal cycles by altering light-dark regimes, which in turn affects sexual maturation and reproductive cyclicity. Moreover, ecosystem-level impacts, such as prey decline, can cascade to predator reproduction, highlighting the interconnectedness of species and the broader ecological consequences of climate change.

Rising temperatures and altered weather patterns have also expanded the distribution of vector-borne diseases, including those transmitted by mosquitoes and ticks (Reynolds et al., 2010). This increases the risk of infectious diseases in animal populations, such as West Nile virus and dengue fever (Banhazi et al., 2008), and elevates the risk of zoonotic transmission to humans (Procopio et al., 2024).

Addressing these challenges on multiple fronts is crucial to ensure the survival and welfare of both domestic and wild animals under changing climatic conditions. Despite extensive research, the interactions between climate change and livestock performance remain incompletely understood. Most studies have been conducted at continental or regional scales, whereas local assessments are crucial for identifying the most vulnerable areas. Additionally, most research focuses on cattle, with limited data on monogastric livestock and wild species. Future studies should prioritize these gaps to develop comprehensive strategies for adaptation and conservation.

Conclusion

This work highlights the impairment of animal reproductive performance and the increased susceptibility to, as well as the emergence of, infectious diseases, two major consequences of global warming. These effects affect both the current generation (e.g., infectious diseases) and future generations (e.g., compromised reproductive performance). Insights from past studies provide a state-of-the-art understanding and serve as a foundation for future research, which is crucial for supporting the livestock sector in mitigating the impacts of climate change. Addressing these challenges requires interdisciplinary collaboration among professionals, including agronomists, physicists, meteorologists, engineers, economists, and veterinarians, to pursue shared solutions. It is increasingly evident that human health and well-being are closely linked to the condition of the planet that sustains us. Climate change has already altered the seasonal reproductive patterns of many plants, insects, amphibians, and birds. Whether these species can adapt to new conditions or face extinction will become evident over time. A healthy environment is essential for the well-being of all living systems on the planet. Living in harmony with the environment requires adopting sustainable practices that respect natural systems. It is hoped that, in the future, even this pressing "hot topic" of climate change will be considered a matter of the past.

Article Information

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The authors' contribution. All authors contributed equally to the work.

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