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Review

Impact of Climate Change on Semen Quality and Fertility of Bull

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Abstract

Male fertility is impacted by heat stress in a complicated and multifaceted pathway. Unfavourable climatic conditions can modify the complex regulatory systems that the testes and scrotum can protect developing sperm during their most vulnerable stages. Testicular heat stress has a negative correlation with sperm quality and fertility. It can alter morphology, viability, motility, and the ability of spermatozoa to fertilise. High temperature interferes with the oxidative metabolism of glucose in spermatic cells which resulting in mitochondrial dysfunctions. This adverse effect produces reactive oxygen species on sperm DNA and also affects antioxidant production, which generally protects sperm from oxidative attacks, resulting in derangement of the spermatogenesis process and epididymal dysfunction. This dysfunction leads to the development of abnormal and dead spermatozoa, which adversely affects the fertility of the bull. This review aimed to assess the early sequential effects of heat stress and impacts of climate change on semen quality and on the mechanisms leading to defective sperm production.

Keywords: Climate Change, Oxidative Stress, Semen Quality, Thermoregulation.

Introduction

Climate change can affect animal health and production by altering climatic factors such as air temperature, relative humidity, precipitation, and the frequency and intensity of extreme weather events (Gaughan *et al.,* 2009; Lacetera, 2019). Heat stress is the primary cause of fertility and productivity declines in impacted locations, making global warming a major problem for livestock production as well as animal welfare and wellbeing (Rashamol *et al.,* 2018). Heat stress can impair testicular thermoregulation, leading to testicular degeneration and a decline in sperm quality. This results in male infertility due to reduced sperm concentration and motility as well as increased abnormalities such as proximal protoplasmic droplets, diadems, vacuoles and other nuclear defects (Brito *et al.,* 2003; Rahman *et al.,* 2011; Das *et al.,* 2016). High ambient temperatures are considered one of the most significant factors contributing to subfertility in bulls in tropical countries (Takahashi, 2011). Spermatogenesis relies on testicular thermoregulation to maintain testicular temperature 2 to 6 °C below core body temperature (Alves *et al*., 2016).

Elevating the temperature of the testicles leads to an increase in cellular metabolism and autophagy of germ cells, DNA damage, which in turn produces an increase in the formation of reactive oxygen species (ROS) and oxidative stress (Ziaeipour *et al.,* 2021). ROS are oxygen-based molecules that are involved in several aspects of sperm physiology, such as the acrosomal reaction, capacitation process, hyperactivation, and sperm attachment to the oocyte's zona pellucida (De Lamirande and Lamothe, 2009; Agarwal *et al.*, 2014). Sperm cells, however, are particularly susceptible to ROS damage due to the high quantities of polyunsaturated fatty acids in the plasma membrane and the low levels of antioxidant enzymes in the cytoplasm (Nichi *et al*., 2017). The factors that contribute to the cascade of lipid peroxidation, which is not regulated properly, can affect sperm chromatin integrity and increase the frequency of DNA breaks (Aitken and Krausz, 2001). Oxidative damage can happen on many molecular categories, including proteins, carbohydrates, lipids, and nucleic acids (DNA, RNA). Accordingly, cell nuclei and mitochondrial membranes may be attacked by oxidative stress. The testes tissue that has high rates of metabolism and cellular reproduction is especially susceptible to oxidative stress.

Temperature Humidity Index (THI) to Assessment of Heat Stress Level

Temperature-humidity index (THI) is the universal and most precise indicator of stress assessment due to temperature and humidity. Hot climatic conditions lead to a decline in production and nutrient intake of animals. Heat generated by metabolizing nutrients contributed to body temperature maintenance in a cold environment. However, in a hot climate, heat needs to be dissipated to maintain body temperature and normal physiological functions. Marai *et al*. (2008) reported that exposure of animals to hot climatic conditions leads to drastic changes in the biological functions, which include a decrease in feed intake and its utilization, disturbances in enzymatic activity, metabolism of water, protein, energy, and mineral balances. THI accounts for the combined effects of environmental temperature and relative humidity and animal response. THI can be calculated by the formula (McDowell, 1972),

$$THI = 0.72 (C\ db + C\ wb) + 40.6$$

Where C db = dry bulb temperature (°C), C wb = Wet bulb temperature (°C). RH: Relative humidity (RH%)/100. If THI 72 = Absence of heat stress, 73 to 78= Mild heat stress, 79 to 88= Moderate heat stress, 89-98 = severe heat stress. Several studies report the classification of different zones based on THI values, whether the animals are comfortable.

Regulation of Scrotal and Testicular Temperature

A bull scrotum with an optimal scrotal thermoregulatory mechanism, a balanced left-right symmetry, and a reduction in temperature from the top to the bottom of the testis section had produced good-quality semen (Ruediger *et al.,* 2016; Sinha *et al.,* 2021). Digital infrared thermography is a non-invasive imaging examination with high accuracy. There is a positive correlation between testicular temperature and scrotal surface temperature. According to Coulter (1988), the bull testis produces morphologically viable sperm cells; its temperature must be lower than body temperature. Additionally, high environmental temperatures with inadequate testicular thermoregulation result in lower-quality semen in bulls.

Environmental factors are one of the most significant factors that affect sperm quality and semen production in

bulls. A lower testicular temperature of about 32°C is necessary for the production of viable spermatozoa. High testicular temperatures can cause thermal stress that can affect seminal and biochemical parameters, leading to reproductive problems in bulls (Cardozo *et al*., 2006). Each 1°C increase in testicular temperature leads to a decrease by 14% in the spermatogenesis process (Durairajanayagam *et al.*, 2015). High ambient temperatures significantly reduce sperm motility by decreasing mitochondrial activity and ATP synthesis (Gong *et al.*, 2017). High ambient temperatures, either alone or in combination with high humidity, impede evaporative heat escape from the scrotal surface, resulting in elevated testicular temperature. The testis is kept cool via a cooling system that includes the scrotum, muscles, and pampiniform plexus (Senger, 2003). The bovine testes and epididymis are placed in the scrotum, which is below the abdomen, to keep them at a lower temperature for proper functioning (Brito *et al.*, 2004). Scrotal skin in bulls is thin, hairless, and devoid of subcutaneous fat. It has a higher density of sweat glands than the skin on other regions of the body, allowing heat to be evacuated through sweating to facilitate heat transfer (Blazquez *et al*., 1988). To regulate temperature, smooth muscles in the cutaneous arterioles of the scrotum contract in cold weather and dilate in hot weather (Waites *et al.*, 1990). In cold weather, the testes might be drawn towards the belly, whereas in hot weather, they can hang away from the body. The dartos muscle is a thin sheet of smooth muscle located just beneath the scrotal skin that lowers the volume of the scrotum during contraction. The vascular system also regulates temperature; the testicular artery provides blood to the testes and generates an extensive network of superficial capillaries. The testicular vascular cone functions as a classic counter-current heat exchanger. In the vascular cone, the artery is tortuous and interwoven with the venous complex (pampiniform complex), transferring heat from the artery to the vein, contributing to testicular cooling (Brito *et al.*, 2004).

Impact of Heat Stress on Spermatogenesis

Heat stress has a major effect on dairy animal reproduction and bull fertility. The process of spermatogenesis is affected by temperature, humidity, and THI. The bovine testes and epididymides are located in the scrotum, just below the abdomen, where they are kept at a 4–5 degrees Celsius lower than in the abdomen for proper functioning (Brito *et al.*, 2004). Spermatogenesis is a complex biological process that takes place in the seminiferous tubules of the testis. High temperatures are known to result in male infertility, and normal spermatogenesis takes place in vivo between 34 and 35 degrees Celsius (Jobran *et al.*, 2024). Male germ cells are produced in the testis, maturation process in the epididymis, and are then dormant stored in the cauda epididymis. The sperm and epididymal fluid are mixed with secretions from accessory sex glands and deposited in the female reproductive system after ejaculation. Elevated temperature in the scrotum affects the spermatogenesis process and has a negative impact on testicular function, which causes degeneration of sperm cells, reducing spermatozoa's fertilizing capacity, and ultimately causing infertility (Paul *et al*., 2008; Rasooli *et al.*, 2010). Because the spermatogenesis process in the bull takes around 60 days, changes in ejaculate characteristics caused by an increase in testicular temperature would not be apparent immediately. Various factors will impact the transcription and translation of genes during the spermatogenesis process, which will not only change of the protein content but also alter the expression and localization of certain proteins (Chalmel and Rolland, 2015; Wang *et al*., 2020). When testes are exposed to heat, their temperature regulation mechanisms are broken, which leads to a rise in temperature, an oxidative stress response in germ cells, changes in protein expression, and a decrease in sperm function and activity, which ultimately leads to male infertility (Kesari *et al.*, 2018; Morrell, 2020)

Heat stress can cause apoptosis, DNA damage, disruption of the blood-testis barrier (BTB), and abnormalities in hormone release, among other effects on different types of testicular cells. Heat stress affects the bull sperm in terms of sperm motility and viability (Zhao *et al.*, 2021). Heat stress on testicular tissue causes DNA damage and apoptosis through mitochondrial mechanisms (Shahat *et al.*, 2020). Furthermore, damaged sperm in the vas deferens may break their DNA when exposed to high ambient temperatures, which can lead to male infertility (Kim *et al.*, 2013). Thus, heat stress is a high-risk factor that increases the likelihood of infertility, destroys testicular tissue, and reduces sperm quality.

Elevated testicular and scrotal temperatures in fertile bulls during high ambient temperatures have a negative effect on sperm production and quality, which may result in less sperm count and motility and more morphological abnormalities (Luceno *et al.*, 2020). The elevated testicular and epididymal temperatures can extremely harm sperm morphology (Shahat *et al.*, 2020), and the exposure of the testes to body temperature as a result of varicocele, cryptorchidism, testicular hyperthermia, and spermatogenesis imperfection can cause the death of germ cells (Thonneau *et al.*, 1998; Jhun *et al.*, 2022).

Oxidative Stress

As testicular temperature rises, so increase testicular metabolism and oxygen consumption to sustain aerobic metabolism (Rahman *et al.,* 2018). If there is insufficient blood flow to maintain a suitable degree of oxygenation, testicular tissue may become hypoxic and undergo tissue oxidative stress (Rahman *et al.,* 2018; Rizzoto and Kastelic *et al.,* 2020). In several animal, heat stress-induced poor sperm quality has been associated with increased ROS production and the resulting lipid peroxidation as well as DNA damage (Paul *et al.,* 2008; Kim *et al.,* 2013; Aitken *et al.,* 2016). Sperm lipid peroxidation causes chromatin degradation, sperm morphological defects, mitochondrial membrane dysfunction, which impairs energy production and sperm motility, loss of plasma membrane integrity and sperm abnormalities (Paul *et al*., 2008; Kim *et al.,* 2013; Leite *et al*., 2022).

Sperm lipid membranes are more susceptible to peroxidation than seminal plasma lipids (Garcia-Oliveros *et al.,* 2020). Because hydrogen peroxide (ROS) affects the sperm tail's contractility processes, bovine sperm motility is extremely vulnerable to oxidative stress (de Castro *et al.,* 2016). Cell homeostasis depends on the mitochondria's regulation of redox balance. When ROS production is more than antioxidant ROS neutralization, oxidative stress triggers apoptotic pathways (Abdelnour *et al.,* 2019; Garret *et al*., 2008). Spermatozoa are vulnerable to oxidative stress because their cytoplasm is too tiny to hold a sufficient number of defence enzymes, hence reducing their antioxidant capacity. Sperm mitochondria are vulnerable to electron leakage during oxidative phosphorylation, which produces reactive oxygen species (Aitken *et al.,* 2017).

Heat stress, impairs mitochondrial function and increase the formation of ROS (Lucio *et al.,* 2016). Acute and chronic exposure to heat stress has been shown to enhance oxidative DNA damage, sperm membrane fluidity, and mitochondrial ROS production in ruminants, which hinders in vitro fertilization and embryo development (Teixeira *et al.,* 2018).

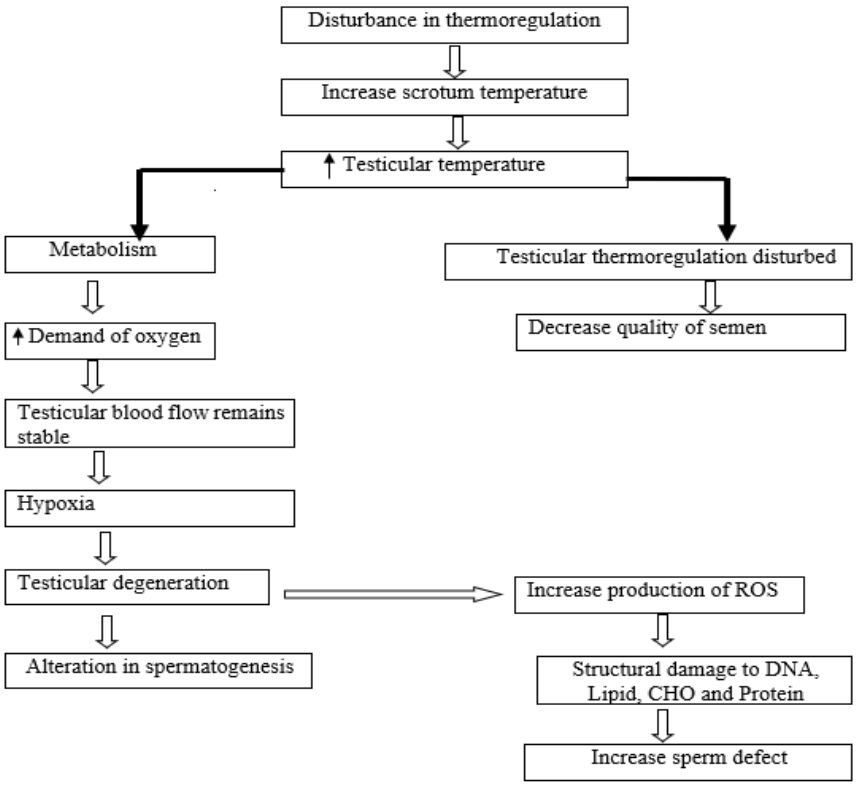

Fig 1. Impact of environmental factor on semen quality in bull

Effect of Heat Stress on Semen Quality

Elevated body temperature may alter the testicular temperature in the bull. The rise in testicular temperature leads to increased metabolism and thereby testicular oxygen demand. But testicular blood flow remains stable, and this increase does not offset the demand, resulting in hypoxia, generation of reactive oxygen species and alterations of spermatogenesis (Setchell, 2006) and dramatically reduces sperm production, motility and the number of live sperm per ejaculate, and increases the percentage of morphologically abnormal sperms (Kastelic *et al.*, 2019). The increase in testicular temperature leads to testicular degeneration and results in derangement of the spermatogenesis process. The decreased sperm concentration may be due to an increase in resorption of abnormal and dead spermatozoa in the epididymal sperm reserves as there is an increase in abnormal and dead spermatozoa due adverse effect of an increase in testicular temperature followed by testicular degeneration and epididymal dysfunction (Dhia and Ali, 2012; Kushwaha *et al.*, 2019). Bulls under heat stress in the summer produced poor-quality sperm with a lot of cytoplasmic droplets and abnormal heads (Koivisto *et al.*, 2009, Wu *et al.*, 2020). Heat stress hinders spermatogenesis process, reduces sperm motility and concentration, and also increases the amount of dead sperm, which results in low rates of fertilization and embryo development resulting infertility or subfertility (Capela *et al.*, 2022). The intensity and duration of heat stress, as well as the developmental phases of the impacted germ cells, determine the percentage, severity of abnormal sperm in the ejaculate (De *et al.*, 2017). Compared to winter the summer season has the lowest percentage of acrosome integrity (Bhakat *et al.*, 2014). A decrease in acrosomal function leads to decreased sperm fertility (Birck *et al.*, 2010). High temperature induced oxidative stress promotes early capacitation and increases the proportion of spermatozoa that are acrosome-reacted and capacitated (Zhu *et al.*, 2004). This condition of early capacitation reduces the lifetime of sperm as well as the rates of fertilization and embryo development. Moreover, sperm cells may carry substances that harm oocytes, such as lipid peroxidation and antioxidant depletion, which could impair the embryo's growth (de Castro *et al.*, 2016). Bulls are exposed to elevated ambient temperatures deteriorate semen quality and ultimately decreased reproductive performance.

Conclusion

The temperature of the testicles must be lower than the body temperature for the bull to produce healthy sperm. High ambient temperatures have a negative impact on bulls' ability to reproduce and the quality of their semen. Spermatogenesis is a continuous process, when bulls are under heat stress, the semen quality is reduced. The importance of cooling techniques increases when the outside temperature rises above 32°C. Sprinkling water on the animals' body surface will enhance evaporative cooling, and offering shade may be beneficial. Since a single bull can affect the pregnancy rate of many cows, bulls need to be protected from heat stress starting two months prior to the start of the breeding season.

Contribution by Authors

All the authors contributed equally to writing the manuscript. The final manuscript was read by all authors and consented to publication.

Conflict of Interests

There is no conflict of interest.

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References

1. Abdelnour, S. A., El-Hack, M. E. A., Khafaga, A. F., Arif, M., Taha, A. E., & Noreldin, A. E. (2019). Stress biomarkers and proteomics alteration to thermal stress in ruminants: A review. *Journal of Thermal Biology, 79*, 120–134. https://doi.org/10.1016/j.jtherbio.2019.01.009
2. Agarwal, A., Virk, G., Ong, C., & du Plessis, S. S. (2014). Effect of oxidative stress on male reproduction. *World Journal of Men's Health, 32*(1), 1–17. https://doi.org/10.5534/wjmh.2014.32.1.1
3. Aitken, R. J. (2017). Reactive oxygen species as mediators of sperm capacitation and pathological damage. *Molecular Reproduction & Development, 84*(9), 1039–1052. https://doi.org/10.1002/mrd.22871

4. Aitken, R. J., & Krausz, C. (2001). Oxidative stress, DNA damage and the Y chromosome. *Reproduction, 122*(4), 497–506. https://doi.org/10.1530/rep.0.1220497
5. Aitken, R. J., Gibb, Z., Baker, M. A., Drevet, J., & Gharagozloo, P. (2016). Causes and consequences of oxidative stress in spermatozoa. *Reproduction, Fertility and Development, 28*(1–2), 1–10. https://doi.org/10.1071/RD15325
6. Alves, M. B. R., Andrade, A. F. C., Arruda, R. P., Batissaco, L., Florez-Rodriguez, S. A., Oliveira, B. M. M., Torres, M. A., Lançoni, R., Ravagnani, G. M., Prado Filho, R. R., Vellone, V. S., Losano, J. D., Franci, C. R., Nichi, M., & Celeghini, E. C. C. (2016). Recovery of normal testicular temperature after scrotal heat stress in rams assessed by infrared thermography and its effects on seminal characteristics and testosterone blood serum concentration. *Theriogenology, 86*(3), 795–805. https://doi.org/10.1016/j.theriogenology.2016.02.009
7. Bhakat, M., Mohanty, T. K., Gupta, A. K., & Abdullah, M. (2014). Effect of season on semen quality of crossbred (Karan Fries) bulls. *Advances in Animal and Veterinary Sciences, 2*(11), 632–637. https://doi.org/10.14737/journal.aavs/2014/2.11.632.637
8. Birck, A., Christensen, P., Labouriau, R., Pedersen, J., & Borchersen, S. (2010). In vitro induction of the acrosome reaction in bull sperm and the relationship to field fertility using low-dose inseminations. *Theriogenology, 73*(9), 1180–1191. https://doi.org/10.1016/j.theriogenology.2010.01.004
9. Blazquez, N. B., Mallard, G. J., & Wedd, S. R. (1988). Sweat glands of the scrotum of the bull. *Journal of Reproduction and Fertility, 83*(2), 673–677. https://doi.org/10.1530/jrf.0.0830673
10. Brito, L. F., Silva, A. E., Barbosa, R. T., & Kastelic, J. P. (2004). Testicular thermoregulation in Bos indicus, crossbred and Bos taurus bulls: Relationship with scrotal, testicular vascular cone and testicular morphology and effects on semen quality and sperm production. *Theriogenology, 61*(3), 511–528. https://doi.org/10.1016/S0093-691X(03)00232-0
11. Brito, L. F. C., Silva, A. E. D. F., Barbosa, R. T., Unanian, M. M., & Kastelic, J. P. (2003). Effects of scrotal insulation on sperm production, semen quality, and testicular echotexture in Bos indicus and Bos indicus × Bos taurus bulls. *Animal Reproduction Science, 79*(1–2), 1–15. https://doi.org/10.1016/S0378-4320(03)00094-1
12. Capela, L., Leites, I., Romão, R., Lopes-da-Costa, L., & Pereira, R. M. L. N. (2022). Impact of heat stress on bovine sperm quality and competence. *Animals, 12*(8), 975. https://doi.org/10.3390/ani12080975
13. Cardozo, J. A., Fernandez-Juan, M., Forcada, F., Abecia, A., Muiño-Blanco, T., & Cebrián-Pérez, J. A. (2006). Monthly variations in ovine seminal plasma proteins analyzed by two-dimensional polyacrylamide gel electrophoresis. *Theriogenology, 66*(4), 841–850. https://doi.org/10.1016/j.theriogenology.2006.02.021
14. Chalmel, F., & Rolland, A. D. (2015). Linking transcriptomics and proteomics in spermatogenesis. *Reproduction, 150*(5), R149–R157. https://doi.org/10.1530/REP-15-0083
15. Coulter, G. H. (1988). Thermography of bull testes. In *Proceedings of the 12th Technical Conference of Artificial Insemination and Reproduction* (pp. 58–63). National Association of Animal Breeders.
16. Das, R., Sailo, L., Verma, N., Bharti, P., Saikia, J., & Imtiwati, K. R. (2016). Impact of heat stress on health and performance of dairy animals: A review. *Veterinary World, 9*(3), 260–268. https://doi.org/10.14202/vetworld.2016.260-268
17. de Castro, L. S., de Assis, P. M., Siqueira, A. F. P., Hamilton, T. R. S., Mendes, C. M., Losano, J. D. A., Nichi, M., Visintin, J. A., & Assumpção, M. E. O. A. (2016). Sperm oxidative stress is detrimental to embryo development: A dose-dependent study model and a new and more sensitive oxidative status evaluation. *Oxidative Medicine and Cellular Longevity, 2016*, 8213071. https://doi.org/10.1155/2016/8213071
18. De Lamirande, E., & Lamothe, G. (2009). Reactive oxygen-induced reactive oxygen formation during human sperm capacitation. *Free Radical Biology and Medicine, 46*(4), 502–510. https://doi.org/10.1016/j.freeradbiomed.2008.11.006
19. De, K., Kumar, D., Balaganur, K., Saxena, V. K., Thirumurugan, P., & Naqvi, S. M. K. (2017). Effect of thermal exposure on physiological adaptability and seminal attributes of rams under semi-arid environment. *Journal of Thermal Biology, 65*, 113–118. https://doi.org/10.1016/j.jtherbio.2017.02.013
20. Durairajanayagam, D., Agarwal, A., & Ong, C. (2015). Causes, effects and molecular mechanisms of testicular heat stress. *Reproductive Biomedicine Online, 30*(1), 14–27. https://doi.org/10.1016/j.rbmo.2014.09.018
21. Garcia-Oliveros, L. N., de Arruda, R. P., Batissaco, L., Gonzaga, V. H. G., Nogueira, V. J. M., Florez-Rodriguez, S. A., Almeida, F. S., Alves, M. B. R., Costa Pinto, S. C., & Nichi, M. (2020). Heat stress effects on bovine sperm cells: A chronological approach to early findings. *International Journal of Biometeorology, 64*(8), 1367–1378. https://doi.org/10.1007/s00484-020-01917-6

22. Garret, L. J. A., Revell, S. G., & Leese, H. J. (2008). Adenosine triphosphate production by bovine spermatozoa and its relationship to semen fertilizing ability. *Journal of Andrology, 29*(4), 449–458. https://doi.org/10.2164/jandrol.107.004648
23. Gaughan, J., Lacetera, N., Valtorta, S. E., Khalifa, H. H., Hahn, L., & Mader, T. (2009). Response of domestic animals to climate challenges. In K. L. Ebi, I. Burton, & G. R. McGregor (Eds.), *Biometeorology for adaptation to climate variability and change* (pp. 131–170). Springer. https://doi.org/10.1007/978-1-4020-8921-3_7
24. Gong, Y., Guo, H., Zhang, Z., Zhou, H., Zhao, R., & He, B. (2017). Heat stress reduces sperm motility via activation of glycogen synthase kinase-3α and inhibition of mitochondrial protein import. *Frontiers in Physiology, 8*, 718. https://doi.org/10.3389/fphys.2017.00718
25. Jhun, H., Lee, W. Y., Park, J. K., Hwang, S. G., & Park, H. J. (2022). Transcriptomic analysis of testicular gene expression in a dog model of experimentally induced cryptorchidism. *Cells, 11*(15), 2476. https://doi.org/10.3390/cells11152476
26. Jobran, A., Lunenfeld, E., & Huleihel, M. (2024). Effect of temperature on the development of stages of spermatogenesis and the functionality of Sertoli cells *in vitro*. *International Journal of Molecular Sciences, 25*(4), 2160. https://doi.org/10.3390/ijms25042160
27. Kastelic, J. P., Wilde, R. E., Bielli, A., Genovese, P., Rizzoto, G., & Thundathil, J. (2019). Hyperthermia is more important than hypoxia as a cause of disrupted spermatogenesis and abnormal sperm. *Theriogenology, 131*, 177–181. https://doi.org/10.1016/j.theriogenology.2019.03.027
28. Kesari, K. K., Agarwal, A., & Henkel, R. (2018). Radiations and male fertility. *Reproductive Biology and Endocrinology, 16*(1), 118. https://doi.org/10.1186/s12958-018-0431-1
29. Kim, B., Park, K., & Rhee, K. (2013). Heat stress response of male germ cells. *Cell and Molecular Life Sciences, 70*(15), 2623–2636. https://doi.org/10.1007/s00018-012-1155-1
30. Koivisto, M. B., Costa, M. T. A., Perri, S. H. V., & Vicente, W. R. R. (2009). The effect of season on semen characteristics and freezability in Bos indicus and Bos taurus bulls in the southeastern region of Brazil. *Reproduction in Domestic Animals, 44*(3), 587–592. https://doi.org/10.1111/j.1439-0531.2008.01016.x
31. Kushwaha, M. K., Bhakat, M., Mohanty, T. K., Kumar, R., Sinha, R., Rahim, A., Singh, P. P., Khosla, K., Shah, N., & Danish, Z. (2019). Use of scrotal infrared thermography and ultrasonography to understand thermoregulation of testis and sperm quality in Karan Fries breeding bull. *Indian Journal of Animal Research, 53*(11), 1420–1424. https://doi.org/10.18805/ijar.B-902
32. Lacetera, N. (2019). Impact of climate change on animal health and welfare. *Animal Frontiers, 9*(1), 26–31. https://doi.org/10.1093/af/vfy030
33. Leite, R. F., Losano, J. D. A., Kawai, G. K. V., Rui, B. R., Nagai, K. K., Castiglioni, V. C., Siqueira, A. F. P., D'Avila Assumpção, M. E. O., Baruselli, P. S., & Nichi, M. (2022). Sperm function and oxidative status: Effect on fertility in Bos taurus and Bos indicus bulls when semen is used for fixed-time artificial insemination. *Animal Reproduction Science, 237*, 106922. https://doi.org/10.1016/j.anireprosci.2021.106922
34. Luceno, N. L., Hostens, M., Mullaart, E., Broekhuijse, M., Lonergan, P., & Van Soom, A. (2020). High temperature-humidity index compromises sperm quality and fertility of Holstein bulls in temperate climates. *Journal of Dairy Science, 103*(10), 9502–9514. https://doi.org/10.3168/jds.2019-17957
35. Lucio, A. C., Alves, B. G., Alves, K. A., Martins, M. C., Braga, L. S., Miglio, L., Alves, B. G., Silva, T. H., Jacomini, J. O., & Beletti, M. E. (2016). Selected sperm traits are simultaneously altered after scrotal heat stress and play specific roles in in-vitro fertilization and embryonic development. *Theriogenology, 86*(4), 924–933. https://doi.org/10.1016/j.theriogenology.2016.02.002
36. Marai, I. F. M., El-Darawany, A. A., Fadiel, A., & Abdel-Hafez, M. A. M. (2008). Reproductive performance traits as affected by heat stress and its alleviation in sheep. *Tropical and Subtropical Agroecosystems, 8*(3), 209–234.
37. McDowell, M. A. (1972). *Improvement of livestock in warm climates.* W. H. Freeman.
38. Morrell, J. M. (2020). Heat stress and bull fertility. *Theriogenology, 153*, 62–67. https://doi.org/10.1016/j.theriogenology.2020.04.018
39. Nichi, M., Rijsselaere, T., Losano, J., Angrimani, D., Kawai, G., Goovaerts, I., & Bols, P. (2017). Evaluation of epididymis storage temperature and cryopreservation conditions for improved mitochondrial membrane potential, membrane integrity, sperm motility and in vitro fertilization in bovine epididymal sperm. *Reproduction in Domestic Animals, 52*(2), 257–263. https://doi.org/10.1111/rda.12910
40. Paul, C., Murray, A. A., Spears, N., & Saunders, P. T. K. (2008). A single, mild, transient scrotal heat stress causes DNA damage, subfertility and impairs formation of blastocysts in mice. *Reproduction, 136*(1), 73–84. https://doi.org/10.1530/REP-08-0036

41. Rahman, M. B., Schellander, K., Luceno, N. L., & Van Soom, A. (2018). Heat stress responses in spermatozoa: Mechanisms and consequences for cattle fertility. *Theriogenology, 113,* 102–112. https://doi.org/10.1016/j.theriogenology.2018.02.012
42. Rahman, M. B., Vandaele, L., Rijsselaere, T., Maes, D., Hoogewijs, M., Frijters, A., Noordman, J., Granados, A., Dernelle, E., & Shamsuddin, M. (2011). Scrotal insulation and its relationship to abnormal morphology, chromatin protamination and nuclear shape of spermatozoa in Holstein-Friesian and Belgian Blue bulls. *Theriogenology, 76*(7), 1246–1257. https://doi.org/10.1016/j.theriogenology.2011.05.018
43. Rashamol, V. P., & Sejian, V. (2018). Climate resilient livestock production: Way forward. *Dairy and Veterinary Science Journal, 5*(1), 555673. https://doi.org/10.19080/JDVS.2018.05.555673
44. Rasooli, A., Jalali, M. T., Nouri, M., Mohammadian, B., & Barati, F. (2010). Effects of chronic heat stress on testicular structures, serum testosterone and cortisol concentrations in developing lambs. *Animal Reproduction Science, 117*(1–2), 55–59. https://doi.org/10.1016/j.anireprosci.2009.04.001
45. Rizzoto, G., & Kastelic, J. P. (2020). A new paradigm regarding testicular thermoregulation in ruminants. *Theriogenology, 147,* 166–175. https://doi.org/10.1016/j.theriogenology.2020.02.020
46. Ruediger, F. R., Chacur, M. G. M., Alves, F. C. P. E., & Ramos, A. A. (2016). Digital infrared thermography of the scrotum, semen quality, serum testosterone levels in Nellore bulls (Bos taurus indicus) and their correlation with climatic factors. *Semina: Ciências Agrárias, 37*(1), 221–232. https://doi.org/10.5433/1679-0359.2016v37n1p221
47. Senger, P. L. (2003). The organization and function of the male reproductive system. In *Pathways to pregnancy and parturition* (pp. 44–79). Current Conceptions.
48. Setchell, B. P. (1998). The Parkes Lecture: Heat and the testis. *Journal of Reproduction and Fertility, 114*(2), 179–194. https://doi.org/10.1530/jrf.0.1140179
49. Shahat, A., Rizzoto, G., & Kastelic, J. (2020). Amelioration of heat stress-induced damage to testes and sperm quality. *Theriogenology, 158,* 84–96. https://doi.org/10.1016/j.theriogenology.2020.09.006
50. Sinha, R., Bhakat, M., Mohanty, T. K., Kumar, R., Ranjan, A., Rahim, A., Lone, S. A., Shah, N., Paray, A. R., Patil, C. S., & Singh, A. (2021). Seasonal variation of sperm kinematics in Murrah bulls under the tropical climatic condition. *Buffalo Bulletin, 40*(1), 87–98.
51. Takahashi, M. (2011). Heat stress on reproductive function and fertility in mammals. *Reproductive Medicine and Biology, 11*(1), 37–47. https://doi.org/10.1007/s12522-011-0105-7
52. Teixeira, J., Deus, C. M., Borges, F., & Oliveira, P. J. (2018). Mitochondria: Targeting mitochondrial reactive oxygen species with mitochondriotropic polyphenolic-based antioxidants. *International Journal of Biochemistry & Cell Biology, 97,* 98–103. https://doi.org/10.1016/j.biocel.2018.02.001
53. Thonneau, P., Bujan, L., Multigner, L., & Mieusset, R. (1988). Occupational heat exposure and male fertility: A review. *Human Reproduction, 13*(8), 2122–2125. https://doi.org/10.1093/humrep/13.8.2122
54. Waites, G. M. H. (1970). Temperature regulation and the testis. In A. D. Johnson, W. R. Gomes, & N. L. Vandemark (Eds.), *The testis* (Vol. 1, pp. 241–281). Academic Press.
55. Wang, X., Zhang, X., Dang, Y., Li, D., Lu, G., Chan, W. Y., Leung, P. C. K., Zhao, S., Qin, Y., & Chen, Z. J. (2020). Long noncoding RNA HCP5 participates in premature ovarian insufficiency by transcriptionally regulating MSH5 and DNA damage repair via YB1. *Nucleic Acids Research, 48*(8), 4480–4491. https://doi.org/10.1093/nar/gkaa162
56. Wu, Y. Q., Rao, M., Hu, S. F., Ke, D. D., Zhu, C. H., & Xia, W. (2020). Effect of transient scrotal hyperthermia on human sperm: An iTRAQ-based proteomic analysis. *Reproductive Biology and Endocrinology, 18*(1), 83. https://doi.org/10.1186/s12958-020-00633-5
57. Zhao, F., Whiting, S., Lambourne, S., Aitken, R., & Sun, Y. P. (2021). Melatonin alleviates heat stress-induced oxidative stress and apoptosis in human spermatozoa. *Free Radical Biology and Medicine, 164,* 410–416. https://doi.org/10.1016/j.freeradbiomed.2020.12.450
58. Zhu, B., & Setchell, B. P. (2004). Effects of paternal heat stress on the in vivo development of preimplantation embryos in the mouse. *Reproduction Nutrition Development, 44*(6), 617–629. https://doi.org/10.1051/rnd:2004061
59. Ziaeipour, S., Piryaei, A., Aliaghaei, A., Nazarian, H., Naserzadeh, P., Ebrahimi, V., Abdi, S., Shahi, F., Ahmadi, H., Fathabadi, F., & Abdollahifar, M. A. (2021). Chronic scrotal hyperthermia induces azoospermia and severe damage to testicular tissue in mice. *Acta Histochemica, 123*(4), 151712. https://doi.org/10.1016/j.acthis.2021.151712

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