

Editorial: The harmfulness of excessive heat on sperm maturation in mammals



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Keywords: scrotal thermoregulation, environmental influence, epididymal transit, spermatogenesis, sperm quality

Over the last few decades, sperm maturation in several mammalian species has been investigated due to its role in providing fertilizing capacity and properties related to sperm motility and plasma membrane structure (Cooper 1995; Souza et al 2016). The epididymis is the organ responsible for sperm maturation, which is located longitudinally on the posterior border of the testis, composed of a single, highly convoluted duct and lined composed of main, basal, apical, and clear cells with pseudostratified, columnar, and stereociliated epithelium, which act for sperm concentration, protection, transport, and storage (Sullivan and Miesusset 2016).

It is postulated that the epididymis promotes sperm maturation by establishing a series of triggers that cause cellular and molecular changes in the emission or near or in the oocyte and prevent the premature occurrence of the events (Amann et al 1993). The organ is segmented and can be divided ultrastructurally and histologically into: initial segment; caput - a dilated region that extends beyond the superior pole of the testis; corpus - the intermediate segment; cauda - the lower and narrower portion (Robaire et al 2006). Each segment presents specific contributions in its performance in the luminal microenvironment, essential for the sperm to mature when it reaches the cauda region, being then stored (Cornwall, 2009). The initial segment and the efferent ducts are responsible for the absorption of the fluid that leaves the testicular network. The main cells of the caput are responsible for the secretion of proteins, which are adsorbed on the surface of the sperm membrane, modifying its protein composition. As for the main cells in the corpus, due to a large amount of lipids present in the supranuclear region, they act by modifying the lipid composition of the sperm plasma membrane. Finally, in the cauda of the epididymis, its epithelium is much shorter than the anterior segments, and clear cells are predominant, which act in the secretion of glycoconjugates (James et al 2020). Clear cells phagocytose cytoplasmic droplets that are released from sperm during epididymal transit, in addition to phagocytosing other luminal debris (Cooper 2011). In the cauda region, excess luminal proteins are reabsorbed, while hormones are secreted to keep sperm quiescent (Robaire et al 2006).

Sperm maturation occurs along with the epididymal transit, resulting from a joint action of specific and well-orchestrated physiological processes, which promote a series of morphofunctional changes in male gametes from different regions of the epididymis (James et al 2020). These changes include releasing and absorbing fluids, ions, antioxidants, and exosomes. During epididymal transit, spermatozoa are known to lose or modify several of their surface proteins and gain transient or permanent surface proteins during maturation (Gervasi and Visconti 2017).

It is known that the process of spermatogenesis is susceptible to fluctuations in body temperature, and ambient temperature has a strong influence on the sperm quality of mammals (Llamas-Luceño et al 2020; Sabés-Alsina et al 2019). Despite all efforts in physiological and metabolic processes for the optimal development of spermatogenesis and sperm maturation, if the individual cannot efficiently thermoregulate their testes in the face of excessive heat stress, their sperm maturation will be compromised, causing loss of sperm quality (Figure 1). Body and testicular thermoregulation play a crucial role in maintaining normal spermatogenesis in the testes, and prolonged exposure to excessive heat contributes to gradual male infertility (Ivell 2007). In humans, the scrotal temperature is on average 2 to 4 °C lower than body temperature, and a 1 to 1.5 °C rise in scrotal temperature can adversely impact sperm production and sperm morphological abnormalities, causing oligozoospermia, azoospermia, and teratozoospermia (Hamerezaee et al 2018). Therefore, any environmental influence promoting increased scrotal temperature will directly affect the spermatogenesis process (Dantas et al 2021). In addition, environmental stresses caused by temperature elevation trigger metabolic changes that activate heat shock proteins (HSPs), among which the most important is HSP70s. This is one of the main classes of proteins responsible for the modeling, assembling, and disassembling of other proteins that play crucial roles in spermatogenesis and sperm maturation (Huang et al



2005). Thus, any environmental factor that disturbs its normal expression and regulation results in a decrease in total sperm count, motility and, therefore, in mammal male infertility (Erataet al 2008).

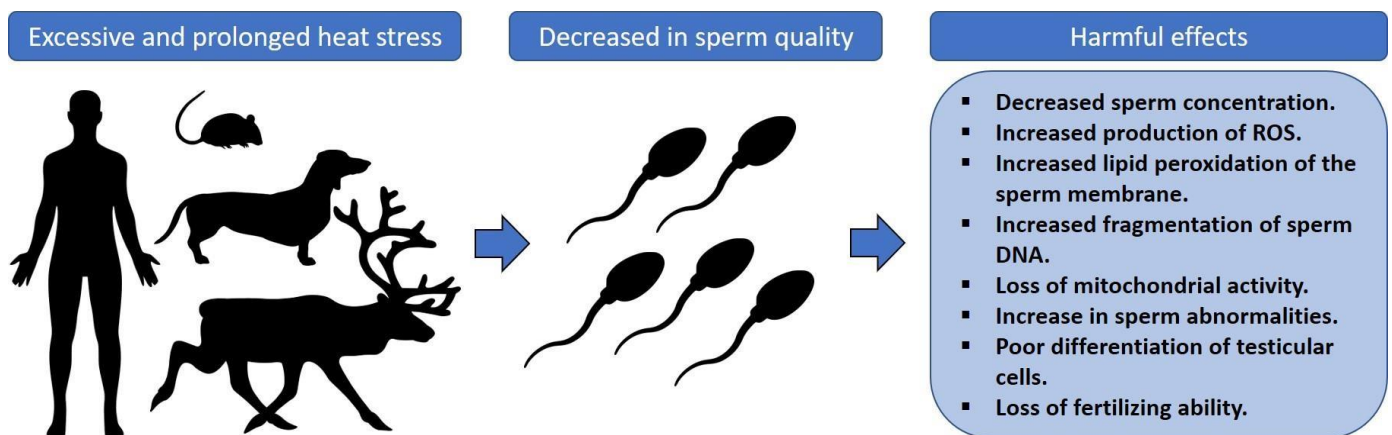


Figure 1 Harmful effects on spermatogenesis and sperm maturation of mammals due to prolonged exposure to heat.

Higher scrotal temperatures result in increased testicular metabolism without increasing blood supply, which leads to tissue hypoxia and oxidative stress at the site (Zhu et al 2019). It is known that inefficient testicular thermoregulation in the face of excessive heat stress causes an increase in the production of reactive oxygen species (ROS), which causes more DNA fragmentation and lipid peroxidation of the sperm membrane induced by oxidative stress due to the high levels of polyunsaturated fatty acids (PUFAs) (Sabés-Alsina et al 2019). In rats, heatstroke stress can cause erectile dysfunction, poorly differentiated seminiferous tubules, decreased sperm quality, interstitial Leydig cells, and Sertoli cells (Lin et al 2021).

Notably, ambient temperature modulates male fertility in mammals, especially testicular thermoregulation and consequent sperm maturation. Adverse environmental factors that alter the body temperature can result in low sperm quality, modifying sperm morphofunctional and biochemical characteristics. Therefore, excessive heat stress must be prevented and mitigated by establishing strategies and protocols that allow for reducing the damage caused by it.

Conflict of Interest

The author declares that there is no conflict of interest.

Funding

Not applicable.

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