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Review Article

Role of Mitochondrial Function in Farm Animals' Health and Production

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Abstract | Mitochondria are organelles found in most cells, play a crucial role in maintaining the body's health by regulating energy metabolism, oxidative stress, and calcium homeostasis which all are essential for most body functions. The efficiency and quantity of mitochondria within cells are essential for several normal activities including grow, reproduce, and respond to environmental stressors. One of the factors that have been suggested as a reason for mitochondria dysfunction is genetics modifications. Farm animals have been genetically modified to produce more products to meet the market demand. However, along with the positive gains in production efficiency have come some unintended consequences such as lower products quality and impair animal's health. Healthy mitochondria are essential for postmortem meat quality, impacting color and tenderness of the meat, while mitochondria dysfunction impairs performance and deteriorating final product quality. Mitochondria dysfunction alters cellular functions leading to a range of problems, including metabolic disorders and higher tissue damages. Although, several strategies have been introduced to improve mitochondria function, but none of them has fully addressed the problem in farm animals. Understanding the relationship between mitochondrial function and farm animal's performance is crucial for the development of targeted therapies aimed at improving their performance and health. This review attempted to summarize recent works on mitochondrial function and its implications for animal health and performance and suggest potential treatment strategies.

Keywords | Mitochondria, Environment, Livestock, Performance, Meat quality, Treatment strategies

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Mitochondria

Mitochondria biology in farm animals: Mitochondria are oval/round organelles (approximately 0.5 to 10 µm in size) that exist in the cytoplasm of most eukaryotic cells with several vital functions in the body. Mitochondria are highly mobile, but they also can stay in one place and constantly changing their shape or performing fission (divide into two) and fusion (fuse two and make a larger one) depends on the body requirement. They are associated with microtubules (provide structure and shape to cells), which determines the orientation and distribution of mitochondria in different types of cells. Mitochondria distribution/population in tissues varies depending on the required amount of ATP (Kara, 2025). Mitochondria structure includes inner and

outer membranes with completely different functions. The outer membrane is permeable to small and large molecules while the inner membrane is lipid bilayer and selectively permeable to only very small molecules into the matrix. The matrix contains DNA of the mitochondrial and the enzymes of the Krebs cycle or citric acid cycle (TCA cycle) (Alberts *et al*., 2002). Mitochondria has the cristae which helps to increase the surface area of the inner mitochondrial membrane, providing a higher number of electron transport chain enzymes and ATP synthase fits into the mitochondrion (Paumard *et al*., 2002). The intermembrane space is essential for protein transport, lipid homeostasis, and signaling pathways like apoptosis, basically coordinating various cellular activities by linking the outer membrane and the inner membrane (Herrmann and Riemer, 2010). The structure of mitochondria is presented in Figure 1.

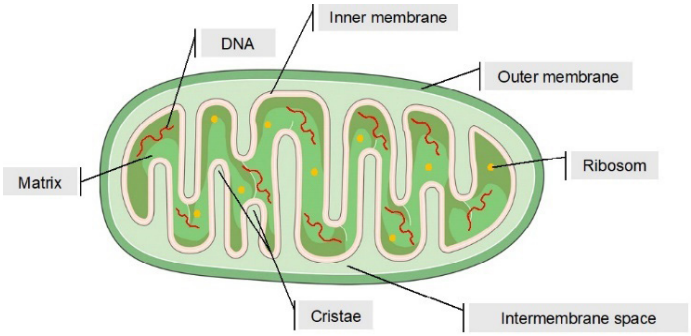

Figure 1: The structure of mitochondria.

The essential function of mitochondria is to generate a high amount of ATP for the body through oxidative phosphorylation and a series of reactions known as TCA cycle in the inner membrane (cristae). Although ATP production is the primary role of the mitochondrial, they are also responsible for several other important tasks including apoptosis, storing calcium, synthesis of heme (an iron-carrying protein) and maintain body temperature (Tzagoloff, 2012). Electrons move through protein complexes in the inner membrane, and energy generated from the electron chain is then used to transport proteins back across the membrane, which power ATP synthase to ATP (Zangari *et al*., 2020).

Apoptosis is a pathway that destroys old or unhealthy cells as part of growth and development (Palmer *et al*., 2021). Because certain diseases involve a breakdown in normal apoptosis, mitochondria are thought to play a role in the disease. The mitochondria release cytochrome C into the cytoplasm as a signal that activates cascade lead to cell degeneration and damage-induced degeneration (Bossy-Wetzel *et al*., 1998).

Calcium is essential for a range of cellular functions including regulating cell survival, aerobic metabolism, neurotransmitter, muscle function and blood clotting (Protasoni and Zeviani, 2021). Mitochondria stores calcium using a transport mechanism named calcium uniporter that helps to move calcium into the mitochondria (Reggiani and Marcucci, 2022) and managing calcium level (Romero-Garcia and Prado-Garcia, 2019). The stored calcium releases when it needed by mitochondria for several purposes such as regulating calcium signaling (Pivovarova and Andrews, 2010), apoptosis and the citric acid (Reggiani and Marcucci, 2022).

Mitochondria respiration play an essential role in maintaining body temperature by generating heat within the body through a process called thermogenesis (Lane, 2018). More specifically, mitochondria produce heat through a mechanism called mitochondrial uncoupling where energy that would normally be used to create ATP is instead released as heat due to the movement of protons across the mitochondrial membrane without generating ATP. This occurs mainly through the activity of uncoupling proteins and is considered the primary source of heat generation within the body's cells (Kang *et al*., 2024).

Mitochondria susceptible to mutations: Mitochondrial DNA (mtDNA), the circular chromosome located within mitochondria, is important for cellular energy production, and its integrity, and mutations or damages to mtDNA impacts overall cell's health (Sharma and Sampath, 2019). Different sort of mutations may occur in mtDNA as follow: point mutation (single base change in the mtDNA sequence) (Craigen, 2012), large deletions or rearrangements (Tuppen *et al*., 2010), insertion (addition of DNA sequences) (Fields *et al*., 2022), duplication (creation of extra copies of DNA sequences) (Craigen, 2012), inversions (reversal of DNA sequences) (Tuppen *et al*., 2010) and heteroplasmy (Wallace and Chalkia, 2013). Majority of disorders associated with mitochondrial dysfunction are caused by mutations in mtDNA (Ryzhkova *et al*., 2018) resulting in reduced the production of cellular ATP. mtDNA is highly susceptible to mutations as the mitochondria is major a site of generating reactive oxygen species (ROS) (Shokolenko *et al*., 2009). mtDNA is in the mitochondrial matrix, when mitochondria's health is disrupted, the mitochondria environment generates high levels of ROS, making mtDNA susceptible to oxidative damage (Nissanka and Moraes, 2018). Although several antioxidants within the mitochondria neutralize ROS, but some ROS may inflict damage to mtDNA. In addition, extensive oxidative damages caused by external factors may also increase damages to mtDNA.

Environmental Stressors and Mitochondria Dysfunction

Several environmental factors, such as high environmental temperature, high level of ammonia, and heavy metals, alter

mitochondria function by increasing oxidative stress resulting in higher mutations in mtDNA followed by disruptions in cellular ATP production and potentially contributing to various diseases (Figure 2). In fact, mitochondrial health directly impacts livestock productivity, and it could be compromised by environmental factors.

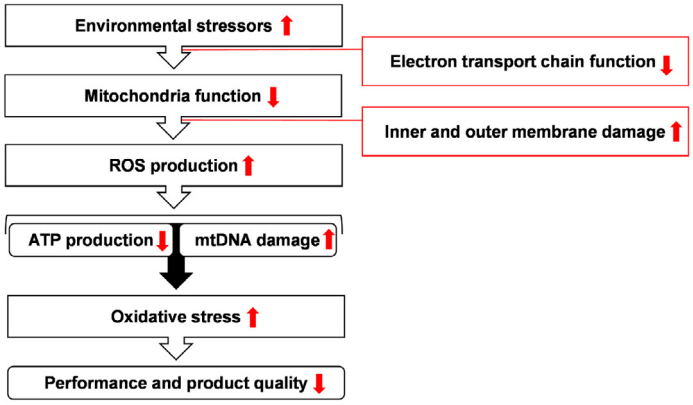

Figure 2: Links between environmental stressors, mitochondrial function and livestock outcomes. Under stress, reactive oxygen species (ROS) production increases resulting in higher mitochondria DNA (mtDNA) damage and oxidative damage which impairs animal's production.

High environmental temperature: Farm animals require optimum environmental temperature to improve the immune system, meat quality and growth performance (Belhadj Slimen *et al*., 2016; Shakeri *et al*., 2020). Under heat stress conditions, when body's temperature passes a certain level, it will add additional pressure on animal's body and will push them to use more nutrients to cool their body temperature than improving their performance. Heat stress negatively impacts biological molecules, cell functions and induces oxidative cell damage and activates of apoptosis pathways (Pandey *et al*., 2012). Long-term high temperature increases the production of ROS leading to higher oxidative damages to tissues by altering mitochondria function (Belhadj Slimen *et al*., 2016). ROS are a group of unstable molecules that can damage cell components when generated in high amount.

Although mitochondria are not the only place that generate ROS, they are responsible for the highest amount of ROS production in the body. When normal concentration of ROS is disturbed, mitochondria are the first cellular compartment to be damaged (England *et al*., 2004). Alterations in mitochondria structure caused by increased ROS production under heat stress lead to swollen mitochondria and broken cristae (Song *et al*., 2000), mitochondrial protein denaturation (Mujahid *et al*., 2007) and higher cell death (Du *et al*., 2008) which leads to higher tissue damage and potentially impairs animal's performance and health. In fact, heat stress misfolds and aggregates proteins within the mitochondria by increasing ROS, leading to impaired mitochondrial function (Wilkening *et al*., 2018). Additionally, heat stress disrupts the mitochondrial membrane, which is important for ATP production and can increase apoptosis (Qian *et al*., 2004).

Ammonia: Livestock produce ammonia (colorless gas) by breakdown of animal's urea/feces (Atia, 2006). When ammonia reaches to a danger level, increases lysosomal pH and results in the termination of lysosomal ammonia storage and ammonia reflux into mitochondria leading to higher mitochondrial damage and apoptosis (Shahid M *et al*., 2014). Ammonia damages mitochondria by disrupting the mitochondrial membrane leading to increased production of ROS, ultimately increasing oxidative stress and potentially causing cell death (Niknahad *et al*., 2017). Higher oxidative stress means higher tissue damages and deteriorating the production quality. In fact, if high amount of ammonia accumulates in the blood, damages mitochondria by inhibiting key enzymes in tricarboxylic acid cycle resulting in low energy production within mitochondria (Felipo and Butterworth, 2002).

Heavy metals: Heavy metals such as lead, cadmium, zinc and nickel can be found in livestock feed or water which can be toxic at high levels (Ebrahimi *et al*., 2023; Tahir and Alkheraije, 2023). Heavy metals can generate high amounts of ROS as well as ROS clearance malfunction lead to higher DNA damage (Itziou *et al*., 2011), alter mitochondria membrane permeability and inhibit antioxidant enzyme activity resulting in mitochondrial dysfunction (Sun *et al*., 2022) through Fenton reaction. The reaction acts as a catalyst to break down hydrogen peroxide and generates ROS (Shahid M *et al*., 2014). Furthermore, heavy metals disrupt the electron transport chain (within mitochondria) and increase damages to the mitochondrial membranes (inner and outer membranes), which can disrupt mitochondrial function, leading to reduced ATP production and increased ROS production (Sun *et al*., 2022).

Livestock Production and Mitochondria

Dairy: In dairy animals, mitochondria play a crucial role in milk production by generating the necessary ATP through cellular respiration, making them essential for the biosynthesis of milk components like proteins and fats, as the mammary gland requires a significant amount of ATP to synthesize milk. mtDNA is an important factor in mitochondrial function and impacts milk composition and energy density (Brajkovic *et al*., 2025). Conditions like heat stress can disrupt mitochondrial function in dairy cows, impacting their milk production and overall health (Favorit *et al*., 2021). Therefore, healthy mitochondrial is vital for high milk yield and overall dairy cow health (Favorit *et al*., 2021). Increased milking frequency in dairy cattle is

associated with increased mitochondrial density in the mammary gland (Favorit *et al*., 2021).

Pig: In pigs, mitochondrial function is directly linked to their overall performance, particularly in terms of muscle growth and feed efficiency, as healthy mitochondria are crucial for producing energy needed for muscle contraction, meaning that pigs with better mitochondrial function tend to exhibit improved growth rates and better conversion of feed into muscle mass; research suggests that analyzing mitochondrial function in muscle tissue can be a good indicator of a pig's overall performance potential (Qiao *et al*., 2023). Researchers often take muscle biopsies from pigs to analyze mitochondrial function by measuring parameters like oxygen consumption, ATP production, and mitochondrial membrane potential (Bekebrede *et al*., 2021; Fu *et al*., 2017).

Broiler chickens: In broiler chickens, mitochondrial function is directly linked to their performance, as healthy mitochondria are crucial for efficient energy production in muscle tissue, which is essential for optimal growth and feed conversion efficiency. However, factors like heat stress can significantly impair mitochondrial function, leading to reduced broiler performance and potential meat quality issues (Bottje *et al*., 2006; Shakeri *et al*., 2023). Studies have shown a correlation between mitochondrial function and feed efficiency (Algothmi *et al*., 2024; Bottje *et al*., 2006) and meat quality (Zhang *et al*., 2023), with broilers exhibiting better feed conversion ratios having more efficient mitochondria. Mitochondrial dysfunction can contribute to meat quality issues like woody breast (Shakeri *et al*., 2024) and spaghetti (Shakeri *et al*., 2024) in broilers, characterized by tough and fibrous muscle texture or broken fibers. Heat stress is a major concern for broiler production, as it can significantly impair mitochondrial function and lead to reduced growth rates (Algothmi *et al*., 2024).

Aquatic: Mitochondria in fish are important for energy production, heart function, and thermal tolerance (John *et al*., 2024). Mitochondrial generate heat during ROS production. Mitochondrial properties adjust during thermal acclimation, and the acclimation potential of cardiac mitochondria could be key to fish temperature tolerance/adaptation (John *et al*., 2024). Mitochondria from aquatic invertebrates show an increase in the rate of ROS efflux/formation as the assay temperature increases (Banh *et al*., 2016). Furthermore, mitochondrial internal temperature can be significantly higher than the surrounding cellular environment (Lane, 2018). This higher mitochondrial temperature acts like a "thermostatic radiator" to help maintain a stable body temperature. Additionally to thermal tolerance function and ATP production in aquatic, mitochondria are important for osmotic balance in aquatic, which means increases fish adaptation to different aquatic environments (Brijs *et al*., 2017). In fact, when cells experience osmotic stress, mitochondria initiate a rapid metabolic shift by changing glucose metabolism (suppressing pyruvate dehydrogenase) from oxidative phosphorylation to aerobic glycolysis, resulting in a decrease in oxygen consumption rate and an increase in extracellular acidification rate (Ikizawa *et al*., 2023).

Table 1: Strategies to improve mitochondria function in farm animals.

Name	Function
Alpha-lipoic acid and acetyl-L-carnitine	Helps to reduce oxidative stress
β-hydroxy-β-methylbutyrate	Enhances muscle health
N-acetylcysteine	Anti-inflammatory agent
Resveratrol	Mitigating oxidative stress
Ribonucleotide reductase	Improves mtDNA health

Potential Ways to Improve Mitochondria Function

Mitochondrial function in livestock can be improved through a variety of nutritional and pharmacological approaches (Table 1). Nutritional strategies, such as α-lipoic acid and acetyl-L-carnitine (increase mitochondrial ATP production) (Pizzorno, 2014) by reducing oxidative stress and increasing flow of oxygen-rich blood. In farm animals, studies showed that alpha-lipoic acid and acetyl-L-carnitine reduced oxidative stress while improved performance and health in goats and poultry (Li *et al*., 2019; Wang *et al*., 2017). β-hydroxy-β-methylbutyrate is a branched-chain amino acid leucine (increases mitochondrial mass, respiration capacity, and mitochondrial biogenesis) (Zhong *et al*., 2019) by increasing oxidative metabolism and gene expression of mitochondrial biogenesis. In farm animals, β-hydroxy-β-methylbutyrate enhanced muscle health, improve performance and immunity (Szcześniak *et al*., 2015). N-acetylcysteine (improves mitochondrial function) (Lapointe, 2014) by reducing oxidative stress and supporting cellular repair mechanisms, and antioxidants (improve mitochondrial efficiency) (Lowes *et al*., 2013) by reducing ROS production. In farm animals, N-acetylcysteine has antioxidant properties and acts as an anti-inflammatory agent (Ninković *et al*., 2024). Pharmacological strategies such as resveratrol (enhances mitochondrial biogenesis) (Ungvari *et al*., 2011) by regulating mitochondria. In farm animals, resveratrol as an antioxidant, showed improved health, growth, and meat quality in livestock, mitigating oxidative stress as well as improved various physiological functions (Meng *et al*., 2023). Ribonucleotide reductase activity (improve mitochondria function) (Shakeri *et al*., 2023) by improving mtDNA health. In poultry, reduction in ribonucleotide reductase activity showed to have adverse effects on meat quality (Shakeri *et al*., 2024).

CONCLUSIONS AND RECOMMENDATIONS

Mitochondria as a source of energy are essential for having functional cells. Malfunctioning mitochondria may increase oxidative damage in tissue resulting in deteriorating the quality of products. Environmental factors such as heat stress have major impact on mitochondria function. Although, many solutions have been introduced to improve mitochondria function, it seems nutritional modifications might be good an approach so far to cope with the problem but not a complete solution. Therefore, further investigations will be required to provide more reliable strategies such as genetics modifications (maybe edit mtDNA) and controlling environmental factors to have better outcomes. In short, mitochondrial-targeted therapies may bridge the gap between intensive farming and animal welfare.

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NOVELTY STATEMENTS

The novelty of this review lies in its specific focus on the causal link between environmental stress-induced mitochondrial impairment and its downstream negative effects on key animal production traits, paving the way for targeted mitigation strategies.

AUTHOR'S CONTRIBUTIONS

Mohsen Shakeri: Writing – original draft.
Majid Shakeri and Hieu Le: Writing – review and editing.

Ethical Statement

As this manuscript does not involve research on humans or animals, nor does it include vulnerable populations, an ethical statement is not applicable.

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Ethics Approval

Not applicable.

Consent for Publication

All the authors have read and approved the work.

Data Availability Statement

All data have been included in the article or referenced in the article.

Conflict of Interest

Authors declare no conflict of interest.

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