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*Mini Review*

# Innate Immune Signalling via Mitochondria

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**Abstract**

Organelles like mitochondria can serve a variety of purposes. In addition to fulfilling the traditional function of providing the cell with energy, mitochondria actively control innate immune responses to sterile and viral stimuli. When produced or exposed in response to dysfunction or damage, mitochondrial components can be directly recognised by innate immune system receptors and cause an immunological response (Edem et al., 2012). Additionally, even though their mitochondrial regulation may not be necessary for their start, many innate immune responses nonetheless depend on or require mitochondria for specific steps of their signalling cascades. The precise immune response is modulated, and the direction and nature of an innate immune cell's response to stimuli are shaped by mitochondrial metabolites and the metabolic state of its mitochondria. Together, these mechanisms cause mitochondria to regulate innate immune responses in a complex and targeted manner (Idowu et al., 2016).

**Keywords:** ASC, ASK1, Apoptosis signal-regulating kinase 1, ATP, Adenosine tri-phosphate, CAPS, Cryopyrin associated periodic syndromes, CARD, Caspase activation and recruitment domain, CL, Cardiolipin, CLR, C-type lectin receptor, CREB, cAMP response element binding protein, Cgas, Cyclic GMP-AMP synthase

## INTRODUCTION

According to the endosymbiotic idea, the original bacterial endosymbiont's DNA has mostly been lost or moved to the nucleus, leaving the modern mitochondrial DNA (mtDNA), a considerably smaller (16 kb in mammals), circular molecule. In budding yeast, the ability of mtDNA to leave mitochondria and integrate into the nuclear genome, as well as the genes that control this process, was found. It is now understood that numerous innate immune signalling pathways are activated when mitochondrial DNA (mtDNA) is released into the cytoplasm, outside of the cell, or into circulation (Ozer et al., 2008). Mitochondria have emerged as important regulators of innate immunity. Here, we first go over the mechanisms—which include a number of inducible mitochondrial pores and impaired mitophagy or autophagy—by which mtDNA is released into the cytoplasm. Next, we discuss how particular innate immune nucleic acid sensors and inflammasomes are triggered by various released mtDNA forms. Finally, we go over how human diseases, bacterial and viral infections, senescence, and ageing are related to intracellular and extracellular mtDNA release; including circulating cell-free mtDNA that promotes systemic inflammation (Wahab et al., 2008).

The evolution of mitochondria from a symbiotic organism living in the cytosol of a eukaryotic cell to an independent prokaryotic organism suggests that this association was advantageous to both parties. The mitochondrion produces energy for the cell, and the cell provides the mitochondrion with resources and protection. The idea that mitochondria have a bacterial origin fits in well with research showing that when exposed, the special mitochondrial components betray their prokaryotic heritage and are recognised as foreign by innate immune receptors, inducing an inflammatory response. It's intriguing to note that more recent research suggests that the importance of mitochondria to the innate immune response goes beyond only their role in identifying invasive bacteria and instead has a significant influence on a variety of different innate immune responses (Kingsley et al., 2016). The negatively charged matrix that permits the flow of electrons along the electron transport chain, which is composed of specialised complexes organised on the inner

mitochondrial membrane, is necessary for mitochondria to produce ATP. Antioxidants, substrates for oxidative phosphorylation, and membrane uncoupling agents are just a few of the stresses that can cause the matrix's negative charge to become disrupted. The failure of ATP synthesis and the formation and release of reactive oxygen species (ROS), which have the ability to inflict extensive damage, are caused by this loss of negative potential. Additionally, injured cells' leaking of previously contained mitochondrial components into the cytosol or the circulation due to defective mitochondria that have lost membrane integrity (Saif et al., 2015).

# DISCUSSION

Stem cells have been suggested for use in regenerative medicine over the past ten years, with mesenchymal stem cells (MSCs) garnering the majority of the attention due to their therapeutic potential in tissue engineering and regenerative medicine. When MSCs were first used therapeutically in regenerative medicine, they were delivered directly to the wounded tissue and differentiated into several functional cell types, which facilitated tissue repair and regeneration. The second method was tissue engineering, which created a tissue structure by fusing stem cells or differentiated cells with a biodegradable framework (Ogori et al., 2016). MSCs taken from bone marrow have been used in the majority of clinical trials. It is important to note that MSCs obtained from human dental pulp tissue have also been used as a regenerative medicine technique in clinical studies. Clinical data continue to demonstrate the efficacy of MSC applications, and The National Institutes of Health's database, www.clinicaltrials.gov (accessed on 1 November 2021), has a list of completed experiments. Reports also indicate that MSCs derived from various sources are successful in tissue regeneration, although their use in regenerative medicine is currently constrained by a number of significant barriers (Ashaye et al., 2006).

One of the main reasons for the loss of tooth-supporting tissues is inflammatory periodontal disease. In-depth study is being done on novel methods for periodontal apparatus regeneration. The employment of appropriate regenerative cells, transported through a suitable scaffold, and guided by signalling molecules is implied by periodontal tissue engineering. An increasing number of studies on dental tissue engineering have utilised dental pulp stem cells (Wahab et al., 2008). These cells have mesenchymal (stromal) stem cell-like traits, such as the capacity for self-renewal and multilineage differentiation, in addition to being relatively accessible and easy to handle. This article's goal is to cover the scientific underpinnings of periodontal tissue engineering as well as the difficulties in creating a reliable and clinically useful platform for tissue regeneration. An updated review of dental pulp is provided in this piece (Kingsley et al., 2016).

# CONCLUSION

At both the cellular and organismal levels, mitochondria have established themselves as controllers of numerous aspects of mammalian function from their rather humble beginnings as ancient bacteria. In addition to precisely and delicately controlling innate immune activation and signalling, this regulation goes much beyond simply supplying vital bioenergetics. New paths via which these ostensibly separate systems are actually connected are likely to be revealed as a result of developments in our knowledge of the role mitochondria play in the innate immune response and in our ability to investigate the mitochondria.

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