



Significant Role of Mitochondrial-Associated Endoplasmic Reticulum Membrane in Cellular Activities

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DESCRIPTION

The outer mitochondrial membrane and the endoplasmic reticulum membrane are separated by the Mitochondrial-Associated Membrane (MAM). The mitochondrial associated membrane is involved in many different cellular processes, including as calcium signalling, mitochondrial division and fusion, endoplasmic reticulum stress, lipid synthesis, and transport. The MAM is involved in the pathophysiology of Diabetic Nephropathy (DN), according to recent investigations.

In the following decades, it is expected that the number of people with Diabetes Mellitus (DM) would rise quickly, reaching 642 million by 2040. One of the most prevalent and harmful chronic consequences of DM is Diabetic Nephropathy (DN). About 30% of people with type 1 diabetes (T1DM) and 40% of patients with type 2 diabetes (T2DM) experience this microvascular complication. Epidemiological research indicates that DN is the main global contributor to End-Stage Renal Disease (ESRD). DN is a significant but underappreciated burden on world public health. According to several studies, people with DN have 10-year mortality rates that are comparable to the average mortality rates for all cancers. Hence, there is a compelling case for studying DN.

Glomerular hyperfiltration, increasing albuminuria, a reduced GFR, and ultimately ESRD are all symptoms of DN disease progression. Glial hypertrophy, mesangial cell proliferation and hypertrophy, thickness of the Glomerular Basement Membranes (GBM) and Tubular Basement Membranes (TBM), glomerulosclerosis, tubulointerstitial inflammation, and renal fibrosis are some of the pathological alterations.

The exact DN mechanism has been thoroughly investigated. The production of advanced glycosylation products, oxidative stress, endoplasmic reticulum stress, inflammatory responses, alterations in the kidney's hemodynamics, and problems of lipid metabolism are typically the main causes and contributors to the pathogenesis of DN. These processes aid in the development of

Kimmelstiel-Wilson lesions, thickening of the GBM and TBM, damage and deletion of the podocytes, and interstitial fibrosis. It is important to note that numerous factors, rather than just one, are thought to be involved in the beginning of DN. Researchers are now aware that the mitochondria-associated endoplasmic reticulum membrane also contributes significantly to the development of DN.

The Endoplasmic Reticulum (ER) and mitochondria are crucial eukaryotic cell organelles that work in tandem to carry out cellular processes in humans. The bioenergetic and biosynthetic organelles known as mitochondria are also referred to as "power stations." They can offer a consistent source of energy for human endeavours. They also serve as a platform for producing building blocks through biosynthesis. Moreover, because it is in charge of protein and lipid synthesis as well as their modification and processing, the ER is referred to as the "base station of protein and lipid synthesis." The ER is an intriguingly active intracellular organelle. The ER's structure and function change depending on what the cell is doing at any given time.

The MAM, which is made up of the ER and a small portion of the outer mitochondrial membrane, is found in many different cell types. The MAM is a morphological adaptation that facilitates communication between the mitochondria and the ER, according to a number of studies. Cellular activities benefit greatly from MAM. Its involvement in calcium signalling, lipid production and trafficking, the ER stress response, dynamic alterations, and mitochondrial autophagy is being supported by more and more research.

Calcium signaling

The maintenance of calcium homeostasis is essential for cellular functions. Cell death is a result of dysregulated calcium levels, which also lead to other physiological problems. The ER and mitochondria both play important roles in maintaining calcium homeostasis. Normally, the ER is where calcium is kept. Calcium is released from the ER to the cytoplasm in response to cellular activation, where it is absorbed by the mitochondria.

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Lipid biosynthesis and trafficking

Lipids are crucial elements of cell membranes that play a role in energy storage, signal molecule transmission, and the creation of chemicals with biological activity. While some lipid alterations take place in the mitochondria, the majority of lipids are synthesised in the ER. Lipid is transferred from the ER to the mitochondria with the help of the MAM.

Er stress response

The maintenance of ER homeostasis is crucial for cellular functions. In DN, an ER stress response known as the Unfolded Protein Reaction (UPR) has been noted. Inositol-Requiring Enzyme 1 (IRE1), Protein Kinase Rna-Like Kinase (PERK), and activating transcription factor 6 are the three primary components that the activated UPR activates (ATF6). According to a prior study, the MAM is reduced in the absence of PERK, which weakens the ER stress-induced apoptosis. Moreover, the efficiency of IP3R, which aids in the transfer of calcium from the ER to the mitochondria, can be controlled by IRE1 in the MAM.

Dynamic change and autophagy in mitochondria

Being dynamic organelles, mitochondria move along the cytoskeleton while continuously undergoing fission and fusion.

Acute renal injury and cardiac ischemia/reperfusion injury are both caused by mitochondrial damage. Bax Inhibitor-1 (BI1) was shown by Wang et al. to be a key regulator of renal tubular function by maintaining prohibitin 2's mitochondrial location (PHB2). In general, an abundance of nutrients causes mitochondria to divide, whereas hunger causes them to fuse.

Dynamin-related GTPases are responsible for controlling mitochondrial fusion and division. Mfn1 and Mfn2 mediate the fusion of the OMM. Opa1 and Mgm1 control how the Mitochondrial Inner Membrane (MIM) fuses. Opa1 is an optical atrophy protein. In addition, Dynamin-Related Protein 1 (Drp1) regulates mitochondrial division. Consequently, the MAM-based dynamin-related GTPase controls dynamic alterations in the mitochondria. DN has surpassed all other microvascular complications to become the main global cause of ESRD.

The function of mitochondria in DN was previously regulated by Phosphatase and Tensin Homolog (PTEN)-Induced Kinase 1 (PINK1), and targeting PINK1 may be a viable therapeutic approach. The MAM has structural properties and is a very plastic structure. The MAM houses a number of chemicals, enzymes, and chaperones. Consequently, greater MAM production is engaged in cellular activities under pathological situations such as hyperglycemia, ER stress, and inflammation.