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Editorial

Mitochondria Organelles: Editorial

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Mitochondria are double membrane-bound organelles that, through the oxidation of carbohydrates, fats, and amino acids, are responsible for producing energy in cells. They are semi-autonomous organelles with a genetic material of their own, a genetic system and a genome that is limited. In addition, they have a diameter of approximately 0.5-1.0 μm.

In addition to supplying energy for cells, many important metabolic pathways are also associated with mitochondria, such as the tricarboxylic acid cycle (TCA cycle), the β-oxidation of fatty acids, and the single carbon cycle. The metabolites formed by these routes can also be used to monitor the role of the MSCs as retrograde signals. In addition, in size, number, and appearance, the mitochondria possessed by various MSCs differ. The number of mitochondria depends on the cell's metabolic level; more mitochondria are therefore present in cells with high metabolic activity.

Glycolysis, the TCA cycle, and OXPHOS are the principal mechanisms of mitochondrial energy metabolism in cells. Reduced nicotinamide adenine dinucleotide (NADH), reduced flavin adenine dinucleotide and other energetic molecules are formed by glycolysis and the TCA cycle, while OXPHOS uses these substances to reduce O2 and release energy to synthesize ATP. If a cell is in a hypoxic environment, it switches to anaerobic respiration; at this stage, the pyruvate formed by glycolysis in the mitochondria no longer enters the TCA cycle, but continues to react and is eventually reduced by NADH to fermentation products such as ethanol or lactic acid instead of ATP. In amino acids, fatty acids, and steroid metabolism, mitochondria also play essential roles.

In addition, these substrates of mitochondrial fatty acid and amino acid metabolism[23] are used for malonylation, succinylation, and glutarylation of amino acid lysine. Different stem cells may undergo a change in energy metabolism as well as various biological processes of the same cell. By altering their energy metabolism pathways, mitochondria may thus control the role of MSCs. In addition, mitochondria are essential organelles in the MSCs responsible for signal transmission. In regulating cell signals provided by ROS, calcium homeostasis, and membrane potential, they play an important role. As mitochondrial OXPHOS produces large amounts of ROS as a by-product, mitochondria are the major source of intra-cellular ROS production.

O2-, H2O2, OH-, and LOOH are included in the ROS that can include O2 free radicals in biochemical reactions and have powerful biological activities. In addition, in the respiratory chain of the mitochondrial inner membrane, NADH-CoQ oxidoreductase (complex I) and ubiquinone-cytochrome C oxidoreductase (complex III) can release electrons to generate O2-, which is the precursor of most ROS.

In addition, other metabolic intermediates, including 2-ketoglutarate dehydrogenase, pyruvate dehydrogenase, and glycerol-3-phosphate dehydrogenase, are also involved in MSC upregulation of ROS development . To maintain the activity of the MSCs, the development of ROS at the normal level is essential; however, ROS levels increase dramatically during oxidative stress in cells. Therefore, it is of great importance to remove high levels of ROS and its subsequent side effects in mitochondria by maintaining a normal physiological level of ROS to improve the function of the MSCs.

Finally, main factors such as hypoxia-inducible factor-1 alpha (HIF-1 alpha), PPARγ coactivator-1 alpha (PGC-1 alpha), sirtuin (SIRT), superoxide dismutase 2 (SOD2), protein kinase activated by adenosine 5'-monophosphate (AMPK) and uncoupling protein (UCP) also play a regulatory role in the MSC feature.

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