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Perspective

Improving Pulmonary Hypertension by Using Pyrroloquinoline Quinone

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DESCRIPTION

Pulmonary Hypertension (PH) is brought on by excessive Pulmonary Artery Smooth Muscle cell (PASMC) and Pulmonary Artery Endothelial Cell (PAEC) growth, inflammation, as well as mitochondrial and metabolic instability (PH). It is well known that the natural antioxidant Pyrrolo Quinoline Quinone (PQQ), which has anti-diabetic, neuroprotective, and cardio protective effects, encourages mitochondrial biogenesis. However, nothing is known about how it affects PH-related mitochondrial and metabolic changes, apoptosis resistance and cellular proliferation. The goal of the current study was to find out how PQQ works to treat PH. Hypoxia was utilized to generate PH-like phenotype in Human Pulmonary Artery Smooth Muscle Cells (HPASMCs), endothelial cells and primary cultured cardiomyocytes. In addition, Monocrotaline (MCT) injected Sprague Dawley (SD) rats gradually acquired pulmonary hypertension. A mitochondrial-dependent mechanism was used by PQQ administration to decrease cellular proliferation and increase apoptosis. Additionally, PQQ administration in HPASMCs reduced insulin resistance, enhanced mitochondrial bioenergetics while retaining respiratory complexes and prevented mitochondrial and metabolic dysfunctions. Altogether, the findings demonstrate that PQQ can reduce metabolic and mitochondrial abnormalities in PASMCs and can also stop rats receiving MCT from developing PH, suggesting that PQQ could be used as a possible therapeutic agent to treat PH.

A higher resting mean pulmonary arterial pressure and increased pulmonary vascular resistance are the two main symptoms of the complicated, multifactorial heterogeneous disease known as Pulmonary Hypertension (PH), which ultimately results in right ventricular failure and mortality. Active vasoconstriction and vascular remodeling caused by abnormal growth, excessive cellular proliferation, and apoptosis resistance of Pulmonary Artery Smooth Muscle Cells (PASMCs) and Pulmonary Artery Endothelial Cells (PAECs) are the primary processes in the pathophysiology of pulmonary hypertension. The study over the last few decades has shown that the proliferating cells of the pulmonary vasculature have altered cellular metabolism and mitochondrial activities, which contribute to the PH.

There have also been reports of metabolic alterations in smooth muscle cells, endothelial cells and fibroblasts from PH patients. In PH, mitochondrial abnormalities prevent cells from producing Adenosine Triphosphate (ATP) through Oxidative Phosphorylation (OXPHOS), resulting in a metabolic switch to glycolysis for ATP synthesis. Apoptosis, proliferation, angiogenesis, gene expression, inflammation, mitochondrial biogenesis and mitochondrial structure and dynamics fission/fusion are subsequently altered. The glycolytic shift, which encourages cell survival and proliferation while also evading mitochondrial-dependent apoptosis, is caused by the inhibition of Pyruvate Dehydrogenase (PDH) by HIF-dependent up-regulation of Pyruvate Dehydrogenase-Kinase (PDK). This is made possible by the hyperpolarization of the mitochondrial membrane. A PDK inhibitor called dichloroacetate inhibits PDH from being phosphorylated and inhibited, which reverses the glycolytic shift and encourages mitochondrial-dependent apoptosis. Additionally, a number of PH models have shown decreased levels of genes associated with mitochondrial mass and biogenesis, including proliferator-activated receptor. The development of PH may be influenced by anomalies in mitochondrial and metabolic activities, and blocking or rectifying these abnormalities might be a treatment focus for PH. (PPAR)-co-activator. The development of PH may be influenced by anomalies in mitochondrial and metabolic activities and blocking or rectifying these abnormalities may be a treatment focus for PH. Pyrroloquinoline quinone has been shown in preclinical and clinical research to have preventive effects against a variety of diseases, including cardiovascular ailments, making it an important component of human nutrition.

Bacterial quinoprotein dehydrogenases contain PQQ, a redox component that resembles a vitamin that is not covalently bound. It is a potent, naturally occurring antioxidant that can be found in minute amounts in human milk, soil and plants. Studies have demonstrated that excluding PQQ from a mammalian diet causes issues with immune system functioning. In the presence of reductants, PQQ neutralizes Reactive Oxygen

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Species (ROS) in bacteria and guards against oxidative stress while also significantly increasing antioxidant capability in humans even after a single dose. Additionally, it increases energy generation by involving important enzymes and enhances mitochondrial biogenesis. PQQ has been demonstrated to improve cardiac remodeling and cell hypertrophy, reduce cardiac pressure-overload-induced mitochondrial dysfunction and prevent the onset of chronic heart failure.

Together, these studies show that PQQ is cardio-protective and can control problems associated to mitochondria and metabolism. However, the impact of PQQ on metabolic and mitochondrial changes in PH has not yet been investigated. In order to determine if PQQ administration may impact on mitochondrial activity, OXPHOS, energy metabolism, and proliferation in experimental PH models. The study's findings demonstrated that PQQ therapy improved mitochondriogenesis in hypoxic HPASMCs and decreased metabolic and mitochondrial abnormalities. In hypoxic HPASMCs, PQQ therapy decreased mitochondrial and metabolic defects and enhanced mitochondriogenesis.