a fórmula

PQQ *(Pirroloquinolina Quinona)*

A coenzima antioxidante mitocondrial mais potente que a Vitamina C

Estudos

http://aformulabr.com.br/qrcode/pqqafv01.pdf

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DESCRIÇÃO

Vitamina hidrossolúvel do complexo B presente em diversos alimentos (soja fermentada, kiwi, salsa, espinafre, etc.) assim como nos tecidos humanos atuando como coenzima de oxirredução dotada de grande estabilidade molecular capaz de realizar milhares de transferências de elétrons.

MECANISMO DE AÇÃO

O **PQQ** possui propriedades vitamínicas e antioxidantes associadas a funções de reparo cognitivo com importante papel no processo de envelhecimento, proteção das células nervosas e na estimulação natural dos níveis energéticos ligados à concentração. Nos idosos, as mitocôndrias são menos numerosas resultando numa perda de energia em problemas cognitivos e na degradação celular acelerada. O **PQQ** aumenta o número de mitocôndrias ativando os genes que controlam a sua reprodução, mesmo no interior das células senescentes, e auxilia à saúde cardiovascular ao estimular a função do músculo cardíaco. Além disso, estimula a utilização do oxigênio celular e protege as membranas celulares do estresse oxidativo provocando um poder antioxidante 100 vezes superior ao da vitamina C. A privação de **PQQ** resulta na deficiência do sistema imune reduzindo os níveis de interleucina-2, necessária para o desenvolvimento da memória imunológica das células T5, justificando sua importância sob a forma de suplementação.

INDICAÇÕES

- ✓ Antioxidante; Neuroprotetor;
- ✓ Função cognitiva;
- ✓ Síntese mitocondrial; Anti-aging;
- ✓ Sistema imunológico;
- ✓ Proteção isquêmica a tecidos cardíacos.

DOSE USUAL

Recomendação oral de 10 a 20mg de **PQQ (Pirroloquinolina Quinona)** ao dia.

SUGESTÕES DE FÓRMULAS

PQQ (Pirroloquinolina Quinona).....10mg

Modo de uso: 1 dose, 2 vezes ao dia, antes ou durante as refeições.
Indicação: antioxidante, cardioprotetor e neuroprotetor.

PQQ (Pirroloquinolina Quinona).....10mg
Magnésio quelato.....80mg
Glycoxil®.....50mg
SiliciuMax® pó.....100mg

Modo de uso: 1 dose, 2 vezes ao dia.
Indicação: prevenção do envelhecimento cerebral.

PRINCIPAIS REFERÊNCIAS

CHOWANADISAI, W. et. al. Pyrroloquinoline Quinone Stimulates Mitochondrial Biogenesis through cAMP Response Element-binding Protein Phosphorylation and Increased PGC-1α Expression. **J Biol Chem**. v. 285, n.1, p. 142-152, Jan 2010. Disponível em: < http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2804159/> Acesso em: 03 de Agosto de 2017, às 15:10.

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a fórmula

PQQ (Pirroloquinolina Quinona)

ESTUDOS CLÍNICOS

Pyrroloquinoline-quinone and its versatile roles in biological processes

Pyrroloquinoline-quinine (PQQ) was initially characterized as a redox cofactor for membrane-bound dehydrogenases in the bacterial system. Subsequently, PQQ was shown to be an antioxidant protecting the living cells from oxidative damage in vivo and the biomolecules from artificially produced reaction oxygen species in vitro. The presence of PQQ has been documented from different biological samples. It functions as a nutrient and vitamin for supporting the growth and protection of living cells under stress. Recently, the role of PQQ has also been shown as a bio-control agent for plant fungal pathogens, an inducer for proteins kinases involved in cellular differentiation of mammalian cells and as a redox sensor leading to development of biosensor. Recent reviews published on PQQ and enzymes requiring this cofactor have brought forth the case specific roles of PQQ. This review covers the comprehensive information on various aspects of PQQ known till date. These include the roles of PQQ in the regulation of cellular growth and differentiation in mammalian system, as a nutrient and vitamin in stress tolerance, in crop productivity through increasing the availability of insoluble phosphate and as a bio-control agent, and as a redox agent leading to the biosensor development. Most recent findings correlating the exceptionally high redox recycling ability of PQQ to its potential as anti-neurodegenerative, anticancer and pharmacological agents, and as a signalling molecule have been distinctly brought out. This review discusses different findings suggesting the versatility in PQQ functions and provides the most plausible intellectual basis to the ubiquitous roles of this compound in a large number of biological processes, as a nutrient and a perspective vitamin.

Pyrroloquinoline Quinone Stimulates Mitochondrial Biogenesis through cAMP Response Element-binding Protein Phosphorylation and Increased PGC-1α Expression

Abstract: Bioactive compounds reported to stimulate mitochondrial biogenesis are linked to many health benefits such increased longevity, improved energy utilization, and protection from reactive oxygen species. Previously studies have shown that mice and rats fed diets lacking in pyrroloquinoline quinone (PQQ) have reduced mitochondrial content. Therefore, we hypothesized that PQQ can induce mitochondrial biogenesis in mouse hepatocytes. Exposure of mouse Hepa1–6 cells to 10–30 μM PQQ for 24–48 h resulted in increased citrate synthase and cytochrome *c* oxidase activity, Mitotracker staining, mitochondrial DNA content, and cellular oxygen respiration. The induction of this process occurred through the activation of cAMP response element-binding protein (CREB) and peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α), a pathway known to regulate mitochondrial biogenesis. PQQ exposure stimulated phosphorylation of CREB at serine 133, activated the promoter of PGC-1α, and increased PGC-1α mRNA and protein expression. PQQ did not stimulate mitochondrial biogenesis after small interfering RNA-mediated reduction in either PGC-1α or CREB expression. Consistent with activation of the PGC-1α pathway, PQQ increased nuclear respiratory factor activation (NRF-1 and NRF-2) and Tfam, TFB1M, and TFB2M mRNA expression. Moreover, PQQ protected cells from mitochondrial inhibition by rotenone, 3-nitropropionic acid, antimycin A, and sodium azide. The ability of PQQ to stimulate mitochondrial biogenesis accounts in part for action of this compound and suggests that PQQ may be beneficial in diseases associated with mitochondrial dysfunction.

Antioxidant and pro-oxidant properties of pyrroloquinoline quinone (PQQ): implications for its function in biological systems

Abstract: Pyrroloquinoline quinone (PQQ) is a novel redox cofactor recently found in human milk. It has been reported to function as an essential nutrient, antioxidant and redox modulator in cell culture experiments and in animal models of human diseases. As mitochondria are particularly susceptible to oxidative damage we studied the antioxidant properties of PQQ in isolated rat liver mitochondria. PQQ was an effective antioxidant protecting mitochondria against oxidative stress-induced lipid peroxidation, protein carbonyl formation and inactivation of the mitochondrial respiratory chain. In contrast, PQQ caused extensive cell death to cells in culture. This surprising effect was inhibited by catalase, and was shown to be due to the generation of hydrogen peroxide during the autoxidation of PQQ in culture medium. We conclude that the reactivities of PQQ are dependent on its environment and that it can act as an antioxidant or a pro-oxidant in different biological systems.

Potential physiological importance of pyrroloquinoline quinone

Abstract:Pyrroloquinoline quinone (PQQ) is a novel biofactor for which a proposition can be made for physiological importance. PQQ was first recognized as an enzyme cofactor in bacteria. It has recently been tentatively identified as a component of interstellar dust. Thus, PQQ may have been present throughout early biological conception and evolution. PQQ is also a potent plant growth factor. Consequently, for animals and humans, there has been constant exposure to PQQ. In animals, PQQ is reported to participate in a range of biological functions with apparent survival benefits (e.g., improved neonatal growth and reproductive performance). There are also benefits from PQQ supplementation related to cognitive, immune, and antioxidant functions, as well as protection from cardiac and neurological ischemic events. Although PQQ is not currently viewed as a vitamin, its involvement in cell signaling pathways, particularly those important to mitochondriogenesis in experimental animal models, may eventually provide a rationale for defining PQQ as vital to life. For humans, such evidence suggests there may be similar parallels or benefits from improving PQQ status.

Altering Pyrroloquinoline Quinone Nutritional Status Modulates Mitochondrial, Lipid, and Energy Metabolism in Rats

We have reported that pyrroloquinoline quinone (PQQ) improves reproduction, neonatal development, and mitochondrial function in animals by mechanisms that involve mitochondrial related cell signaling pathways. To extend these observations, the influence of PQQ on energy and lipid relationships and apparent protection against ischemia reperfusion injury are described herein. Sprague-Dawley rats were fed a nutritionally complete diet with PQQ added at either 0 (PQQ−) or 2 mg PQQ/Kg diet (PQQ+). Measurements included: 1) serum glucose and insulin, 2) total energy expenditure per metabolic body size (Wt3/4), 3) respiratory quotients (in the fed and fasted states), 4) changes in plasma lipids, 5) the relative mitochondrial amount in liver and heart, and 6) indices related to cardiac ischemia. For the latter, rats (PQQ− or PQQ+) were subjected to left anterior descending occlusions followed by 2 h of reperfusion to determine PQQ's influence on infarct size and myocardial tissue levels of malondialdehyde, an indicator of lipid peroxidation. Although no striking differences in serum glucose, insulin, and free fatty acid levels were observed, energy expenditure was lower in PQQ− vs. PQQ+ rats and energy expenditure (fed state) was correlated with the hepatic mitochondrial content. Elevations in plasma di- and triacylglyceride and β-hydroxybutryic acid concentrations were also observed in PQQ− rats vs. PQQ+ rats. Moreover, PQQ administration (i.p. at 4.5 mg/kg BW for 3 days) resulted in a greater than 2-fold decrease in plasma triglycerides during a 6-hour fast than saline administration in a rat model of type 2 diabetes. Cardiac injury resulting from ischemia/reperfusion was more pronounced in PQQ− rats than in PQQ+ rats. Collectively, these data demonstrate that PQQ deficiency impacts a number of parameters related to normal mitochondrial function.

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