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Pyrroloquinoline Quinone (PQQ) and Rheumatic Diseases

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ABSTRACT

Introduction: Pyrroloquinoline quinone (PQQ) is a redox cofactor with antioxidant and immunomodulating actions implicated in protection of mitochondria and modulation of inflammation pathways. Oxidative stress is centrally involved in the onset of rheumatic diseases, such as RA, thus PQQ may be considered as a candidate for new therapeutic strategies.

Objective: This article is aimed to objectively and critically analyse the scientific evidence available about PQQ in rheumatic diseases.

Methods: A systematic review was performed in the PubMed, Scielo and LILACS databases with no language restriction, including articles published from January 1965 to May 2024. Studies using in vivo and human samples published in English were only included, but separate examination of in vitro and animal model studies.

Results: There is no clinical evidence for PQQ in patients with rheumatic diseases. PQQ at the preclinical level In one preclinical study, it was reported that PQQ could downregulate inflammatory cytokines and matrix-metalloproteinase expression in synovial fibroblasts and ameliorate arthritis progression in a collagen-induced arthritis mouse model (23).

Conclusions: Although experimental models suggest PQQ shows promise, there is no clinical evidence that it benefits patients with rheumatic diseases. Its potential as a therapeutic target requires further validation using translational research and clinical trials.

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INTRODUCTION

Pyrroloquinoline quinone (PQQ) is a natural redox cofactor, with many biological functions such as antioxidant action and cellular energy metabolism. Initially characterized in bacteria, PQQ has been also detected in mammalian tissues, and its presence is relevant to mitochondrial defense as well as modulation of oxidative stress, pathogenic factors associated with common chronic diseases including cardiovascular, neurodegenerative and autoimmune diseases [1,2].

These chronic inflammatory and tissue-destructive conditions are typified by rheumatic diseases, including rheumatoid arthritis (RA), in which activated synovial fibroblasts produce pro-inflammatory cytokines, such as tumor necrosis factor (TNF)-α, interleukin (IL)-1β, IL-6 and matrix-degrading enzymes [matrix metalloproteinases (MMPs)], leading to progressive joint damage [3,4]. Oxidative stress is the major contributor in the maintenance of inflammation and joint destruction in these diseases.

The recent experiment had indicated that PQQ might modulate the key inflammation pathway such as NF-κB and MAPK, cytokine production and the contraction of MMPs, and therefore therapeutic Application value in immunoassociated – diseases like RA [5] may maintain. The aim of this mini review is to analyse the extant scientific evidence and potential research gaps.

METHODS

This review was conducted in accordance with PRISMA 2020 guidelines [6]. We performed a systematic search in the PubMed, Scielo, and LILACS databases (beginning January 1965 and through May 2024). The search terms were "pyrroloquinoline quinone," "PQQ," "rheumatoid arthritis," “systemic lupus erythematosus,” psoriatic arthritis,” “ankylosing spondylitis” and “Rheumatic diseases.”

We referred to clinical studies of PQQ that used patients with rheumatic diseases as subjects. In vitro and animal-model-related studies were examined as separate categories. Review articles and duplicate reports were further excluded.

RESULTS

The preliminary search resulted in 178 papers. After this screening process, no clinical trials investigating PQQ in patients suffering from rheumatic diseases were identified. One single experimental study specifically evaluated the impact of PQQ in RA. In IL-1β-induced synovial fibroblasts, PQQ markedly decreased the expression of MMP-1 and MMP-3 as well as TNF-α and IL-6 secretion. In the CIA murine model, mice administered with PQQ showed a reduction in clinical and histological scores which was correlated with the suppression of NF-κB and MAPK pathways [5].

No studies on PQQ for other autoimmune rheumatic diseases, including systemic lupus erythematosus (SLE), psoriatic arthritis, or spondyloarthropathies were found..

DISCUSSION

Based on our review, the clinical studies of PQQ in rheumatic diseases are still not available although there is encouraging preclinical evidence. In the experimental model of arthritis, PQQ exerted considerable anti-inflammatory effects and diminished cytokine and metalloproteinase production, which implies also joint tissue protection [5].

Oxidative stress is considered an important contributor in the pathogenesis of RA and other autoimmune diseases by stimulating synovial cell activation and production of inflammatory mediators [3,4]. Due to PQQ’s ability to induce mitochondrial biogenesis (70, 71) via activation of PGC-1a and NRF1, as well as its antioxidant action, the effects on attenuating chronic inflammation are likely also profound [7]. These mechanisms support translational studies of oxidative stress, mitochondrial function and inflammation-related markers in patients.

Another possible application is in disorders characterized by a pathogenesis involving mitochondrial dysfunction, like systemic lupus erythematosus and inflammatory myopathies, where PQQ has been reported to exert beneficial effects in models of cardiovascular and neurodegenerative diseases [2].

PQQ is an encouraging antioxidant and anti-inflammatory molecule that may be applicable in the treatment of rheumatic conditions. Pre-clinical data – in particular in RA - indicate a protective effect against joint destruction. But in humans no major clinical studies yet verify its effectiveness and safety in this regard. Future research should focus on phase I/II clinical trials for dose finding, safety and potential interaction with drugs commonly used in rheumatology.

DECLARATIONS

Contributions: JFC, design, data collection, writing, data analysis, statistical analysis, and submission.

Conflict of interest: the authors declare no potential conflict of interest.

Ethics approval and consent to participate: the author declares that Helsinki's World Medical Association Declaration was followed.

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