Review A ti e e A e

Mitochondrial Inner Optic Atrophy 1 (*OPA1*) Gene is Necessary for Regulating and Activating Lysosome, Related Orphan Receptor A (*ROR-α*) genes, and *APOL1* Gene Involved in Autophagy Cells for Anti-inflammation Processes, Where *ROR-α* Genes Stored as Lysosomal Security Granules Within Autophagy Cells.

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Abstract

Tumor Necrosis Factor alpha ( ) subunits deficiency or the involvement in will lead to Sickle Cell Disease (SCD) which marked by a phenotypic variability and inflammation plays the major role in SCD pathophysiology, which linked strongly to genes expression and functions, and also linked to subunits expressions and. Activities, where subunits are so essential for anti-inflammation processes and linked to genes activities and functions, and necessary for regulation of bone homeostasis in several chronic immune and inflammatory joints and tissues diseases. The inhibition of TNFα due to inhibition or variations in genes lead d to significant inflammations improvements involved in SCD pathophysiology, and also will lead to increasing in Nuclear factor-κB pathways (NFκB) catabolic pathways and any remaining of will be involved in the NF-κB signaling pathway due to inhibition in their mitochondrial activities. The inhibition or deficiency in presence of Thymine in the Related Orphan Receptor-A (RORA) genes can reflect deficiency in mitochondrial synthetase enzyme (where mitochondrial *OPA1* gene depending on ribosomal genes activities), that'll lead to down activities in genes functions, and reductions in , TXA2, and in VEGF-A. APOL1 protein(lipoproteins) will need phospholipase enzyme for activating

APOL1 gene involved in autophagy cells which expressed from mitochondrial membrane for activating the autophagic *APOL1* gene for expressing genes from the autophagy cells for fast acting on inflammations, where that previous fact reveal "and I consider it as" that genes are stored within autophagy cells as active lysosomal security granules for fast acting on tumors and on inflammations molecules which are involved with the pathogenesis of various disorders, including cancer, neurodegeneration, and inflammatory diseases. But in the case of deficiency of mitochondrial activities or in case of inhibitions of phospholipase enzyme expressed from mitochondrial membrane will lead to un activating *APOL1* genes lead to inhibition in releasing gene from autophagy cells and then involved in tumor contents and in interstitium fluid as inactive molecules.

Ke ords: Thromboxane; Vascular endothelial gro th factor; Tumor necrosis factor alpha; *NF-* ... *C P7A* ... (*O* -) ... (-) ... –) ... *APOL1* ... (*OPA1*)

Introduction

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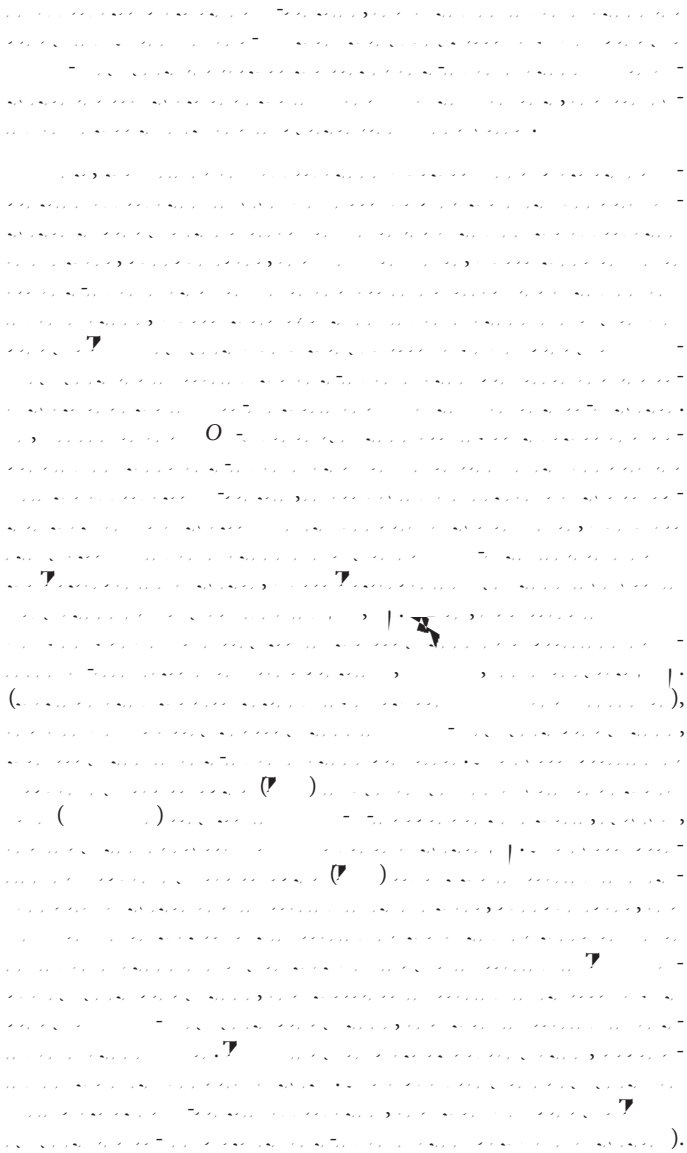

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Conflict of Interest Statement

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