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Do Mitochondria have hidden answer for RA aetiology?

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Abstract

Background: Rheumatoid arthritis (RA) is a systemic, debilitating and chronic inflammatory disorder affects 1% of the total world population. Due to absence of a defined aetiology and associated side effects of conventional medication has made obtaining permanent cure a challenge. Finding "one for all" cure is difficult due to genetic variations among populations. Deciphering genetic information, difference in regulatory network and epigenetic changes between populations can lead us to design a cure based on the principles of personalized medicine. Mitochondrial dysfunction, being a significant player in many autoimmune diseases, has also been accused for RA. Nucleotide changes can result in amino acid change, deletion or addition in enhancer, transcription factor or translation factor binding sites hampering overall function of mitochondria.

Methods: Mitochondrial DNA from peripheral blood mononuclear cells was accused as primary culprit and its genomic information was assessed through next generation sequencing (NGS) for both healthy controls and RA patients. NGS data were compared with rCRS (revised Cambridge Reference Sequence) and Indian genomic sequences to find out any variations, polymorphisms (SNPs) and heteroplasmies involved. Further, qPCR was performed to check the behaviour of several mitochondrial genes in case of both healthy controls and RA patient samples.

Results: Careful evaluation of NGS data confirmed presence of several SNPs in mitochondrial genes especially involved in OXPHOS system. Several subunits of NADH dehydrogenase and ATP synthase were found to be altered in case of RA. Similarly, expression profiles of mitochondrial genes were found to be different in RA samples when compared with healthy controls.

Conclusion: Initial investigation into mitochondrial genome confirms our suspicion of involvement of mitochondrial dysfunction in RA.

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