

A LOW MITOCHONDRIAL DNA COPY NUMBER OF LEUKOCYTES ARE ASSOCIATED WITH PROGRESSION OF METABOLIC SYNDROME

Chen-Ling Kuo², Wen-Ling Cheng², Ching-Shan Huang², Chin-San Liu^{1,2}

Department of Neurology¹ and Vascular and Genomic Center², Changhua Christian Hospital, Changhua, Taiwan

Objective:

The metabolic syndrome (MetS) is a group of risk factors of metabolic origin that are accompanied by increased risk for type 2 diabetes mellitus and cardiovascular disease. MetS is an emerging health problem that will require our immediate attention if we hope to reduce the incidence of type 2 diabetes, cardiovascular disease, and other associated disease that evolve from MetS. One of the defects in MetS and its associated diseases is excess cellular oxidative stress and mitochondrial alternation. A low copy number of mitochondrial DNA (mtDNA) has been found to be associated with enhanced risk for the development of diabetes. Therefore, the objective of this study was to investigate the characteristics of lipid metabolic molecules, redox molecules and mtDNA copy number of leukocytes in MetS.

Methods:

Seventy nine normal subjects and 42 metabolic syndrome subjects of Taiwanese were recruited and Quantitative polymerase chain reaction (QPCR) and real time PCR were applied in present study. An unpaired t test, multi-covariate ANOVA, and Spearman's rho correlation analysis were used for statistic analysis.

Results:

The results revealed that compared to normal subjects, a significant lower copy number of mtDNA in the leukocytes and higher ApoB, Homocysteine, IL 8, E-selectin, hsCRP in the plasma of MetS subjects.

Conclusion:

The present study shows that a low mtDNA copy number of leukocytes play an important role in the progression of MetS. The results may provide new diagnostic and therapeutic strategies to MetS and other associated disorder.

Funding:

The study was supported by research grant from the Changhua Christian Hospital of Taiwan.

Key words:

Metabolic syndrome, mtDNA copy number, inflammation.