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The Future of Biomarkers Tests and Genomic Medicine in Global Organ Disease

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Editorial

Interests in global organ diseases have accelerated with links between nonalcoholic fatty liver disease (NAFLD) and various chronic diseases with relevance to the metabolic syndrome and neurodegenerative diseases. Early diagnosis of global organ disease involve genomic, lipidomic and proteomic biomarker tests that may diagnose early neuron dysfunction with the prevention of various organ diseases [1]. Diet and nutrition are closely linked to accelerated aging and may allow biomarker tests to provide adequate information with relevance to the immune system dysfunction and the severity of chronic diseases. In spite of various biomarker tests and analyte measurements for chronic diseases such as obesity and diabetes abnormal nuclear-mitochondria interactions [2] persist with inflammation involved in the induction of programmed cell death.

Various diagnostic technologies have been used with relevance to genomics, lipidomics and proteomics to generate heat maps [3-5] that may allow more sensitive interpretations of cell dysfunction. Analysis of plasma lipidomic and proteome heat maps in NAFLD, obesity and diabetes are required to determine relationship between these heat maps (lipid/protein interactions) with relevance to genomic heat maps. The diagnostic technologies encompass the genome, transcriptome, proteome and metabolome (central dogma of biology, Wikipedia) and determine the cell genome and transcription factor alterations with relevance to concentrations of plasma lipids and proteins [6]. The projected cost of plasma and cell biomarker analysis is expected to cost by the year 2012 approximately 52 billion dollars [7]. Major efforts with proteomic biomarkers have identified plasma protein panels to assess progression and severity of diseases with relevant proteomic biomarkers that delay the severity of progression from mild cognitive impairment to prodromal disease and dementia [8]. Lipidomics and genomics have become important as technologies that may supersede proteomic biomarker tests with the analysis of plasma ceramides and sphingolipids that may be relevant to single gene inactivation [1,9] and multiple organ dysfunction syndrome (MODS).

Interests in genomic tests and autoimmune disease have accelerated with Sirtuin 1 (Sirt 1) inactivation associated with immunometabolism defects and related to defective heat shock protein metabolism and natural killer cell activation [10-14] linked to NAFLD. Genomic biomarkers in predictive medicine [15] must now include nuclear, cytoplasmic and plasma Sirt 1 analysis to avoid expensive diagnostic technologies (Figure 1) with biomarker analysis that do not assess the severe progression of cell disease that involve mitochondrial apoptosis. Mitophagy is now relevant to various chronic diseases such as NAFLD, obesity, diabetes and Alzheimer's disease [16-18]. Diet and nutrition have become important to the immune system and mitophagy with the correct

consumption of fat critical to maintain the nuclear-mitochondria interaction [19] with the prevention of mitochondrial apoptosis and cell death.
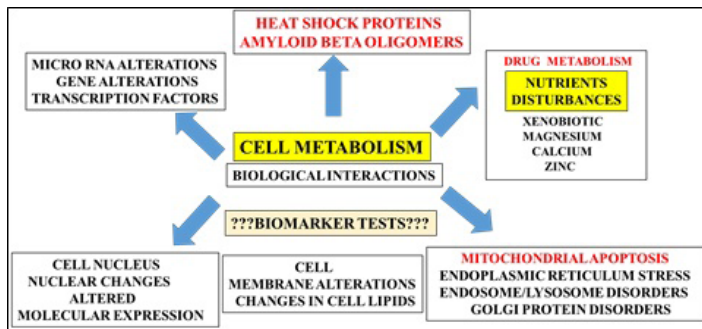

Figure 1: Various diagnostic technologies for biomarker analysis may not assess the severe progression of cell disease that involve mitochondrial apoptosis. Altered biological interactions, immune system dysfunction and cell lipid metabolism defects do not reflect the sensitivity of various biomarker test assays. Altered plasma micro RNA levels and cell transcription factors may not be connected to increased heat shock proteins that induce natural killer cell activation with mitochondrial apoptosis. Diagnostic technologies for biomarker analysis may now also need assays for LPS, xenobiotic, magnesium and calcium that are associated with nutritional disturbances.

Technologies that cost billions of dollars have become of major concern with relevance to inadvertent errors that may not allow early interpretations of disease progression that may be irreversible. In the developing world bacterial lipopolysaccharides (LPS) levels [20] should be carefully assessed to prevent repression of Sirt 1 with complete nitric oxide and immune dysregulation related to mitophagy, MODS and global organ disease [9,15,20]. Plasma heat shock protein analysis [14] is relevant to immune dysregulation and associated with inactivation of the heat shock gene Sirt 1 needs measurement to avoid unexpected errors with relevance to various biomarker tests (Figure 1). The relevance of LPS that may corrupt magnesium, calcium and albumin formulas may be require recalculation [15] with relevance to the biomarker test limitations with relevance to severity of inflammatory pathways for various chronic diseases [21]. Proteomic, lipidomic and genomic biomarker test should be carefully interpreted with relevance to individuals from the developed world that visit or stay in the developing world with mitophagy in these individuals associated with xenobiotic toxicity [22].

Conclusion
Diagnostic technologies for biomarker analysis have become important to the immune system and mitophagy to prevent early programmed cell death. Projected costs for biomarker analysis is expected to increase to billions of dollars in the next few years but altered biological and cell membrane interactions may not allow early diagnosis of immune system dysfunction related to early progression of global organ disease. In the developing world LPS and xenobiotic levels may be responsible for altered biological interactions and supersede diagnositic technologies for biomarker analysis. Inactivation of Sirt 1 by increased ceramide levels and nutritional disturbances may be responsible for increased inflammation (autoimmune disease) and mitophagy in NAFLD and global chronic disease.

References
1. Martins IJ. Early diagnosis of neuron mitochondrial dysfunction may reverse global metabolic and neurodegenerative disease. Global Journal of Medical Research. 2016; 2: 1-8.
2. Martins IJ. Unhealthy Nutrigenomic Diets Accelerate NAFLD and Adiposity in Global communities. Journal of Molecular and Genetic medicine. 2015; 9: 1-11.
3. Perez-Llamas C, Lopez-Bigas N. Gitools: analysis and visualisation of genomic data using interactive heat-maps. PLOS One. 2011; 6: e19541.
4. Chitraju C, Trötzmüller M, Hartler J, et al. Lipidomic analysis of lipid droplets from murine hepatocytes reveals distinct signatures for nutritional stress. J Lipid Res. 2012; 53: 2141–2152.
5. Key M. A tutorial in displaying mass spectrometry-based proteomic data using heat maps. BMC Bioinformatics. 2012; 13: S10.
6. Central dogma of molecular biology - Wikipedia. https://en.wikipedia.org/wiki/Central_dogma_of_molecular_biology
7. Biomarkers Market worth 53.34 Billion USD by 2021. www.marketsandmarkets.com › Press Releases.
8. Martins IJ. The Role of Clinical Proteomics, Lipidomics, and Genomics in the Diagnosis of Alzheimer's Disease. Proteomes. 2016; 4: 1-19.
9. Martins IJ. Single Gene Inactivation with Implications to Diabetes and Multiple Organ Dysfunction Syndrome. J Clin Epigenet. 2017; 3: 1-8.
10. Martins IJ. Defective Inter¬play between Adipose Tissue and Immune System Induces Non Alcoholic Fatty Liver Disease. Updates Nutr Disorders Ther. 2017; 1: 1-6.
11. Multhoff G, Mizzen L, Winchester CC, et al. Heat shock protein 70 (Hsp70) stimulates proliferation and cytolytic activity of natural killer cells. Exp Hematol. 1999; 27: 1627-1636.
12. Multhoff G. Activation of natural killer cells by heat shock protein 70. Int J Hyperthermia. 2002; 18: 576-585.
13. Specht HM, Ahrens N, Blankenstein C, et al. Heat Shock Protein 70 (Hsp70) Peptide Activated Natural Killer (NK) Cells for the Treatment of Patients with Non-Small Cell Lung Cancer (NSCLC) after Radiochemotherapy (RCTx) - From Preclinical Studies to a Clinical Phase II Trial. Front Immunol. 2015; 15: 162.
14. Martins IJ. Heat shock gene Sirtuin 1 regulates post-prandial lipid metabolism with relevance to nutrition and appetite regulation in diabetes. International Journal of Diabetes and Clinical Diagnosis. Int J Diab Clin Diagn. 2016; 3: 1-3.
15. ResearchGate.https://www.researchgate.net/profile/Ian_Martins2/info

16. García-Ruiz C, Baulies A, Mari M, et al. Mitochondrial dysfunction in non-alcoholic fatty liver disease and insulin resistance: cause or consequence? Free Radic Res. 2013; 47: 854-868.

17. Sivitz WI, Yorek MA. Mitochondrial Dysfunction in Diabetes: From Molecular Mechanisms to Functional Significance and Therapeutic Opportunities. Antioxid Redox Signal. 2010; 12: 537–577.

18. Onyango IG, Dennis J, Khan SM. Mitochondrial Dysfunction in Alzheimer's Disease and the Rationale for Bioenergetics Based Therapies. Aging Dis. 2016; 7: 201–214.

19. Martins IJ. Food intake and caffeine determine amyloid beta metabolism with relevance to mitophagy in brain aging and chronic disease. European Journal of Food Science and Technology. 2016; 4: 11-17.

20. Martins IJ. Magnesium deficiency and induction of NAFLD and Type 3 diabetes in Australasia. AMJ. 2017; 10: 235-237.

21. Bown MJ, Nicholson ML, Bell PR, et al. Cytokines and inflammatory pathways in the pathogenesis of multiple organ failure following abdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg. 2001; 22: 485-495.

22. Martins, IJ. Induction of NAFLD with Increased Risk of Obesity and Chronic Diseases in Developed Countries. Open Journal of Endocrine and Metabolic Diseases.2014; 4: 90-110.