"NUTRIGENETICS & NUTRIGENOMICS : perspectives for a POST-GENOMIC ERA"

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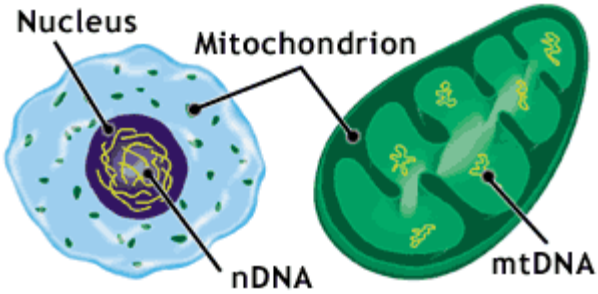
Definitions** : The term "**Nutrigenetics**" refers to the research on the impact of changes in inherited traits of n-DNA, on the response to a specific metabolic disfunctions outcomes getting health chronic damages and disorders; the term " **Nutrigenomics** " refers to the effect of specific diets, including functional food or nutraceutical supplement, on a specific **gene expression to control metabolic patways getting wellness and well-being.



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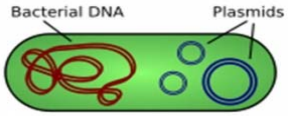
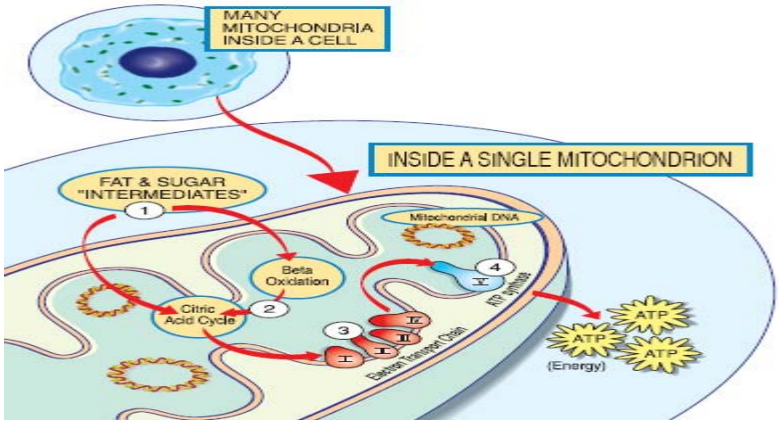
Nuclear DNA (**n-DNA**), is found in the cell's nucleus ; a different hereditary pattern e.g the mitochondrial genome (**mt-DNA**), separate from the n-DNA, is found in "***mitochondria***" that are the most prominent organelles within human cells.Normally there are about one thousand mitochondria per mammalian cell and each mitochondrion has four to five "***plasmids***".



http://bioinfo2010.files.wordpress.com/2009/07/plasmid.jpg

In each living cell mitochondria, (as enclosed-primordial bacteria) can be reproduced, semi-indipendently from n-DNA instruction , in a variable number between 1,000 to 10,000 to satisfy the various energy-production needs as other metabolic functions of different organs of the body. This means that an average about 2% to 20% percent of total human DNA is located in the mitochondria. All mt-DNA is transcribed, whereas only about 1 percent of the n-DNA is transcribed. Thus the mtDNA makes up a large fraction of the total transcribed DNA in a mammalian cell. Each of these mitochondria contains a copy of mt-DNA which is very small in comparison to the nuclear genome . So that the mt-DNA are primarily known for the central role that they play in control of generation of metabolic energy (ATP) and other important living functions. In humans (and most animals), mitochondria are exclusively inherited through the mother because eggs (and not sperm) are the major contributor of cytoplasm to the fertilized cell (Zygote) . Each of mitochondria contains a copy of the mitochondrial genome (mt-DNA) which is very small in comparison to the nuclear genome (n-DNA) ; therefore whether inherited n-DNA sporadically acquired mutations, mt-DNA is more an easy subject on mutations during their elevated rate of reproduction and "***apoptosis***" ("progammed dead").



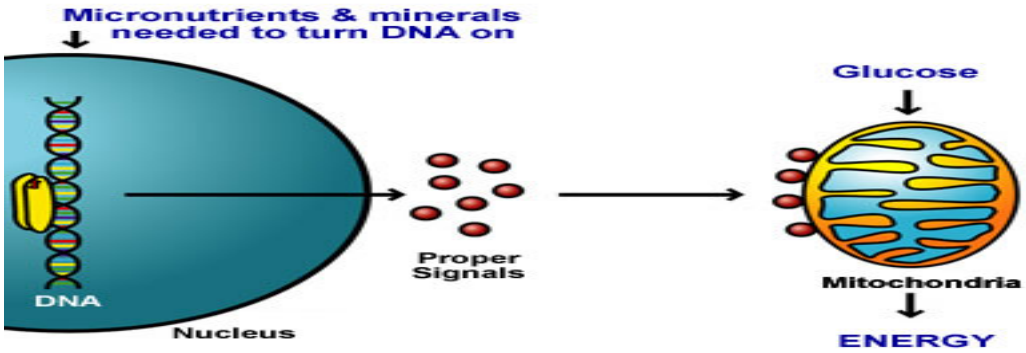
http://t3.gstatic.com/images?q=tbn:Dijf_oTbYVY1WM:http://www.gentle-interventions.org/what_are_mitochondrial_diseases_files/image003.jpg

The mitochondrion interact with the presence of oxygen, with the risk to be heavily exposed to high levels of damaging "***free radicals"***; the last are produced from a variety of sources, as the lack of antioxidants, during the breakdown of various metabolites deriving from sugar and fatty acids. So that mt-DNA alterations can occur almost anywhere during the so speedy changes of coding and decoding mitochondrial gene in the matabolic production od energy in the form of ***"ATP"*** ("adenosine triphosphate").

Remenbering that **"Nutrigenetics"** is the study that reply to how mutation of nDNA or mt-DNA can be responsible of chronic dieseases and/or alimentary syndromes. For that reason recently "Nutrigenetics" research is more strongly involved in researching ***mitochondrial diseases*** in relation to chronic disorders probably caused by mutations in the mt-DNA. Differenly **"Nutrigenomics"** is the research based on the understanding the role of metabolites in activating different pathways of expression of mt-DNA and n-DNA.

Hence to examine the changes and the modulation in gene expression that occur in the human respiratory chain based on mitochondrial activity, ***future "Nutrigenomics"*** **endeavours will be focused on the defects of interactive communication between "n-DNA and mt-DNA ",** aiming to undestand the role of the interaction and/or signal communication, as they are necessary to control in simultaneity the complex transcription of many genes , this in order to better counseling diets and improving health through developing new therapeutic approaches abling people to avoid mitochondrial diseases

The undesatanding of these interactive communications between "n-DNA and mt-DNA " may be the focus of the Nutrigenetics & Nutrigenomics future trans-disciplinay research perspectives, for getting an effective knowledge how nucleic acid information work to guide functional metabolism.

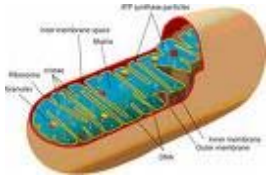


Lesions in both types of DNA can lead to cell metabolic cycles arrest, or viceversa they can cause interruption to "**n-DNA-mt-DNA** " interactions. A large number of components of food intake can directly damage both DNAs. These include carcinogens and free radicals that cause DNAs damage, either by direct reactions on the four types of bases—adenine, guanine, cytosine, and thymidine or via metabolic changes in transcription patterns, occurring via replication of the damaged DNAs till they become "***fixed mutation"*** **.** It is estimated that there are several thousand n-DNA and mtDNA alterations in each cell of the human organism per day; so that need to be a speed and continuous n-DNA and mt-DNA repairing processes through a variety of dynamic pathways of DNAs resynthesis. In particular the mt DNA does not is protected

by a chromatin structure and thus is particularly exposed to formation of oxidative DNA base lesions.

Therefore the stability and intactness of both nDNA and mtDNA is a prerequisite for normal cellular functions; there is good evidence that damage to the two types of DNA can lead to cellular dysfunction, and other diseases, or cancer and cell death. Therefore the process of **"DNA damages and repair" can be seen as a fundamentel research of Bio-physics communication**, in order to obtain an innovative conceptual reform about human heath and wellness driven through advanced "**Nutrigenetic & Nutrigenomic"** integrated studies favouring a major controllable dietary factors and micronutrients intake to obtain the reduction of occurrence of diet-related diseases and disorders including obesity and allergies.

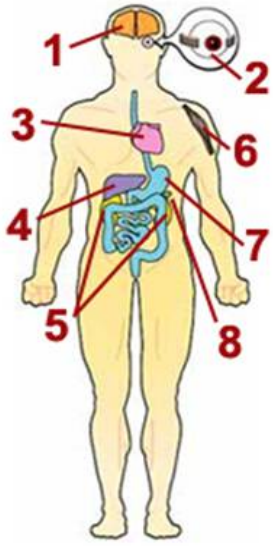
As a matter of facts the prevention or repair of DNA damage is thus a major concern in contemporarily Nutrigenetcs& Nutrigenomic integrated area ; in fact an efficient n-DNA resyntesis is very interactive mt-DNA replication and expression that direct metabolic processes of ATP-production and also is tightly linked to the all transcription process guided by a number of signal transduction pathways.



http://www.health-spy.com/mitochondrial.html

Many ***Mitochondrial disease symptoms*** can result from the disharmonies in time of functional defects in metabolism , generating a loss of coordination or interruption on the ***interactive coordination between mtDNA and the nDNA.*** This happen when dietary component's toxicity interacts with the dysfunction of ***apoptosis and Immunomodulation*** . In fact ***"Apoptosis control***" become extemely necessary when the destruction of cells considered a living threat, such as cells too old or cells with DNA damage, or infected , or cells of the immune system after they have fulfilled their functions.

In conclusion the complex coordination of alternative timing expression of both DNAs therefore get an critical evaluation of the entire biological knowledge system from an advanced nutrigenomics perspective.
To develop a transdisciplinary approach of Nutrigenomic's advanced research focused on the dietary bioactive agents and the interactive modulation of n-DNA/ mt.DNA gene expression, "**Egocreanet research group"** (www.egocreanet.it) recently proposes to build up a ***Nutrigen-Knowledge Innovation Community*** (in achronym: ***N-KIC***) to put in significant evidence that bioactive components in foods are important modifiers of risk's on heath and wellness of people . Therefore **N-KIC** alliance would favor a shared understanding of ***Bioactive Food Components and of Micronutrients*** in genetic nutrition for the promotion and dissemination of scientific and biotech-research that has an high potential impact in the development of the contemporary ***Knowledge Based Bio Economy (KBBE)***,



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