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NUTRIGENOMIC & EPIGENETIC: NEW LONGEVITY GENETIC PANELS IN CLINICAL PRACTICE.

The science of nutrition is designed to understand the role of nutrients and other dietary components in health and disease throughout the human life cycle. It is based on the so-called "knowledge base": characterization of nutrients, biochemistry and physiology of their metabolism, the signaling pathways and their role in the homeostasis of the organism. It studies errors of metabolism present at birth, even in terms of simple susceptibility and, finally, deals with the early understanding of gene-nutrient interaction mechanisms. And so we enter in the new era of medicine, called nutrigenomics. The latter can be framed as the study of interactions between the various nutrients contained in food and our genes. This way we can observe how a particular type of diet can influence gene expression and consequently affect the susceptibility of the organism against various diseases and disorders. In nutrigenomics nutrients are seen as signals that tell the body how to behave. The latter cells respond to these signals by altering the expression of genes, which affects a different protein synthesis and therefore the same metabolism. Dietary chemicals may affect gene expression directly or indirectly.

Epigenetic alter the physical accessibility to the genome by gene expression and it can condition the degree of how genes work. The epigenetic mechanisms are the basis for understanding the relationship between environmental stimuli (i.e., diet) and alteration of cell functions (i.e. metabolic properties of the cell).

Most of the common diseases are also complex diseases. It means that several genetic and environmental factors contribute to the risk of developing any of these diseases. That makes it more difficult to study the disease-gene associations and also to interpret the genotyping results. A lot of associations have been discovered during recent years thanks to new more powerful technologies and new associations between genetic variations and different common conditions are reported in scientific journals every month. Complex, multifactorial diseases tend to be the type where genotype influences the probability of an outcome, instead of acting in a deterministic way. Knowing your own personal high risk is often much more motivating to take preventive measures than knowing the general risks and what you should or should not do.

For this reason we present a new gene's risk profile panel, where the markers for each condition have been selected on the evidence available in scientific literature about the association of these markers with a given disease or condition. Genome-wide analysis is to date the most powerful method for finding genetic markers associated with different common diseases and health conditions. In addition replication studies published in journals of lower impact were also taken into account since these studies may be performed to high standards but are considered less novel. Replications that reach appropriately corrected significance and that are confirmed in samples of sufficient statistical power can often be found in journals specializing in the disease or phenotype in question. In cases of more rare disease where multiple large independent studies are not expected a single, large consortium-based study or meta-analysis was considered sufficient.

Although only markers that have shown similar results in different studies are included, the OR-s (Odds Ratios) reported generally show some variation from one study to another. In the risk calculations OR from only one study has been used. PubMed ID of that study is reported in the SNPs table after each marker. As a general rule the OR from the study with the highest statistical power to detect the observed effect (typically the study with the largest sample size) has been used, with a preference towards more recent studies.

Since linkage patterns are not identical between populations of different ethnicity, a highly associated SNP linked to an unknown causal variant in one population may not be linked to the causal variant in another. GWASs usually identify a genetic association in a sample of one ethnicity and attempt to confirm the association in samples of a different ethnicity or in large multi-ethnic cohorts. Only markers that have shown association with given condition in populations of European ethnicity (also known as white or caucasian) have been included. In some cases populations with mixed ethnicity are also considered.

The study of individual genotype may provide the clinician with dietary therapy, for the individual, can prevent or delay the onset of diseases related to food.