# NUTRITICIENOMICS

NUTRITIONAL GENOMICS

nutrigenomics

nutritional biochemistry

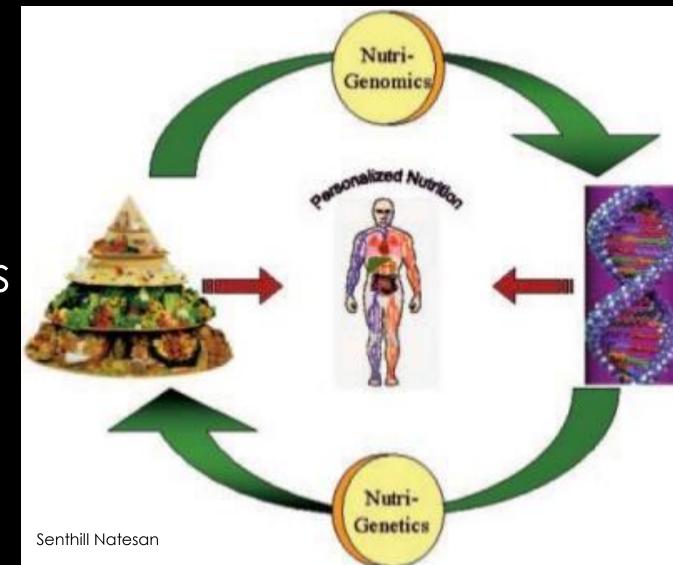
Figure 1. The three pillars of nutritional genomics: nutritional biochemistry, nutrigenetics, nutrigenomics.

**Nutritional biochemistry**: Understanding the biochemical processes and pathways driven by bioactive molecules and the disruption thereof, resulting in potential health perturbations and disease. (biochemical processes)

**Nutrigenetics**: Understanding the influence of gene variants on interactions with bioactive molecules in the molecular environment surrounding our cells and the consequences of that interaction. (gene variants on nutrient uptake)

**Nutrigenomics**: Understanding the direct or indirect effects of bioactive molecules on gene expression. (nutrient impact on gene expression)

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**Figure 2.** Bioactive food components influence gene expression in nutrigenomics. Gene variants may affect the influence of bioactive molecules in nutrigenetics.

Monogenic diseases (phenylketonuria)

A mutation in one gene is sufficient to cause a disease

nutrigenetics

Polygenic diseases

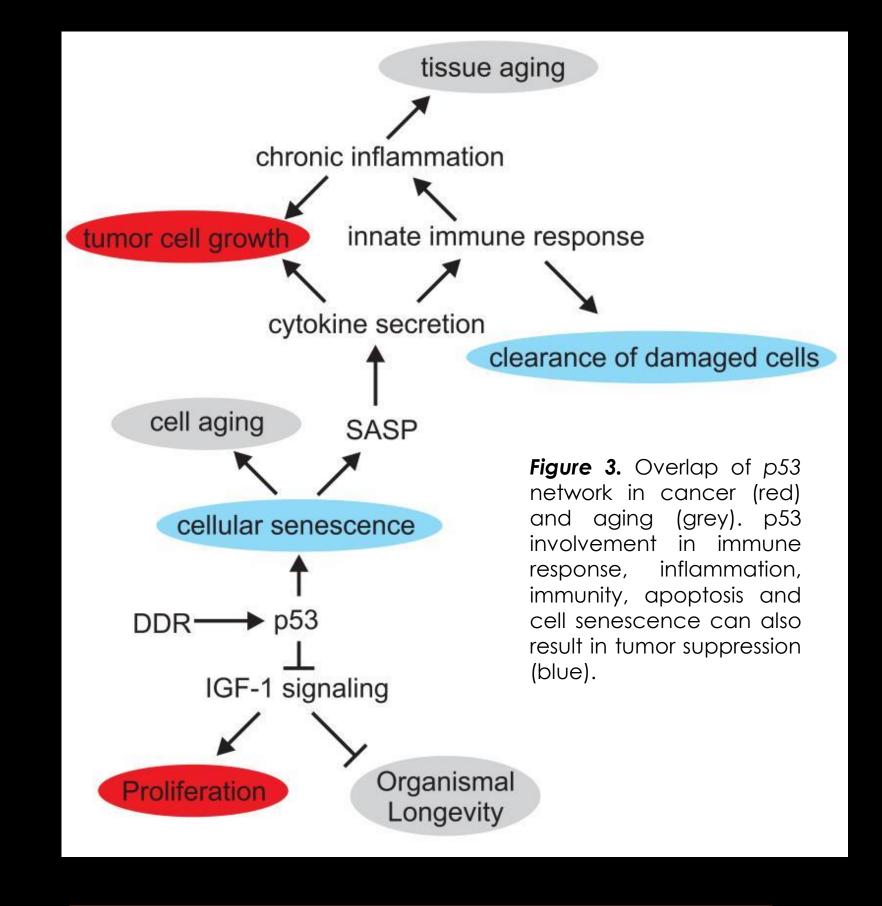
(cancer, obesity, cardiovascular disease, diabetes)

Dysfunction in a cascade of genes leads to disease

nutrigenomics

Dietary constituents participate in regulation of gene expression by modulating...

- Transcription factors
- DNA methylation
- Oxidative stress
- DNA integrity
- DNA repair
- Cellular functions, including
- Inflammation
- Telomerase activity



Other factors influencing disease and aging:

MICROBIOME

Massive complexity in discovering

interrelations by the presence of ~22,000 genes, 8–10 million polymorphisms, and daily exposures to >25,000 bioactive food components.

#### Tumor suppressor gene p53

Involved in cancer through its ability to respond to a variety of stressors including cell-cycle arrest, apoptosis, or senescence. Also involved in aging through mediation of the connection between telomere and mitochondrial dysfunctions.

- p53-dependent pathways regulated by cruciferous vegetables, spices such as ginger
- high consumption of red meat are associated with p53 disease pathway

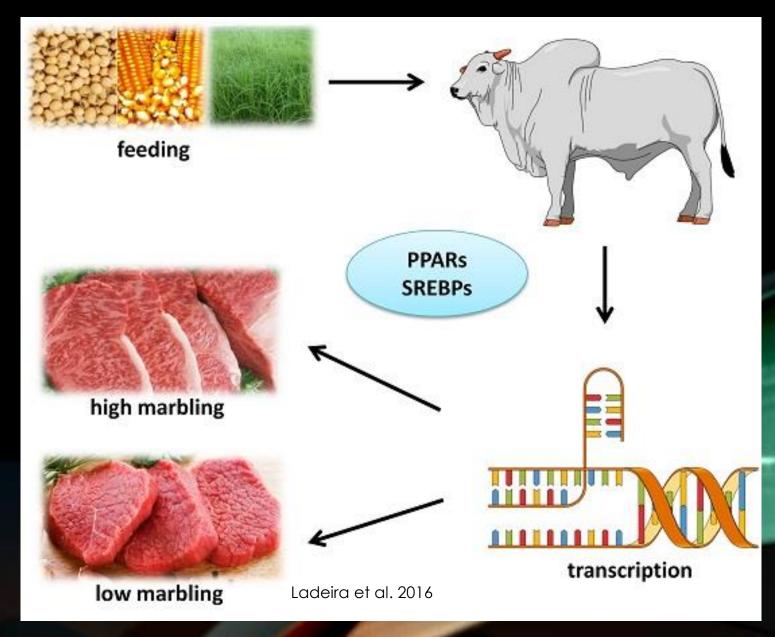
Example	Deficiency/ toxicity/ antioxidant	Alteration	Result
Choline	deficiency	DNA methylation	Increased cancer risk
Folate	deficiency	Induce DNA damage	Accelerate tumor development
Curcumin	antioxidant	Protect telomeres from oxidative damage	Inhibits growth of cancerous cells
Red meat poultry	Toxicity	p53 disease pathway	modified colorectal cancer risk)

**Table 1.** Examples of nutrients that have been identified as antioxidant, deficiencies, or toxicities affecting gene expression and ultimately in disease manifestation.

### Additional Applications of Nutrigenomics

#### **Nutrigenomics and Beef Quality**

Ladeira et al. (2016) shows how nutrition affects the expression of genes involved in cattle lipid metabolism resulting in marbling and fat composition of beef. Focusing on the fatty acid metabolism and the involvement of transcription factors — the peroxisome proliferator-activated receptors (PPARs) and sterol regulatory elementbinding proteins (SREBPs)) in particular – they discover how their mRNA synthesis is regulated by nutrients and their metabolic action may be potentiated by diet compounds. Diet manipulation through changes in polyunsaturated fatty acids, starch concentrations, forage ratios and vitamins might modulate lipogenesis.



**Figure 4.** Ladeira et al. 2016, nutrition affects lipid metabolism resulting in variable fat composition in beef.

## Nutrigenomics study of novel diets in Salmon

By-products resulting from the processing of seafood were potential investigated as nutritional source for atlantic salmon. Hepatic gene expression profiling of fish fed 30% marinebyproduct, 70% basal diet was compared to that of fish fed 100% basal diet at 14 days and 56 days, revealing a significant amount of differentially expressed genes for each diet. Gagné et al. (2016) were able to identify nutritional elements affecting specific gene families.



Figure 5. Atlantic salmon, Salmo salar.

#### References

1. Joffe, YT and Houghton C.

- 2. Riscuta G. 2016
- 3. Ladeira et al. 2016
- 4. Gagné et al. 2016