Basic definitions, division and correlations
Types of responses upon the nutrition
Nutrition and their influence on the genome with examples
Nutrigenomics in genetic variation & epigenetics
Types of diseases
Future perspectives
Conclusions and discussions
NUTRITIONAL GENOMICS

NUTRIGENOMICS

NUTRIGENETICS
GENETIC VARIATION

PHENOTYPE

EPIGENOME

NUTRIENTS

Gene expression

GENETIC RESPONSE

NUTRITIONAL RESPONSE
<table>
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<th>GENETIC RESPONSE</th>
<th>NUTRITIONAL RESPONSE</th>
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<tr>
<td>• Effect on genome evolution</td>
<td>• Effect on nutrients absorption</td>
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<td>• Mutation</td>
<td>• Nutrients utilization and requirement</td>
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<td>• Selection</td>
<td>• Food/nutrient tolerance</td>
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<td>• Programming</td>
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<td>• Viability</td>
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<td>• Chromosome stability</td>
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<td>• Signal transduction and metabolic</td>
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<td>• Protein synthesis and structure</td>
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<td>• Epigenetic events</td>
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<td>• Chronic diseases</td>
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NUTRITION AND THEIR INFLUENCE ON THE GENOME

- Niacin – a polymerase poly(ADP)-rybose substrate which controls the telomeres structure
- Zinc – a cofactor of many antioxidant enzymes and endonuclease IV and also a component of glycosylase OOG I that removes 8oxoG
- Magnesium – polymerases cofactor involved in DNA repair and DNA replication
- Choline – affects methylation of CpG islands and prevents DNA damage
- Vitamin C and E – inhibit oxidation of nucleotides
- Calcium – inhibits the chromosome breakage
Table 2. Examples of the role and effect of specific micronutrients deficiencies on genomic stability.

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<tr>
<th>Micronutrients</th>
<th>Role in genomic stability</th>
<th>Consequence of deficiency</th>
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<tr>
<td>Vits C and E</td>
<td>Prevention of DNA and lipid oxidation.</td>
<td>Increased baseline level of DNA strand breaks, chromosome breaks, oxidative DNA lesions and lipid peroxide adducts on DNA.</td>
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<td>Vit D</td>
<td>Antioxidant activity by increasing glutathione level in normal cell, induction apoptosis in cancer cells.</td>
<td>uracil misincorporation in DNA, increased chromosome breaks and DNA hypomethylation.</td>
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<td>Folate and Vits B2, B6, B12</td>
<td>Maintenance methylation of DNA, synthesis of dTMP from dUMP and efficient recycling of folate.</td>
<td>Increased level of unrepaired nicks in DNA, increased chromosome breaks and rearrangement, sensitivity of mutagens.</td>
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<tr>
<td>Niacin, Nicotinic acid</td>
<td>Maintenance and DNA repair</td>
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<td>Zn, required as a cofactor for Cu/Zn superoxide dismutase, endonuclease IV, P53 function, DNA replication and Zinc finger proteins such as poly (ADP-ribose) polymerase.</td>
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<tr>
<td>Zinc, Manganese and Selenium</td>
<td>Mn, required as a component of mitochondrial Mn superoxide dismutase.</td>
<td>Increased DNA breaks and oxidation, elevated chromosomal damage rate.</td>
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<td>Se, required as a component of peroxidases e.g. glutathione peroxidase.</td>
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<td>Iron</td>
<td>Required as a component of ribonucleotide reductase and mitochondrial cytochromes.</td>
<td>Reduced DNA repair capacity, increased propensity for oxidative damage to mitochondrial DNA.</td>
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<td>Mg, required as a cofactor for a variety of DNA polymerases, in nucleotide excision repair, base excision repair and mismatch repair, essential for microtubule Polymerization and chromosome segregation.</td>
<td>Reduced fidelity of DNA replication, reduced DNA repair capacity, chromosome segregation Errors, survival of genomically aberrant cells.</td>
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<tr>
<td>Magnesium, Calcium</td>
<td>Ca, plays an important role in chromosome segregation and is required for apoptosis.</td>
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GENETIC VARIATION IN NUTRIGENOMICS

- Connected with human migration from east Africa in waves through 6 continents
- The successive splitting off a portion of the gene pool decreased genetic diversity in the migrating group
- Food availability and other factors contributed selective pressures for specific gene variants during migration and dispersal into new environments.
  e.g. lactose intolerance
Diet-informed epigenetic modifications of chromatin (DNA methylation and histone acetylation) that can alter gene function and long term health outcomes.
DISEASES

- Adrenoleukodystrophy
- Diabetes, type 1
- Gaucher disease
- Glucose galactose malabsorption
- Hereditary hemochromatosis
- Lesch-Nyhan syndrome
- Maple syrup urine disease
- Menkes syndrome
- Niemann-Pick disease
- Obesity (PPAR-α)
- Pancreatic cancer
- Phenylketonuria
- Prader-Willi syndrome
- Porphyria
- Refsum disease
- Tangier disease
- Tay-Sachs disease
- Wilson's disease
- Zellweger syndrome
FUTURE PERSPECTIVES - DIETARY RESTRICTIONS

- Restricted diet = longevity
- Restricted diet = prevent diseases
- Short-term restricted diet = decrease risk of heart diseases and stroke
CONCLUSIONS & DISCUSSIONS

- Lead to cancer development
- Diet has an influence on the every step of gene expression (genome, transcriptom, proteom, metabolom)
- Personalized diet as a good way to improve health
CONCLUSIONS & DISCUSSIONS
WHY IT IS IMPORTANT?

- Common dietary chemicals act on the human genome in indirect or direct way, to alter gene expression and/or structure
- Diet can be a serious risk factor for a number of diseases for some individuals
- Some diet-regulated genes are likely to play a role in onset, incidence, progression and/or severity of chronic diseases
- The balance between healthy and diseases state may depend on an individuals genetic background
THANK YOU FOR YOUR ATTENTION!

ANY QUESTIONS?
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