Nutrigenomics in Clinical Practice: Genes, Food, and Specialty Diagnostics

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Nutrigenomics in Clinical Practice: Genes, Food, and Specialty Diagnostics

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Objectives

- To have a general understanding of nutrigenomics
- To learn how to combine the basics of genetics + nutrition
- To see how nutrigenomics can be applied through various examples
- To learn about specific SNPs for the Genova platform
Food Is…

- Medicine
- Connection
- *Information*
Food sends informational signals to the genes
Although genes are critical for determining function, nutrition modifies the extent to which different genes are expressed and thereby modulates whether individuals attain the potential established by their genetic background.

Your genes are not your destiny
Personalized Nutrition (Healthcare)

Targeted dietary prescriptions for the individual based on genetics and lifestyle
Levels of personalized, functional medicine-based health

- Genetics
  - Typing
  - SNPs

- Epigenetics
  - Methylation
  - Phosphorylation

- Biochemical
  - Standard Labs
  - Functional Biomarkers
Can including genetic information to personalize a patient’s diet (nutrigenetics) improve long-term weight management?

- N=50 patients in genetic group; N=43 patients in control group
- Standard Mediterranean diet, modified for nutrigenetic group

**Research**

**Improved weight management using genetic information to personalize a calorie controlled diet**

Ioannis Arkadianos¹, Ana M Valdes², Efstathios Marinos³, Anna Florou¹, Rosalynn D Gill⁴ and Keith A Grimaldi*⁴

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After 300 days of follow-up individuals in the nutrigenetic group were more likely to have maintained some weight loss (73%) than those in the comparison group (32%).
Here, we continuously monitored week-long glucose levels in an 800-person cohort, measured responses to 46,898 meals, and found high variability in the response to identical meals, suggesting that universal dietary recommendations may have limited utility.
Personalized Nutrition

We devised a machine-learning algorithm that integrates blood parameters, dietary habits, anthropometrics, physical activity, and gut microbiota measured in this cohort and showed that it accurately predicts personalized postprandial glycemic response to real-life meals.
Nutrigenomics: The Overview

**FOOD/NUTRIENTS**

Prospective science
The effect of diet on gene expression

**NUTRIGENOMICS** (Gene Expression)

Interaction of Diet-Genome

**NUTRIGENETICS** (Gene Variation)

Retrospective science
The effects of individual's genetic variations to diet

**GENOME**
Nutrigenomics

- The influence of food on genetic expression
- How what you eat turns on or off your genes
- Refers to the interaction between genes and nutrients
- Modification can occur directly or indirectly
- Chronic disease onset, incidence, progression, and/or severity influenced by diet-regulated genes and their common variants
**Nutrigenomics**

- Examples:
  - Sulforaphane in broccoli can turn off oncogenes (cancer-initiating or – causing genes)
  - Resveratrol in grape skin can lead to changes in gene expression that cause a shift in energy production and metabolism
Why is nutrigenomics important for nutrition?

It allows us to question current dogma

- Food is more than calories
- A calorie is a calorie
- Bad foods give you disease unless you have genes to intervene and protect you
New concepts to ‘digest’

• Food is full of informational signals
• A calorie is to be judged upon the context it comes from
• We are continually interacting with dietary signals, in which certain foods enhance a beneficial, neutral or negative effect on genes
• Clinical trials need to include genetic variability in SNPs as a factor
Nutrigenetics

• The genetic makeup a person has that leads them to require certain nutrients or higher/lower levels of nutrients, both of which may be implicated in their propensity towards disease
• Includes SNPs
• A field of study that will play a role in personalized nutrition
Epigenetics: The Wild Card

- Heritable changes that do not impact gene sequence.
- Modification to gene sites or histone proteins
  - Methylation
  - Phosphorylation
  - Acetylation
  - Ubiquinylation
Nutrients play a role in epigenetics
Foodomics is the comprehensive, high-throughput approach for the exploitation of food science in the light of an improvement of human nutrition.

Foodomics is a new approach to food and nutrition that studies the food domain as a whole with the nutrition domain to reach the main objective, the optimization of human health and well-being.

Phytoprofiling

The role of phytochemical modulation of cellular physiology and propose phytochemical profiling, or phytoprofiling, to assist in the facilitation of determining phytonutrient requirements with more effective interventions with plant-derived compounds.
Nutrition & SNPs: Specific Profiles
Here’s the problem:

Nutrient insufficiency
+ Enzyme insufficiency
= Poor methylation capacity
What You Need to Know

SNPs are cutting-edge markers that provide general information about a patient’s propensity toward disease

SNPs may provide insight into an array of patient conditions such as depression, anxiety, cardiovascular disease, cardio-metabolic syndrome, inflammatory conditions, and chronic pain syndromes
What You Need to Know

SNPs are an important tool for personalized nutrition

Nutrition needs to be personalized for it to be effective long-term. Diagnostic labs that assess genetic information, as well as functional biomarkers, can be utilized for this purpose.
What You Need to Know

For best results, couple SNPs with other diagnostic and functional biomarkers

Having a variety of tests, such as NutrEval, together with SNPs, supports broadened clinical insight and enhanced personalization of therapeutics
What You Need to Know

Don’t diagnose or prescribe based on a single SNP

SNPs are good information for a clinician to have about a patient, and are to be seen as part of a complete picture rather than used in isolation to make a diagnosis or to prescribe treatment.
Your patients’ SNPs are not “their destiny”

Many people mistakenly assume that the presence of a certain gene means they are destined to experience the associated disease. However, only a few very rare diseases (such as Huntington or Tay-Sachs diseases) are certainties determined by genetic makeup.

Most genes have flexible expressions and researchers have found that complex interactions among multiple genes plus the environment are fundamentals of disease etiology.
What You Need to Know

The same SNP may not look the same in everyone

SNPs may be differentially expressed based on one’s nutrient status, interacting SNPs, stressors, environment, and lifestyle choices
What You Need to Know

SNPs may express differently in different population groups

Literature used to assess SNPs may be quite varied in findings, and be different depending on population groups, including ethnicity and gender variables
What You Need to Know

The accuracy of genetic testing is not 100%

Results of genetic tests should be taken in the context of clinical representation and familial risk. The prevalence and significance of some allelic variations may be population specific. Your patient may have additional risk that is not measured by this test. Negative findings do not imply that your patient is risk-free.
Methylenetetrahydrofolate reductase polymorphisms:

The BIGGEST SNP in Functional Medicine!
Your health depends on the transfer of a 1-carbon unit and this transfer depends on nutrients!

- Folic acid
- Vitamin B2
- Vitamin B12
- Vitamin B6
- Betaine

Diagram:

- FOLIC ACID
- METHIONINE
- TETRAHYDROFOLATE
- 5-METHYLTETRAHYDROFOLATE
- S-ADENOSYL METHIONINE
- S-ADENOSYL HOMOCYSTEINE
- MTHFR (VIT B2)
- BHMT (VIT B12)
- CYSTATHIONINE
- CYSTEINE
- SULFATE
- CS, CYSTATHIONINE - β - SYNTASE
- CL, CYSTATHIONINE - γ - LYASE
- BHMT, BETAIN HOMEYOCYSTEINE METHYL TRANSFERASE
- DMG, DIMETHYLGLYCINE
More about MTHFR

Two different copies of the MTHFR gene:
- C677T
- A1298C

Wild type: -/-  Full strength of the enzyme
Heterozygous: +/-  Some enzyme activity
Homozygous: +/-  Enzyme reduced 60-70%
Disease risk is most pronounced for the homozygous genotype for C677T

Copy of the MTHFR gene:

- C677T

<table>
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<th>Wild type</th>
<th>+/-</th>
<th>Full strength of the enzyme</th>
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<tr>
<td>Heterozygous</td>
<td>+/-</td>
<td>Enzyme reduced 30-40%</td>
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<tr>
<td>Homozygous</td>
<td>+/-</td>
<td>Enzyme reduced 60-70%</td>
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Reduced methylation
Reduced Activity of MTHFR: Clinical Conditions

- Alzheimer’s Disease
- Anxiety
- Cancer
- Cognitive Decline
- Depression
- Heart Disease and Stroke
- Obsessive Compulsive Disorder
- Spina Bifida and NTDs
Biomarkers Related to Methylation: Homocysteine (Hcy)

- **Folic acid**
- **Vitamin B2**
- **Vitamin B12**
- **Vitamin B6**
- **Betaine**
Biomarkers Related to Methylation: Homocysteine (Hcy)

• Increased risk of high homocysteine, especially when there are low levels of B vitamins, mainly folate
• Several studies also suggest tendency for lower folate levels
• Moderate total Hcy elevations have been found to correlate with hypomethylation of DNA in lymphocytes
• Vascular problems associated with hyperhomocysteinemia may be partly due to DNA hypomethylation
Can we rely solely on Hcy levels to tell us something about methylation?

Short answer: NO
What nutritional and lifestyle therapies would you recommend for someone with a MTHFR SNP?
Reduced Activity of MTHFR (C677T): Treatment Strategies

**FOOD FIRST**

- Ensure adequate intake of dark-green leafy vegetables and other B vitamin-rich foods
Reduced Activity of MTHFR (C677T): Treatment Strategies

SUPPLEMENTATION

• Consider supplementation with:
  – Folic acid (preferentially 5-methyltetrahydrofolate, which bypasses the MTHFR step)
  – B2
  – B6 (pyridoxal 5-phosphate)
  – B12 (or methylcobalamin)
  – Betaine (trimethylglycine)
Reduced Activity of MTHFR (C677T): Treatment Strategies

LIFESTYLE

• Smoking cessation, if applicable
• Chronic heavy drinking is to be strongly discouraged due to inhibition of methionine synthase, folate depletion in mitochondria and abnormal DNA synthesis and DNA methylation
Methylenetetrahydrofolate reductase polymorphisms:

Summary & Key Clinical Messages
MTHFR Summary Snapshot

1. What is it?
   - Key enzyme involved in methylation

2. When is it clinically indicated?
   - Hyperhomocysteinemia, low B vitamin levels, mainly folate; increased risk of venous thrombosis, CVD, HTN, stroke, diabetic neuropathy or retinopathy, depression, autism, and schizophrenia; increased risk of birth defects (NTDs or congenital heart defects, cleft lip and/or palate, and Down syndrome); increased risk of recurrent pregnancy loss; increased risk of fracture and/or low BMD; increased risk of all cancers; NAFLD

3. How does it compare to other commonly used diagnostics?
   - Should be used in combination with other markers, not as a standalone

4. What are the clinical implications of an abnormal result?
   - Possible impaired methylation, low B vitamins, high Hcy

5. What are other nutrients to consider?
   - Mg, Zn, Fe, B2, B6, B12, Folate/5-MTHF, TMG/Betaine

6. What is the treatment for impaired activity?
   - Dietary and supplemental sources of B vitamins, healthy lifestyle
Catechol-O-Methyltransferase:

Think hormones, toxins, and neurotransmitters!
About COMT

Enzyme that catalyzes the movement of a methyl group from S-adenosylmethionine to a catechol or a catecholamine

- Dopamine
- Epinephrine
- Norepinephrine
- Estrogens
- Various chemicals (endocrine disruptors) and toxins
Adapted from the Neurological Research Institute’s Diagram and simplified by April Ward-Hauge MS, NP.
COMT Variant: 158

• The COMT gene has a well-studied, common variant at codon 158
• Those with valine (Val158) alleles have greater COMT activity compared with those with the methionine (Met158) substitution
COMT 158V→M
+  + (Homozygous, most impaired)

• 3-4-fold reduction in COMT enzyme activity, resulting in decreased methylation
• Increased risk of nervousness/anxiety (especially when history of childhood trauma and PTSD) due to higher baseline levels of catecholamines; may be population dependent
• Acute or chronic stress can compromise working memory, decision-making ability, or mood by producing supraoptimal dopamine levels
• Strong cognitive stability, e.g., ability to focus (due to higher brain dopamine), but lower cognitive flexibility (e.g., ability to adapt to external changes), compared to the other genotypes
COMT 158V→M
+  + (Homozygous, most impaired)

• Conflicting reports for breast cancer risk, possible increased risk in Asian women, but marginally decreased risk in Caucasian women
• Reduced pain threshold which is exacerbated by one’s experience of pain, increased risk of fibromyalgia and chronic pain syndromes
• Increased fracture risk, esp. in men, but greater BMD response to physical activity
• Possible increased risk of substance addiction, including alcoholism
• Possible increased risk of Parkinson’s disease (mixed studies)
• Minimize stress to keep catecholamines low
• Ensure adequate B6, B12, folate, magnesium, betaine, and methionine to support formation of SAM and prevent elevated Hcy; SAH inhibits COMT activity
• Preliminary findings suggest reduced risk of cardiovascular events by taking aspirin or vitamin E
• Exercise caution using CEEs (e.g., Premarin); in-vitro studies show one of its metabolites to inhibit COMT in this genotype
• Individuals with this genotype may have a superior response to SSRI antidepressants (mixed studies)
• In children with ADHD, methylphenidate (Ritalin) may be less effective (mixed studies)
Apolipoprotein E:

Think lipids, CVD and dementia
Unesterified Cholesterol
Apolipoprotein
Cholesteryl Ester
Triglyceride
Phospholipid
VLDDL
B-100
Apolipoprotein E: Physiology and Function

A multifunctional lipid-transport protein with central roles in:

- lipid metabolism
- brain lipid transport
- glucose metabolism
- neuronal signaling
- neuronal inflammation
- mitochondrial function
Apolipoprotein E: Physiology and Function

- Human APOE exists as three major isoforms:
  - APOE2
  - APOE3
  - APOE4

- The parent form, APOE3, promoting clearance of triglyceride-rich lipoproteins and stabilization of plasma lipids
Apolipoprotein E:
Gene Variant Possibilities

- E2/E2
- E2/E3
- E2/E4
- E3/E3
- E3/E4
- E4/E4
Apolipoprotein E: Role in neuroscience & cognition

- Migration
- Axon guidance
- Synaptic plasticity
- Microtubule stability
- Regeneration
- Survival
- Amyloid deposition

Nature Reviews | Neuroscience
Apolipoprotein E: Role in neuroscience & cognition
Apolipoprotein E:
E2/E2

- The E2/E2 genotype is rare, accounting for less than 1% of a given population
- ApoE2 is associated with lower LDL-C and higher HDL-C, but higher TGs
- Slight increased risk of type 2 diabetes and diabetic nephropathy
- Higher uric acid levels in Chinese population
- Generally associated with the lowest risk of atherosclerosis, MI and stroke; however, CAD and MI risk may increase with elevated TGs
- Tendency toward higher plasma C-RP despite lower CV risk.
- Lowest risk of osteoporosis; highest antioxidant activity
- APOE E2/E2 genotype is a potential genetic risk factor for vertebral fractures in humans (newer research, 2014)
Apolipoprotein E: E2/E2 Treatment Options

- The cholesterol-lowering effect of a low saturated fat and low cholesterol diet is least profound in E2 individuals
- Minimize sugar and high-glycemic index foods, which produce the largest TG response in this genotype
- Fish oils may reduce TGs the most effectively in E2 carriers
- Alcohol may reduce LDL-C in men (neutral in women), but may increase risk of hemorrhagic stroke in men (at least in Asians)
- Lipid response to statins, as well as the TG response to fibrates, are usually the best in E2 > E3 > E4; studies are mixed
- Gemfibrozil may help lower TGs and total cholesterol
- HRT improves the lipid profile in this genotype, although oral estrogen may significantly increase TGs
Apolipoprotein E: E3/E3

- Most common (accounting for >50% of most populations) and is the genotype against which E2 and E4 are compared
- E3/E3 may be protective against stroke compared with other genotypes, particularly in females
- ApoE3 confers only a moderate tendency toward elevated total- and LDL cholesterol, and lower HDL-C
- Risk is intermediate between E2 and E4 for atherosclerosis, MI, stroke (in smokers), and osteoporosis
- The E3 genotype led to an approximate 90% increase in the levels of TG in the presence of abdominal obesity
Apolipoprotein E: E3/E3 Treatment Options

- Effects of cholesterol and dietary fat on serum cholesterol levels are least profound with the E2 allele and greatest with the E4 allele; thus, dietary fat restriction produces a moderate cholesterol response in E3/E3 individuals.
- Carbohydrate intake may be inversely correlated with HDL-C.
- Alcohol may have a neutral effect on LDL-C.
- Avoid smoking, which increases risk of CAD in this genotype.
- Lipid response to statins, and triglyceride response to fibrates, are usually the best in E2 > E3 > E4; studies are mixed.
- HRT generally improves the lipid profile in all genotypes, including post-menopausal E3 carriers.
Apolipoprotein E: E4/E4

- The E4/E4 genotype is rare, accounting for less than 3% of a given population
- Highest total- and LDL cholesterol, lowest HDL-C
- Increased risk of stroke (esp. among Asians), hypertension, and MI; also increased risk of cognitive impairment after stroke; possibly lower CRP levels, despite higher CV risk
- May be an independent predictor of CAD and type 2 diabetes, especially in obese individuals and smokers
- Increased risk of low BMD, oxidative stress, also easier toxicity by heavy metals such as lead and mercury
- Possible increased risk and disease severity of multiple sclerosis
Apolipoprotein E: E4/E4 Treatment Options

• Reduce stress due to poor response to stressors; prolonged stress contributes to memory decline
• Restricting saturated fat and cholesterol reduces total- and LDL cholesterol, as well as CAD and MI risk
• Avoid smoking and minimize high-GI foods, both of which augment E4-associated risk of CHD
• Alcohol may raise LDL-C in men (neutral effect in women), increase IL-6 levels, and fail to raise HDL-C
• Reduce excess weight, which synergizes with effects of E4 on insulin and lipids
• Fish oils may lower triglycerides but increase LDL-C; mixed studies
• Physical activity and fiber both benefit lipid levels
• Antioxidants may help to counteract low tissue levels; anti-inflammatories help preserve cognition
• Response to statins/fibrates, usually the most positive in E2>E3>E4; studies are mixed
• Estrogen therapy particularly efficacious for both cholesterol and bone in postmenopausal E4 carriers
• APOE4 carriers with BMI ≥25.5 may need higher intakes of DHA for cardiovascular or other health benefits than do noncarriers (Chouinard-Watkins et al., 2015)
1. What is it?
   - Key protein involved in transport of lipids

2. When is it clinically indicated?
   - Plasma lipid abnormalities such as high TGs, high LDL-C, low HDL-C; increased risk for T2DM, atherosclerosis, MI, stroke; indications of high inflammation such as elevated C-RP and hyperuricemia; low BMD or increased risk for OP and fractures; indications of oxidative stress/low antioxidant status and heavy metal toxicity.

3. How does it compare to other commonly used diagnostics?
   - Should be used in combination with other markers, not as a standalone

4. What are the clinical implications of an abnormal result?
   - Possible increased risk of high lipids, CVD, and/or dementia

5. What are other nutrients to consider?
   - Cardiovascular and neurological supportive nutrients

6. What is the treatment for impaired activity?
   - Minimize stress, healthy diet low in sugar and high in nutrients
Tumor Necrosis Factor-alpha:

The inflammation ‘monster’
TNF-alpha

Diagram showing the effects of TNF-alpha on a macrophage, including apoptosis, IkBalpha degradation by proteasome, NF-kB activation, and pro-inflammatory cytokine synthesis.
TNF-alpha: What is it?

- TNF-alpha (TNF-α) is a pro-inflammatory cytokine that is secreted from activated macrophages.
- TNF-α plays an important role in host defense against infection; however, excessive release of the cytokine increases inflammation and oxidative stress.
Several SNPs in the TNF gene promoter have been identified, some of which may regulate TNF expression.

One of these polymorphisms at position -308 (TNF -308 G/A) had been reported to affect cytokine production and be associated with regulation of TNF expression by, e.g., interfering with transcription factor binding sites or other regulatory elements.
TNF-alpha:
308G→A +/+ (Greatly increased activity)

• Substantially increased production of TNF-α, risk of inflammation and oxidative stress
• All the same clinical issues seen with the +/- genotype
• Increased risk of OA (Kou and Wu, 2014)
• Elevated risk for acne vulgaris among Caucasians (Yang et al., 2014)
TNF-alpha:
308G→A +/+ (Greatly increased activity)
Treatment Options

• Abdominal fat loss; visceral fat produces TNF-α and IL-6, and weight loss is associated with a decrease in these inflammatory cytokines
• Improve insulin sensitivity
• Control stress response
• Individuals with the SNP are more prone to weight gain and an abnormal cholesterol profile from a high intake of saturated fat and/or n-6 fatty acids
• TNF-α levels may be reduced by vitamin E, fish oils, N-acetylcysteine, green tea, Siberian ginseng, nettles, lactobacillus, estrogen, and DHEA
• Possible inferior response to anti-TNF-α medications (e.g., etanercept) in rheumatoid arthritis; also possible resistance to steroid treatment for inflammatory conditions
1. What is it?
   – Proinflammatory cytokine

2. When is it clinically indicated?
   – Presence of all (chronic) inflammatory conditions

3. How does it compare to other commonly used diagnostics?
   – Should be used in combination with other markers of inflammation, not as a standalone

4. What are the clinical implications of an abnormal result?
   – Possible increased risk of inflammation/inflammatory conditions

5. What are other nutrients to consider?
   – Nutrients to reduce inflammation

6. What is the treatment for impaired activity?
   – Minimize stress, low-inflammation diet
SNPs & NutrEval: Mood Sensitization Disorders

Depression:
- Compelling SNPs: APOE, COMT, MTHFR (mixed)
- NutrEval: B vitamins, omega-3 fatty acids, vitamin C
Summary

• There is a lot we now know about genes
• There is a lot we still don’t know about genes and modulation of the epigenome
• There is still less we know about nutrigenomic application to clinical medicine, but there is some recent data emerging
• Food (and eating) is (are) filled with informational signals delivered to our cells.
• Nutrigenetic testing should be coupled with laboratory nutrient assessment for clinical application
• Note how nutrients come together with genes for a more complete picture/assessment
• MTHFR, COMT, APOE, and TNF-a are some important genes that will assist with clinical therapeutic strategies
Questions?

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