Nutrigenomics:
Do our genes determine what we should eat?

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Disclosure

B. G-B is Director of Research and Development at Nutrigenomix Inc.
Using genetic information to investigate how nutrients interact with our genes to impact our health and performance.

Diet $\rightarrow$ Genes $\rightarrow$ Food preferences
- Genes are a sequence of nucleotides (A, C, G and T)

- Differences in nucleotides produce genetic variants
  - Single Nucleotide Polymorphism = “SNP”
    - Example: a “C” replacing an “A”
  - Insertion/deletion = deleted segments (“del”) or additional segments can be inserted (“ins”)

- We inherit two copies of most genes, one from each parent
  - Each gene can have two different forms (e.g. A and C, or ins and del)
  - Three possible variants (or “genotypes”):
    - Example: AA, CA, or CC
The Science of Nutrigenomics
Why are genetic differences important for nutrition?

One size does not fit all
‘Omics’ Technologies

- Genomics
- Transcriptomics
- Proteomics
- Metabolomics
- Nutrients
- Epigenomics
- Transcriptomics
- Proteomics
- Microbiomics
Genetic Association Studies

- Disease associated genes
  - More successful for rare diseases
  - Not so successful for common, chronic diseases
THE ANGELINA EFFECT

Angelina Jolie's double mastectomy puts genetic testing in the spotlight. What her choice reveals about calculating risk, cost and peace of mind

BY JEFFREY KLUGER & ALICE PARK
Disease Risk Genes

vs

Modifier Genes
Genetic Association Studies

- Disease associated genes
  - More successful for rare diseases
  - Not so successful for common, chronic diseases
- Modifier genes
  - Absorption, Distribution, Metabolism, Uptake, Elimination
Is Coffee associated with CVD?

Coffee → Genes → Genotype A → Increase → CVD

Coffee → Genes → Genotype B → No Effect

Coffee → Genes → Genotype C → Decrease
Bioactives in Coffee

- Caffeine
- Magnesium
- Polyphenols
- Aliphatic acids
- Potassium
- Melanoidins
- Diterpenoids
- Polyphenols
Caffeine

CYP1A2

Paraxanthine

1,7-dimethyluric acid
1-methylxanthine
1-methyluric acid
5-acetylamino-6-formylamino-3-methyluracil
Genetic Variation in *CYP1A2* -163 A→C

% Inducibility

- **fast**
  - AA
  - AC
  - CC

- **slow**

*CYP1A2* Genotype
• 2013 cases (myocardial infarction)
• 2013 population-based controls
  - matched (age, sex, area of residence)

• Data collection:
  - food frequency questionnaire
  - health and lifestyle questionnaire
  - fasting blood sample (DNA)
Coffee Intake and Risk of Myocardial Infarction

Odds Ratio

- <1 cup/d
- 1 cup/d
- 2-3 cups/d
- ≥4 cups/d

* P<0.05

Cornelis et al., JAMA 295: 1135-41, 2006
Coffee Intake and Risk of Myocardial Infarction

* P<0.05

Cornelis et al., JAMA 295: 1135-41, 2006
Coffee Intake and Risk of Myocardial Infarction

**CYP1A2 Genotype**

- AA
- AC + CC

**Odds Ratio**

- <1 cup/d
- 1 cup/d
- 2-3 cups/d
- ≥4 cups/d

* P<0.05

Cornelis *et al.*, JAMA 295: 1135-41, 2006
Coffee, CYP1A2 Genotype, and Risk of Myocardial Infarction

Marilyn C. Cornelis, BSc
Ahmed El-Sohemy, PhD
Edmond K. Kabagambe, PhD
Hannia Campos, PhD

JAMA. 2006;295:1135-1141
Why two cups of coffee can damage your heart

Coffee drinkers who consume four or more cups a day increase their chances of having a heart attack by more than 60 per cent if they carry a variant gene, newly published research suggests. The risk for those who drink two to three cups a day was shown to be 36 per cent higher than normal.
Gene that could make your next coffee your last

New research suggests that some people cannot process caffeine as quickly as others and may therefore be more vulnerable to a heart attack, Sam Lister reports.

COFFEE drinkers who have more than three cups a day could significantly increase their chances of suffering a heart attack.

New research suggests that people who carry a particular variation of a gene cannot process caffeine as quickly as other people. Such individuals could be up to 64 per cent more likely to have a heart attack if they drink large amounts of coffee.

High amounts of caffeine can be dangerous, but some doctors suggest coffee also has benefits.
For Coffee Drinkers, the Buzz May Be in Your Genes

By ANAHAD O’CONNOR
JULY 12, 2016

Like most of my work, this article would not have been possible without coffee.
CYP1A2 genotype modifies the association between coffee intake and the risk of hypertension
Paolo Palatini\textsuperscript{a}, Giulio Ceolotto\textsuperscript{a}, Fabio Ragazzo\textsuperscript{a}, Francesca Dorigatti\textsuperscript{a}, Francesca Saladini\textsuperscript{a}, Italia Papparella\textsuperscript{a}, Lucio Mos\textsuperscript{b}, Giuseppe Zanata\textsuperscript{c} and Massimo Santonastasso\textsuperscript{d}

*Journal of Hypertension* 2009, 27:1594–1601
Replication: Risk of Hypertension

- Abstainers
- 1-3 cups/day
- ≥4 cups/day

*CYP1A2 Genotype*

<table>
<thead>
<tr>
<th>Hazard Ratio</th>
<th>CYP1A2 Genotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>AA</td>
</tr>
<tr>
<td>1</td>
<td>AC + CC</td>
</tr>
</tbody>
</table>

*P<0.05

Palatini et al., J Hypertens 27: 1594-1601, 2009
DOI 10.1007/s10654-015-9990-z

CARDIOVASCULAR DISEASE

Association of coffee consumption and CYP1A2 polymorphism with risk of impaired fasting glucose in hypertensive patients

Paolo Palatini · Elisabetta Benetti · Lucio Mos · Guido Garavelli · Adriano Mazzer · Susanna Cozzo · Claudio Fania · Edoardo Casiglia
A government panel said drinking coffee is harmless. Why that might be wrong.

A U.S. panel said coffee can be part of a healthy diet. That might be true only for half of us.
“There are spectacular metabolic differences in people, and to expect that coffee will have the same health effects on everyone is absurd”

- Dr. Sander Greenland
  Professor Emeritus, Epidemiology
  UCLA
“Unfortunately, because genetic testing is expensive and rarely done, most people have little idea which gene variant they carry”

- Panel Member
First Clinical Trial of Vitamin C

A TREATISE ON THE
SCURVY.
IN THREE PARTS.

CONTAINING
An Inquiry into the Nature, Causes,
and Cure, of that Disease.

Together with
A Critical and Chronological View of what
has been published on the Subject.

By JAMES LIND, M. D.
Physician to his Majesty's Royal Hospital at Hotham
near Portsmouth, and Fellow of the Royal
College of Physicians in Edinburgh.

The THIRD EDITION, enlarged and improved.

LONDON:
Printed for S. Crowder, D. Wilson and G.
Nicholls, T. Cadell, T. Becket and Co.
G. Pearch, and W. Woodfall.
MDCCCLXXII.

James Lind, 1772
Original Contribution

Vitamin C Deficiency in a Population of Young Canadian Adults

Leah Cahill, Paul N. Corey, and Ahmed El-Sohemy

Initially submitted March 17, 2009; accepted for publication May 11, 2009.
### Vitamin C Deficiency in Canadian Adults

<table>
<thead>
<tr>
<th></th>
<th>Serum Ascorbic Acid Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Deficient (&lt;11 μmol/L)</td>
</tr>
<tr>
<td></td>
<td>Suboptimal (11-28 μmol/L)</td>
</tr>
<tr>
<td></td>
<td>Adequate (&gt;28 μmol/L)</td>
</tr>
<tr>
<td><strong>N (%)</strong></td>
<td></td>
</tr>
<tr>
<td>All subjects</td>
<td>133 (14)</td>
</tr>
<tr>
<td></td>
<td>325 (33)</td>
</tr>
<tr>
<td></td>
<td>521 (53)</td>
</tr>
<tr>
<td>Women</td>
<td>87 (13)</td>
</tr>
<tr>
<td></td>
<td>218 (31)</td>
</tr>
<tr>
<td></td>
<td>387 (56)</td>
</tr>
<tr>
<td>Men</td>
<td>46 (16)</td>
</tr>
<tr>
<td></td>
<td>107 (37)</td>
</tr>
<tr>
<td></td>
<td>134 (47)</td>
</tr>
</tbody>
</table>

## Serum Ascorbic Acid and Biomarkers of Disease


<table>
<thead>
<tr>
<th></th>
<th>Deficient (≤11 μmol/L)</th>
<th>Suboptimal (11-28 μmol/L)</th>
<th>Adequate (≥28 μmol/L)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BMI (kg/m²)</strong></td>
<td>23.1 ± 0.1a</td>
<td>23.0 ± 0.2a</td>
<td>22.3 ± 0.2b</td>
<td>0.007</td>
</tr>
<tr>
<td><strong>Waist Circ. (cm)</strong></td>
<td>75.0 ± 0.7a</td>
<td>74.6 ± 0.5a</td>
<td>72.8 ± 0.4b</td>
<td>0.003</td>
</tr>
<tr>
<td><strong>Systolic Blood P.</strong></td>
<td>114.8 ± 1.0</td>
<td>114.7 ± 0.6</td>
<td>113.0 ± 0.5</td>
<td>0.06</td>
</tr>
<tr>
<td><strong>Diastolic Blood P.</strong></td>
<td>70.2 ± 0.7a</td>
<td>69.6 ± 0.4a</td>
<td>68.2 ± 0.4b</td>
<td>0.004</td>
</tr>
<tr>
<td><strong>hs-CRP (mg/L)</strong></td>
<td>2.04 ± 0.23a</td>
<td>1.46 ± 0.15b</td>
<td>1.03 ± 0.12b</td>
<td>0.0004</td>
</tr>
<tr>
<td><strong>Insulin (pmol/L)</strong></td>
<td>54.3 ± 3.1</td>
<td>47.8 ± 2.0</td>
<td>48.0 ± 1.6</td>
<td>0.17</td>
</tr>
<tr>
<td><strong>Total chol:HDL</strong></td>
<td>2.74 ± 0.06</td>
<td>2.85 ± 0.04</td>
<td>2.77 ± 0.03</td>
<td>0.22</td>
</tr>
</tbody>
</table>
Functional genetic variants of glutathione S-transferase protect against serum ascorbic acid deficiency\textsuperscript{1-3}

Leah E Cahill, Bénédicte Fontaine-Bisson, and Ahmed El-Sohemy

GST-T Genotype and Serum Ascorbic Acid Deficiency

Caucasians

Vitamin C Intake

\[ \begin{align*}
\text{GST-T genotype} & \quad \text{Serum Ascorbic Acid (µmol/L)} \\
\text{Null} & \quad \text{<RDA} \quad \text{Meets RDA} \\
\text{Functional} & \quad \text{<RDA} \quad \text{Meets RDA}
\end{align*} \]

East Asians

\[ \begin{align*}
\text{GST-T Genotype} & \quad \text{Serum Ascorbic Acid (µmol/L)} \\
\text{Null} & \quad \text{<RDA} \quad \text{Meets RDA} \\
\text{Functional} & \quad \text{<RDA} \quad \text{Meets RDA}
\end{align*} \]

\[ P=0.001 \text{ for diet-gene interaction} \]

\[ P=0.02 \text{ for diet-gene interaction} \]

Cahill et al, Am J Clin Nutr, 2009
Gluten

- Gluten-free diet increasing in popularity
- Gluten-free market valued at $4.2 billion

Maclean’s Magazine, 2013
### Celiac Disease

| Symptoms                  | Gastrointestinal discomfort  
|                          | Joint pain                   
|                          | Skin rashes                  
|                          | Nutrient deficiency          
|                          | Respiratory problems         |
| Prevalence               | 1% of Caucasian population   |
| Diagnosis                | IgA                          
|                          | tTG ✓                        
|                          | EMA ✓                        |
| Genetic Basis            | Human Leukocyte Antigen      
|                          | (Chromosome 6)               |

• 1% prevalence among Caucasians
• 87% undiagnosed cases of CD
Celiac Disease: Diagnostic Approach

- **tTG IgA**
- **EMA IgA**

**HLA-DQ Genotyping**

**HLA-DQ2/8+**
- **30%**

**HLA-DQ2/8-**
- **70%**

- **tTG IgA**
- **EMA IgA**

Celiac Disease ruled out

Celiac Disease: Diagnostic Approach

Biopsy
Parents Risk ≠ Children’s Risk
Parents Risk ≠ Children’s Risk
Folate and Vitamin B12

**FOLATE:**
- Synthesis of nucleic acids, amino acids, and intracellular methylating enzymes
- Deficiency:
  - Abnormal cell growth
  - Impaired DNA methylation
  - Increased risk of point mutations, chromosome damage, and aneuploidy

**B12:**
- Cofactor for folate metabolism
- Methylation of nucleic acids and neurotransmitters
- Helps regulate homocysteine conversion to methionine
- Deficiency:
  - Damage to brain and nervous system
  - Fatigue & lethargy
  - Depression, poor memory
  - Headaches
  - Breathlessness
Association between serum folate and vitamin B-12 and outcomes of assisted reproductive technologies

Audrey J Gaskins,2* Yu-Han Chiu,2 Paige L Williams,3,4 Jennifer B Ford,5 Thomas L Toth,6 Russ Hauser,4–6 and Jorge E Chavarro2,4,7 for the EARTH Study Team


Likelihood of Live Birth, by Folate and B12 Status

Hypothesis: Genetic variants in folate metabolic pathway may affect methylation of peri-centromeric region of chromosome 21, leading to abnormal segregation during maternal meiosis.
The genetics of folate metabolism and maternal risk of birth of a child with Down syndrome and associated congenital heart defects

Fabio Coppedè¹,²*

¹ Section of Medical Genetics, Department of Translational Research and New Technologies in Medicine and Surgery, University of Pisa, Pisa, Italy. ² Interdepartmental Research Center Nutrafood “Nutraceuticals and Food for Health,” University of Pisa, Pisa, Italy

Summary of evidence on whether maternal polymorphisms along folate pathway affect risk of DS in offspring
The Folate Metabolic Pathway

Risk factors for DS offspring?
- **MTHFR 677C>T**
- **MTRR 66A>G**
- **RFC1 80G>A**
Gene-Gene & Gene-Environment Interactions Affect Genetic Risk Factors

Some examples:


- Mothers of children with DS had lower dietary folate intake at the time of conception (James et al., 1999, Am J Clin Nutr 70:495–501)
  - This may have had greater impact on genetically-at-risk mothers
Adequate Folate Intake $\rightarrow$ Especially Important for Genetically-at-Risk Individuals

Percentage of subjects with low serum folate, after repletion with 400 mcg/day of Dietary Folate Equivalents

Adapted from Guinotte et al. J Nutr. 2003;133:1272-1280
Trans-Generational Effects of Folate Metabolism?

Coppede, Front Genet. 2015;6:223
Could Variation in B12 Metabolism Genes Also Affect Risk?

- Fucosyltransferase 2 (FUT2): involved in vitamin B12 absorption and transport between cells

- Despite B12’s role in methylation and nucleic acid synthesis, relationship between this genetic variant and DS remains poorly understood

Adapted from Tanwar et al. Gene. 2013;515:224-228
Nutritional interventions to target trisomy 21 effects?

Some Genes Affected by Trisomy 21:

- SOD (Superoxide Dismutase) $\rightarrow$ Oxidative stress
- APP (Amyloid Precursor Protein) $\rightarrow$ Alzheimer’s disease risk
- CBS (cystathionine beta synthase) $\rightarrow$ Mitochondrial dysfunction

*Can antioxidants ameliorate DS symptoms?*
Nutritional interventions to target trisomy 21 effects?

Oxidative Stress and Down Syndrome. Do Antioxidants Play a Role in Therapy?

J. Muchová¹, I. Žitňanová¹, Z. Ďuračková¹

¹Institute of Medical Chemistry, Biochemistry and Clinical Biochemistry, Medical Faculty, Comenius University, Bratislava, Slovakia

Antioxidants in Down syndrome

Ira T. Lott *

Biochimica et Biophysica Acta 1822 (2012) 657–663

Department of Pediatrics and Neurology, School of Medicine, University of California Irvine (UCI), Orange, CA 92868, USA

Timing of therapies for Down syndrome: the sooner, the better

Fiorenza Stagni, Andrea Giacomini, Sandra Guidi, Elisabetta Ciani and Renata Bartesaghi *

Department of Biomedical and Neuromotor Sciences, University of Bologna, Bologna, Italy

“Review findings from clinical trials of vitamin E, antioxidants, and other dietary approaches or nutritional supplements in individuals with Alzheimer’s disease and/or Down syndrome” (2014)
Nutritional interventions to target trisomy 21 effects?

Some Antioxidants Tested to Date:

- Epigallocatechin-3-gallate (ECGC): Green Tea Extract
- Alpha-Tocopherol: Vitamin E
- Ascorbic Acid: Vitamin C
- Alpha-Lipoic Acid
- Co-Enzyme Q10
- Etc. (list not exhaustive)

These studies have yielded inconsistent results.
This may be partly because:

- Different effects seen in cell and animal models and humans
- Differences in protocol:
  - Dosage
  - Timing of exposure
  - Confounding from genetic variation?
Pharmacological interventions for cognitive decline in people with Down syndrome

Nuala Livingstone¹, Jennifer Hanratty¹, Rupert McShane², Geraldine Macdonald³

¹School of Sociology, Social Policy and Social Work, Queen’s University Belfast, Belfast, UK. ²Radcliffe Department of Medicine, University of Oxford, Oxford, UK. ³School for Policy Studies, University of Bristol, Bristol, UK

Main results
Only nine studies (427 participants) met the inclusion criteria for this review. Four of these (192 participants) assessed the effectiveness of donepezil, two (139 participants) assessed memantine, one (21 participants) assessed simvastatin, one study (35 participants) assessed antioxidants, and one study (40 participants) assessed acetyl-L-carnitine.

Authors’ conclusions
Due to the low quality of the body of evidence in this review, it is difficult to draw conclusions about the effectiveness of any pharmacological intervention for cognitive decline in people with Down syndrome.

In short: We need more research
“Reducing oxidative damage... may require a *multitargeted approach that is preventative* in nature, given that supplementing... adults with DS with antioxidants has shown little or no benefit...

...A combinatorial approach may be particularly valuable for adults with DS who may benefit from a supplement including both antioxidants (e.g. vitamins E and C) and mitochondrial cofactors (e.g., lipoic acid, acetylcarnitine)...

...However, it may be critical to use these approaches as a *preventative measure* rather than as a treatment protocol for AD in DS””
Personalized Dietary Advice

vs

Public Health Recommendations
I have the gene, so I eat healthily.

I have the gene, so what can I do?
Does genetic information influence behaviour?

DNA-based dietary advice resulted in:
- greater understanding of recommendations
- greater interest in learning more
- greater motivation to change eating habits
Greater compliance after 1 year.
An Intervention Study of Individual, apoE Genotype-Based Dietary and Physical-Activity Advice: Impact on Health Behavior

Hanna-Leena Hietaranta-Luoma, Raija Tahvonen, Terhi Iso-Touru, Hannu Puolijoki, Anu Hopia

Effect of an Internet-based, personalized nutrition randomized trial on dietary changes associated with the Mediterranean diet: the Food4Me Study


Can genetic-based advice help you lose weight? Findings from the Food4Me European randomized controlled trial


Diet and exercise changes following direct-to-consumer personal genomic testing

Daiva Elena Nielsen¹,²†, Deanna Alexis Carere³†, Catharine Wang⁴, J. Scott Roberts⁵, Robert C. Green¹,²,⁶,⁷*, for the PGen Study Group
Replication: behaviour change

TAKING ACTION
After receiving genomics results, 42% of 1,051 surveyed people reported positive changes in their health behaviour. Only 1% of all respondents altered a prescription treatment without consulting a doctor.

Changes*
- No change: 58%
- Dietary patterns: 72%
- Exercise habits: 61%
- Supplements: 17% with medical consultation, 21% without medical consultation
- Non-prescription drugs: 10% with medical consultation, 7% without medical consultation
- Prescription drugs: 11% with medical consultation, 2% without medical consultation

*Many respondents reported more than one change, so percentages total more than 100%.

Source: Preliminary data from PGen Study, 2012–13

Genes really do hold the key to fitting into your jeans: Diets personalised to our genetic makeup are far more effective, study finds.
COMPANIES ARE TRYING TO USE YOUR DNA AND BACTERIA TO GIVE YOU PERSONALIZED DIET ADVICE — HERE'S WHAT THE SCIENCE SAYS

In our never-ending quest to get healthy, there’s a constant, nagging hope that we’ll find a hidden key to fitness — some trick or piece of information that finally makes it easy to look and feel how we want.

That’s why bizarre diets take off and nutrition “breakthroughs”...
What the skeptics say

- Single SNPs are useless.
- People won’t change their behaviors.
- It’s the microbiome.
Host genetic variation impacts microbiome composition across human body sites

Ran Blekhman¹,²*, Julia K. Goodrich³,⁴, Katherine Huang⁵, Qi Sun⁶, Robert Bukowski⁶, Jordana T. Bell⁷, Timothy D. Spector⁷, Alon Keinan⁸, Ruth E. Ley³,⁴, Dirk Gevers⁵,⁹ and Andrew G. Clark³

Genetic Determinants of the Gut Microbiome in UK Twins

Julia K. Goodrich,¹ Emily R. Davenport,¹ Michelle Beaumont,² Matthew A. Jackson,² Rob Knight,³ Carole Ober,⁴ Tim D. Spector,² Jordana T. Bell,² Andrew G. Clark,³ and Ruth E. Ley¹,⁵,∗

¹Department of Molecular Biology and Genetics, Cornell University, Ithaca, NY 14850, USA
²Department of Twin Research & Genetic Epidemiology, King’s College London, London SE1 7EH, UK
³Departments of Pediatrics and Computer Science and Engineering, University of California San Diego, La Jolla, CA 92030, USA
⁴Department of Human Genetics, University of Chicago, Chicago, IL 60637, USA
⁵Department of Microbiome Science, Max Planck Institute for Developmental Biology, 72076 Tübingen, Germany

*Correspondence: rle222@cornell.edu
http://dx.doi.org/10.1016/j.chom.2016.04.017

Cell Host & Microbe 19, 731–743, May 11, 2016
Association of host genome with intestinal microbial composition in a large healthy cohort

Williams Turpin¹², Osvaldo Espin-Garcia³⁴, Wei Xu⁴, Mark S Silverberg¹³, David Kevans¹², Michelle I Smith¹³, David S Guttman⁵⁶, Anne Griffiths⁷, Remo Panaccione⁸, Anthony Otley⁹, Lizhen Xu⁴¹⁰, Konstantin Shestopaloff⁴, Gabriel Moreno-Hagelsieb¹¹, GEM Project Research Consortium¹², Andrew D Paterson⁴¹⁰¹³ & Kenneth Croitoru¹³
What the skeptics say

- Single SNPs are useless.
- People won’t change their behaviors.
- It’s the microbiome.
- We need to integrate all of the ‘omics’ technologies.
- Biomarkers are more important.
- We need more evidence. From RCTs.
- Results from genetic tests are too complex.
- Family history is more informative.
- Just follow recommendations for healthy eating.
Where are we today?

- We need to eat......today.
- We currently give dietary advice for healthy eating.
- Current recommendations are based on (old) science.
- How much more evidence do we need?
Is DNA-based Dietary Advice Ready for Prime Time?

- Scientific evidence is robust (studies replicated)
- Independent of ethnic background
- Improved compliance (evidence from RCT)
- Information is actionable and “personalized”
- Increasing consumer awareness and demand
- Focus on wellness/prevention, not disease treatment
Visioning Report 2017: A Preferred Path Forward for the Nutrition and Dietetics Profession

Jana R. Kicklighter, PhD, RDN, LD, FAND; Becky Dorner, RDN, LD, FAND; Anne Marie Hunter, PhD, RDN, LD, FADA, FAND; Marcy Kyle, RDN, LD, CDE, FAND; Melissa Pflugh Prescott, PhD, RDN; Susan Roberts, MS, RDN, LD, CNSC; Bonnie Spear, PhD, RDN, FAND; Rosa K. Hand, MS, RDN, LD; Cecily Byrne, MS, RDN, LDN
“Continuing research and advances in genetics and nutritional genomics, with their ability to predict, prevent, and/or delay illnesses and chronic diseases, will become the mainstay of health care in the future.”

Recommendations:
- Create more professional development opportunities in nutrigenomics
- Include in standards and competencies for nutrition & dietetics practice

Kicklighter et al., JAND 2017
Proposed guidelines to evaluate scientific validity and evidence for genotype-based dietary advice

Nutritional Genomics: What a Dietitian Needs to Know

There is increasing awareness among researchers, educators, healthcare professionals and consumers that the one-size-fits-all, population-based approach to nutritional guidance is inefficient and sometimes ineffective. This awareness has created a growing market for personal genetic testing services. Research in the field of nutritional genomics (also called nutrigenomics) has provided important new insights into the role of human genetic variation in modifying the response to nutrients and food bioactives. Nutrigenomics, the focus of this course, uses genomic tools and genetic information to address issues important to nutrition and human health.

The primary goals of the course are to:
- Review the science of nutritional genomics, with a particular focus on how individual genetic differences (i.e. nutrigenetics) impact our response to the foods, beverages and supplements we consume.
- Provide examples of nutritional genomics research and give examples of their application to...
What to look for in a genetic test

- Does the test look at disease-associated genes or modifier genes?
- Are the results actionable?
- Are the recommendations personalized?
- Is adequate training and support provided?
- Is the science being interpreted correctly? Who developed the test?
- Is it offered DTC or only through a HCP?
- Are samples analyzed in a CLIA-certified lab?
Marketing firms name top 2017 food trends
Test Your Genes to Find Your Best Diet

Nutritional genetic testing can reveal what nutrients you’re missing and if you’re drinking too much coffee.
Ahmed El-Sohemy
Marilyn Cornelis
Daiva Nielsen
Bénédicte Fontaine-Bisson
Dennis Wang
Karen Eny
Leah Cahill
Ilana Platt
Stephen Ozsungur
Joanne Brathwaite
Christine Asik
Cristina Cuda
Sara Mahdavi
Hyeon-Joo Lee
Susana Huang
Lindsay Stewart
Alejandra Navarro-Allende
Nanci Guest
Joseph Jamnik
Andre Dias
Laura Da Costa
Karina Fischer
Andrea Josse
Lilli Mauer
Erica Day-Tasevski
Ohood Alhabri
Hannia Campos
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Canada Research Chairs

Nutrigenomix Inc.
“Positive health requires a knowledge of man’s primary constitution and of the powers of various foods, both those natural to them and those resulting from human skill.”

- Hippocrates (480 BC)

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