Nutrigenomics, Nutrigenetics Individual difference Personalized Diets

Gholamreza Mohammadi-Farsani, MD, PhD

mohammadigh53@gmail.com

Associate Professor of Nutritional Sciences

Department of Clinical Nutrition, School of Nutritional Sciences & Dietetics

Tehran University of Medical Sciences

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

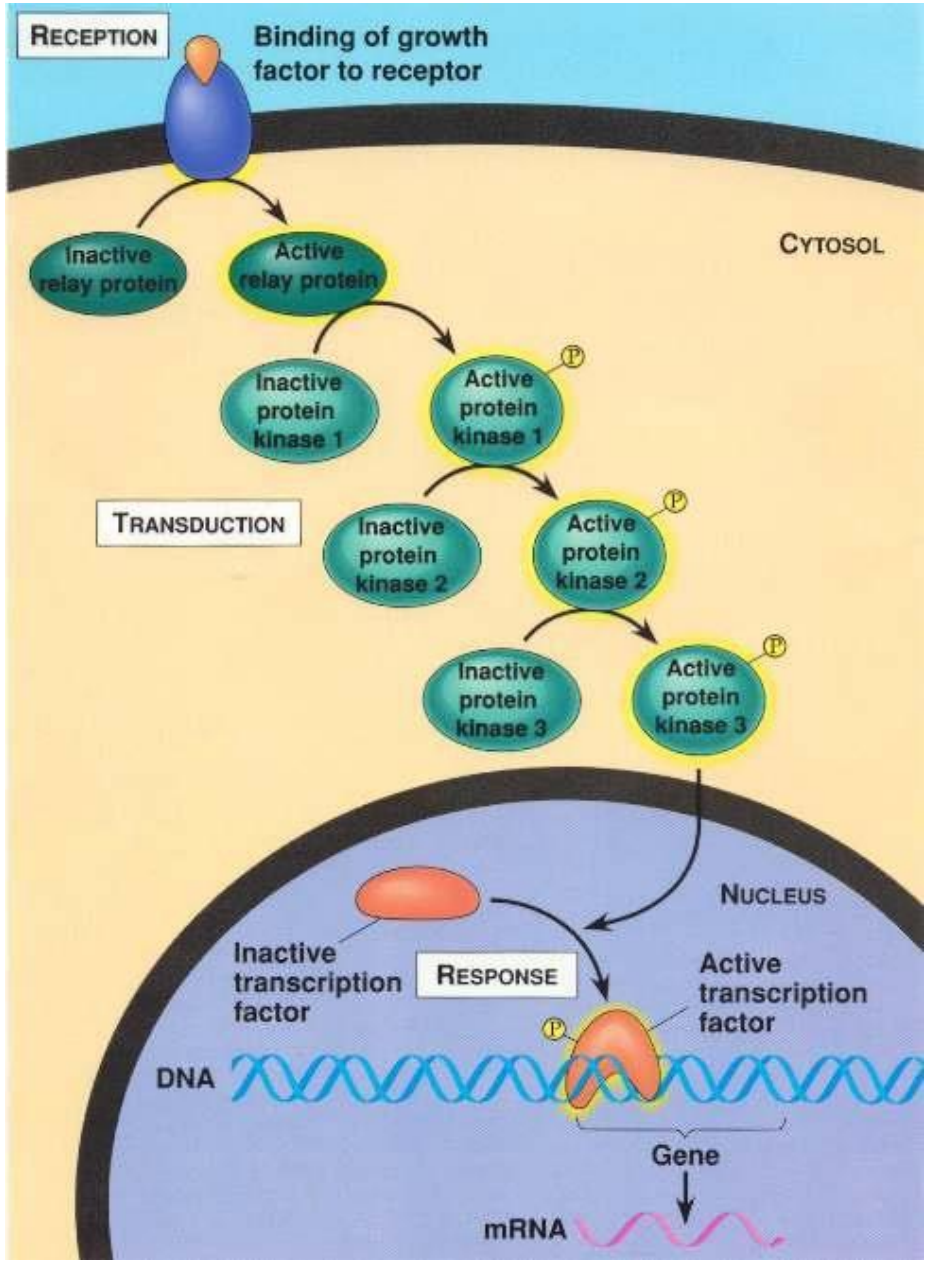
- Personalized Diets

Food Is…

- Medicine
- *Information*
- Connection

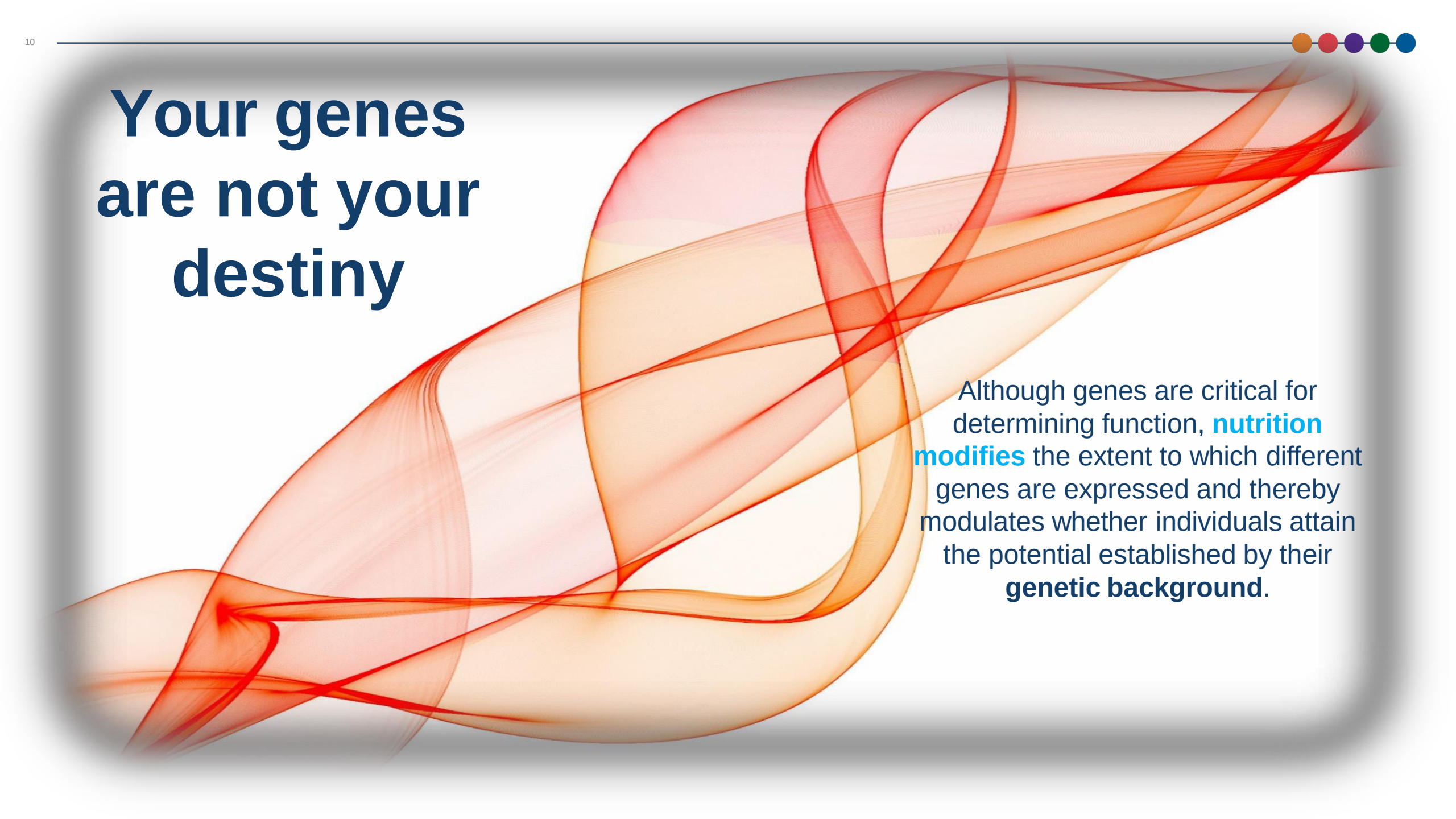
Nutrition in Clinical Practice 20:75-87, February 2005

Food sends informational signals to the genes

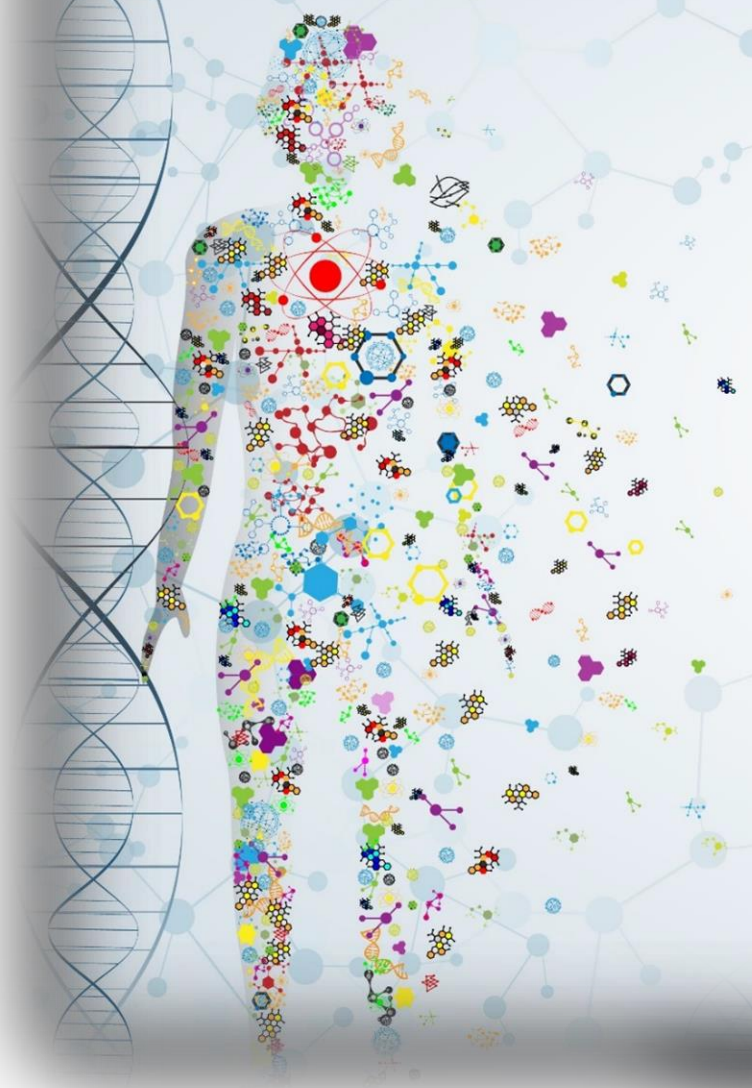


http://www.uic.edu/classes/bios/bios100/mike/spring2003/lect07.htm

Your genes are not your destiny



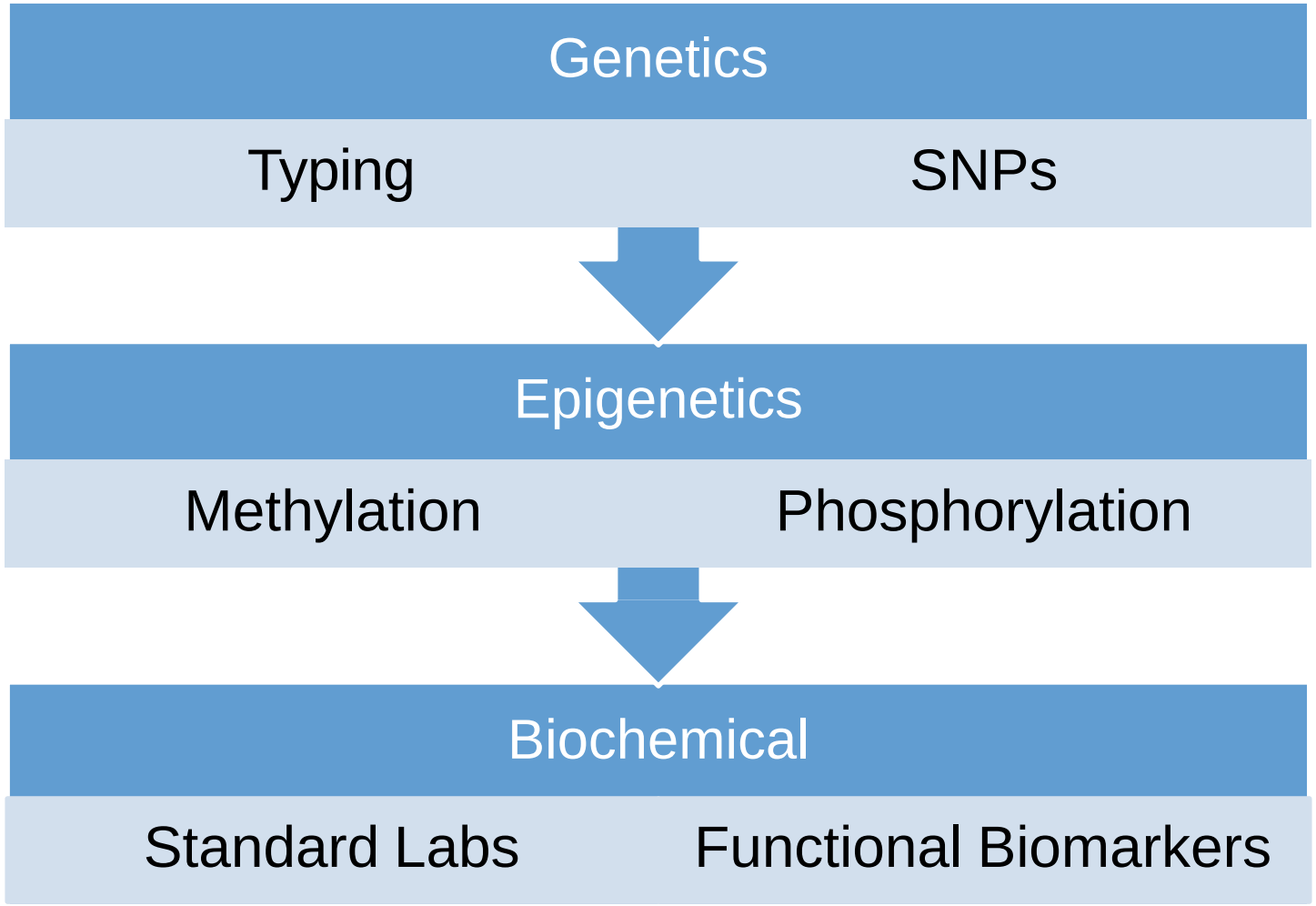
Although genes are critical for determining function, **nutrition modifies** the extent to which different genes are expressed and thereby modulates whether individuals attain the potential established by their **genetic background**.



Personalized Nutrition (Healthcare)

Targeted dietary prescriptions for the individual based on genetics and lifestyle

Levels of personalized, functional medicine-based health



Nutrition Journal

BioMed Central

Research

Open Access

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

Ioannis Arkadianos[1], Ana M Valdes[2], Efstathios Marinos[3], Anna Florou[1], Rosalynn D Gill[4] and Keith A Grimaldi*[4]

Address: [1]The Dr Arkadianos Clinic, Messogion Av, Athens, Greece, [2]Twin Research Unit, King's College London, UK, [3]Biomedical Engineering Laboratory, National Technical University of Athens, Greece and [4]Sciona Inc, Boulder, 80302, Colorado, USA

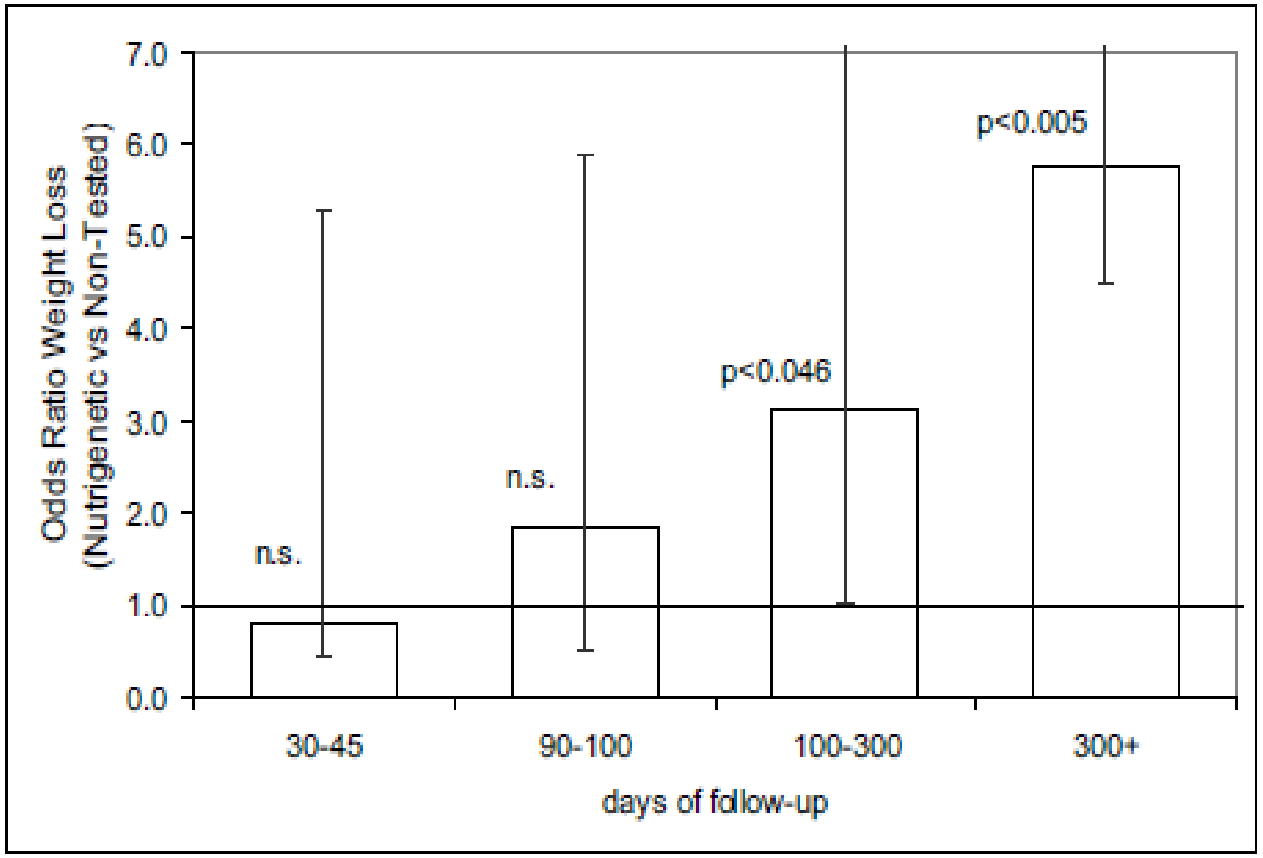
Email: Ioannis Arkadianos - idc.diet@eexi.gr; Ana M Valdes - ana.valdes@kcl.ac.uk; Efstathios Marinos - smarin@biomed.ntua.gr; Anna Florou - tsurekia@gmail.com; Rosalynn D Gill - rgill-garrison@sciona.com; Keith A Grimaldi* - kgrimaldi@sciona.com

* Corresponding author

- 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

Gene	Gene symbol
Angiotensin I converting enzyme	*ACE*
Apolipoprotein C-III	*APOC3*
Cystathionine-beta-synthase	*CBS*
Cholesteryl ester transfer protein	*CETP*
Collagen, type I, alpha 1	*COL1A1*
Glutathione S-transferase M1	*GSTM1*
Glutathione S-transferase pi	*GSTP1*
Glutathione S-transferase theta 1	*GSTT1*
Interleukin 6	*IL6*
Lipoprotein lipase	*LPL*
5-methyltetrahydrofolate-homocysteine methyltransferase reductase	*MTRR*
5,10-methylenetetrahydrofolate reductase	*MTHFR*
5-methyltetrahydrofolate-homocysteine methyltransferase	*MTR*
Nitric oxide synthase 3 (endothelial cell)	*NOS3*
Peroxisome proliferator-activated receptor gamma	*PPARG*
Superoxide dismutase 2, mitochondrial	*SOD2*
Superoxide dismutase 3, extracellular	*SOD3*
Tumor necrosis factor	*TNFα*
Vitamin D receptor	*VDR*

Nutrition Journal 2007, **6**:29 doi:10.1186/1475-2891-6-29



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%).

Nutrition Journal 2007, 6:29 doi:10.1186/1475-2891-6-29

Personalized Nutrition

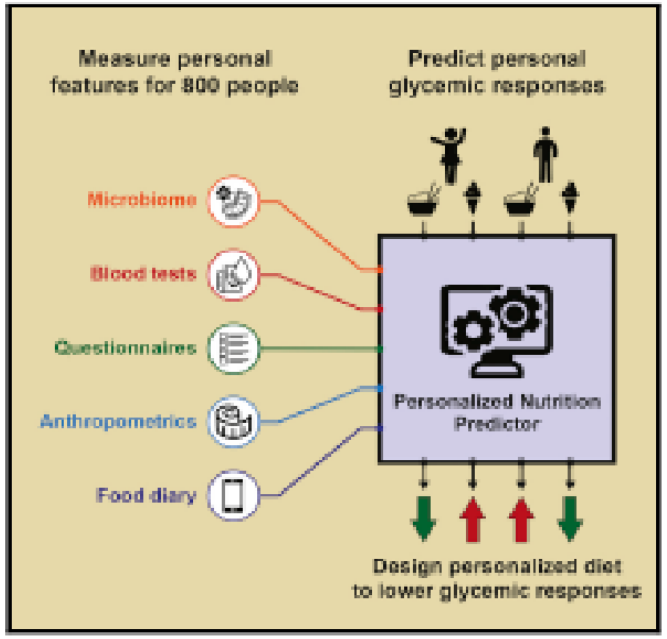
Here, we continuously monitored week-long glucose levels in an 800-person cohort, measured responses to 46,898 meals, and found high variability in the response to identical meals, suggesting that universal dietary recommendations may have limited utility.

Article

Cell

Personalized Nutrition by Prediction of Glycemic Responses

Graphical Abstract



Authors

David Zeevi, Tal Korem, Niv Zmora, ..., Zamir Halpern, Eran Elinav, Eran Segal

Correspondence

eran.elinav@weizmann.ac.il (E.E.), eran.segal@weizmann.ac.il (E.S.)

In Brief

People eating identical meals present high variability in post-meal blood glucose response. Personalized diets created with the help of an accurate predictor of blood glucose response that integrates parameters such as dietary habits, physical activity, and gut microbiota may successfully lower post-meal blood glucose and its long-term metabolic consequences.

Highlights

- High interpersonal variability in post-meal glucose observed in an 800-person cohort
- Using personal and microbiome features enables accurate glucose response prediction
- Prediction is accurate and superior to common practice in an independent cohort
- Short-term personalized dietary interventions successfully lower post-meal glucose

Cell. 2015 Nov 19;163(5):1079-94. doi: 10.1016/j.cell.2015.11.001.

Personalized Nutrition

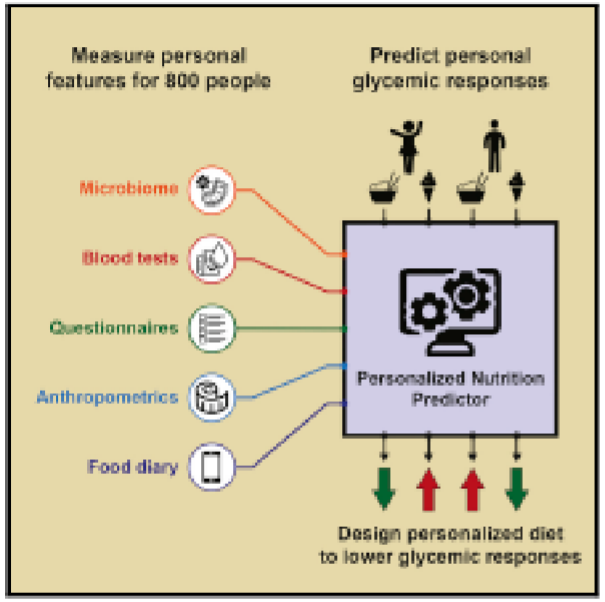
We devised a machine-learning algorithm that integrates **blood parameters**, **dietary habits, anthropometrics, physical activity, and gut microbiota** measured in this cohort and showed that it accurately predicts personalized postprandial glycemic response to real-life meals.

Article

Cell

Personalized Nutrition by Prediction of Glycemic Responses

Graphical Abstract



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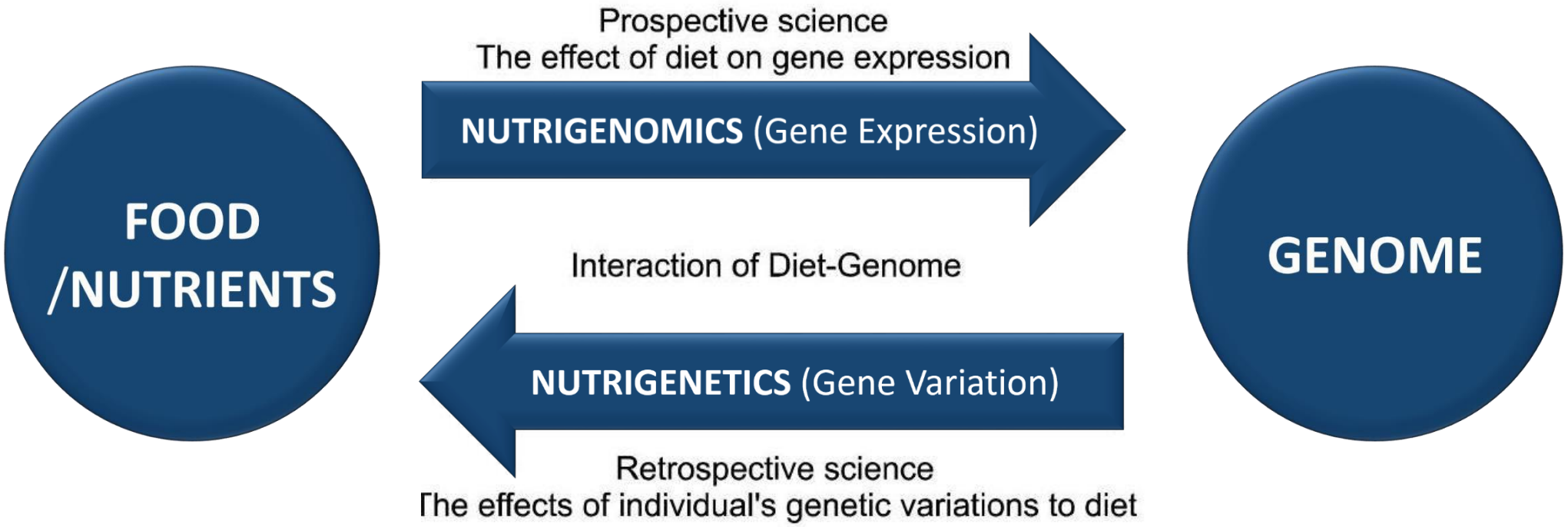
Nutrigenomics: The Overview



Prev Nutr Food Sci. 2015 Mar; 20(1): 1–7.

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

Nutrition in Clinical Practice 20:75-87, February 2005

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

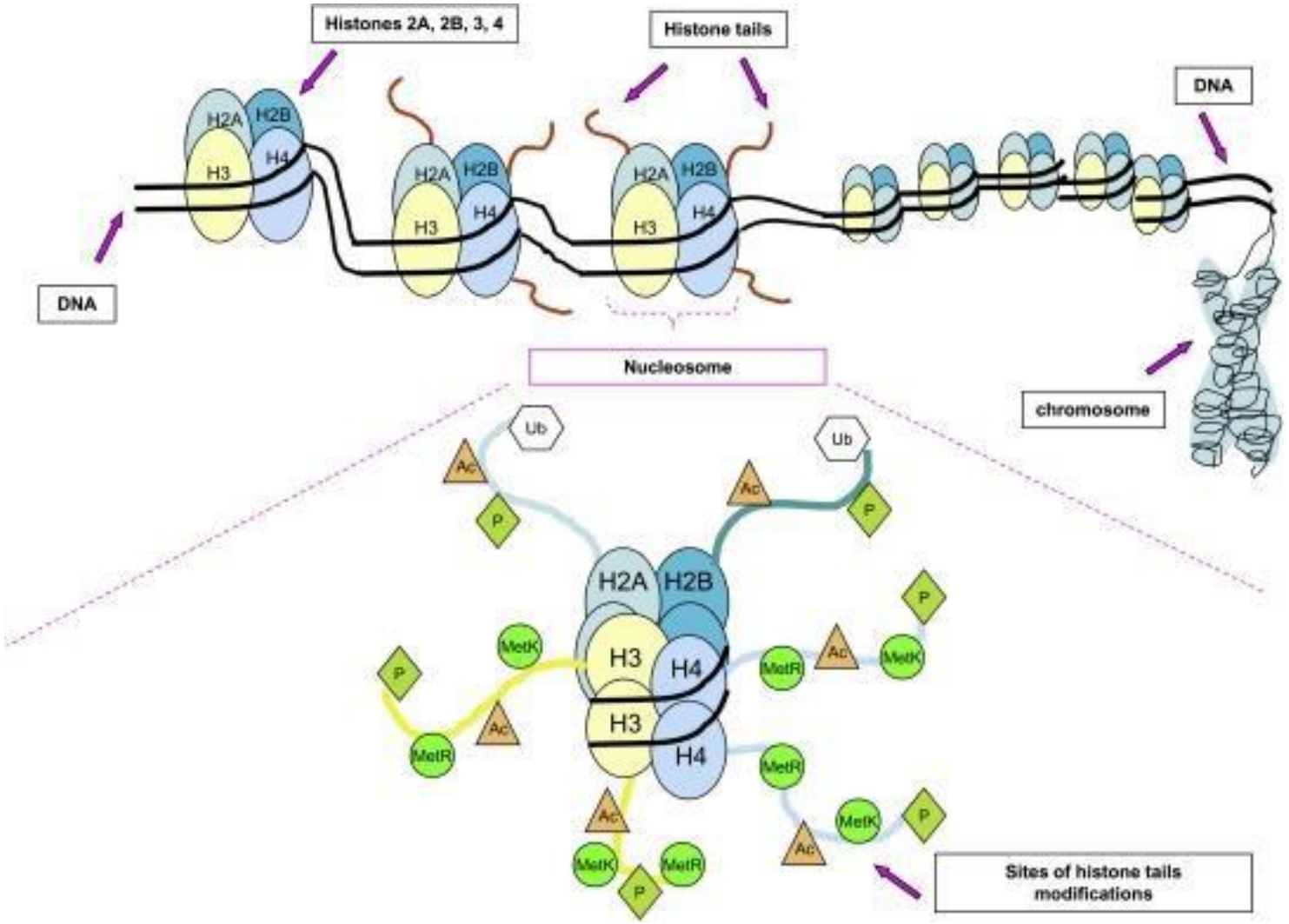
- Clinical trials need to include genetic variability in SNPs as a factor

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

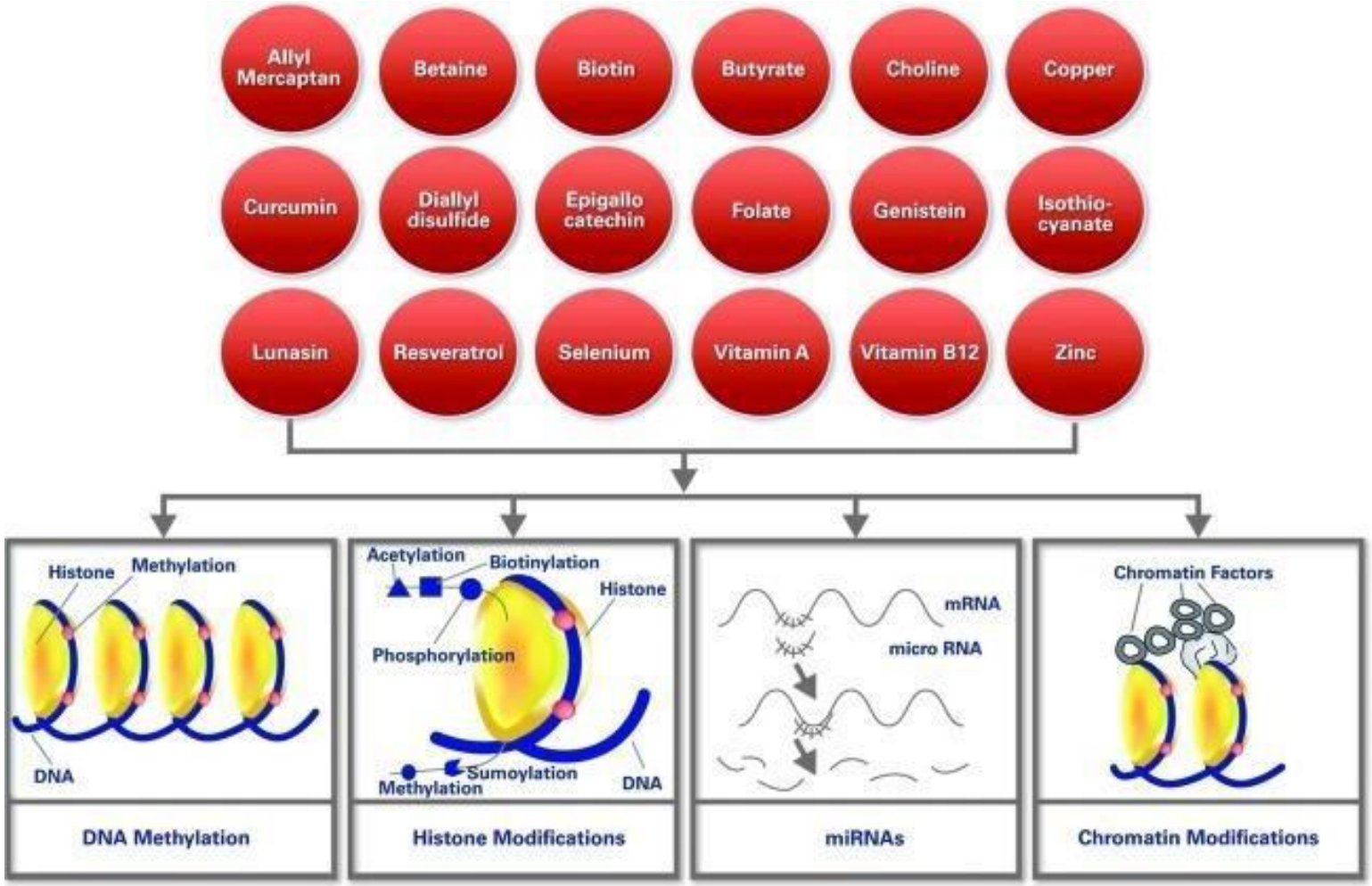
Epigenetics: The Wild Card

- Heritable changes that do not impact gene sequence.
- Modification to gene sites or histone proteins
  - Methylation
  - Phosphorylation
  - Acetylation
  - Ubiquinylation

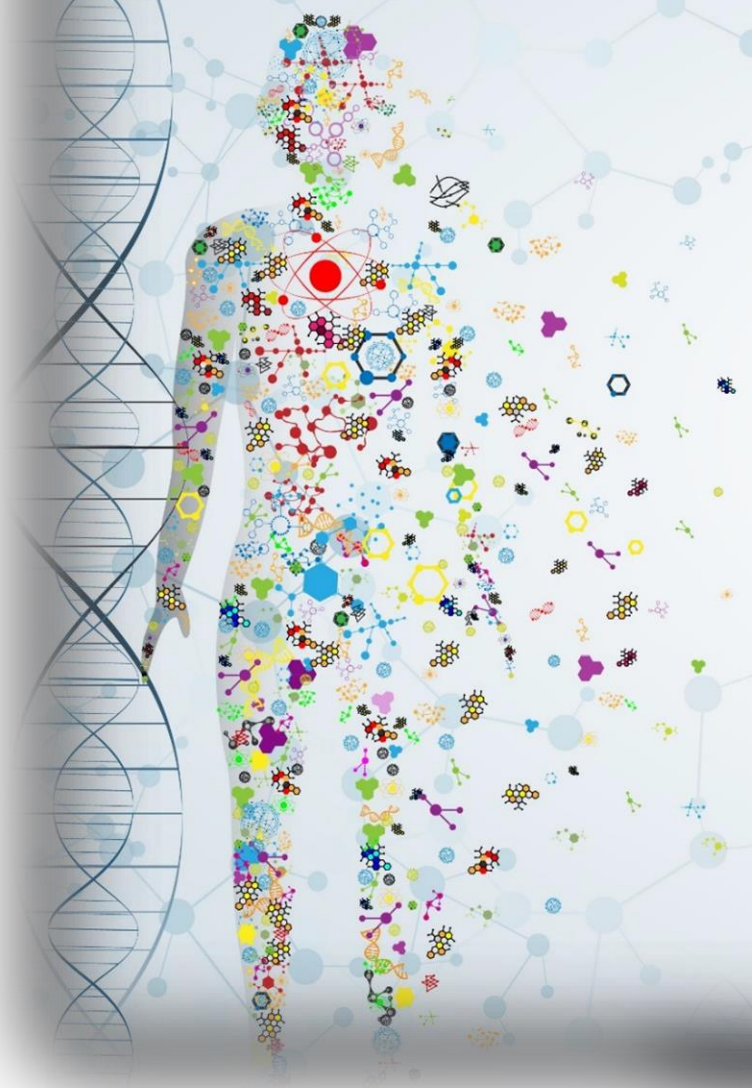


Nutrition in Clinical Practice 20:75-87, February 2005

Nutrients play a role in epigenetics



Antioxid Redox Signal. Jul 15, 2012; 17(2): 355–364.



Personalized Nutrition (Healthcare)

Targeted dietary prescriptions for the individual based on genetics and lifestyle

Nutrition & SNPs: Specific Profiles

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

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

What You Need to Know

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

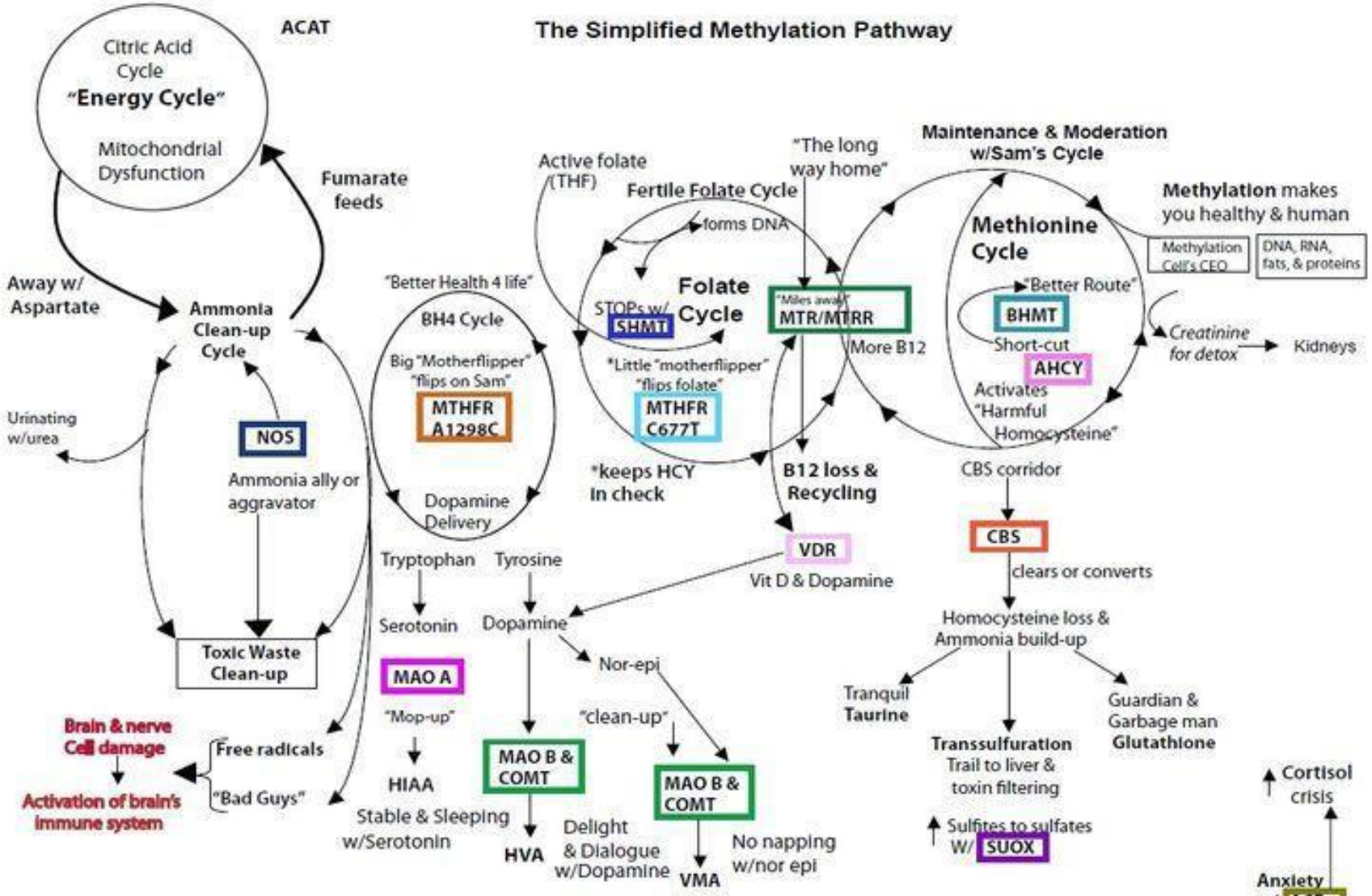
What You Need to Know

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

Methylenetetrahydrofolate reductase polymorphisms:

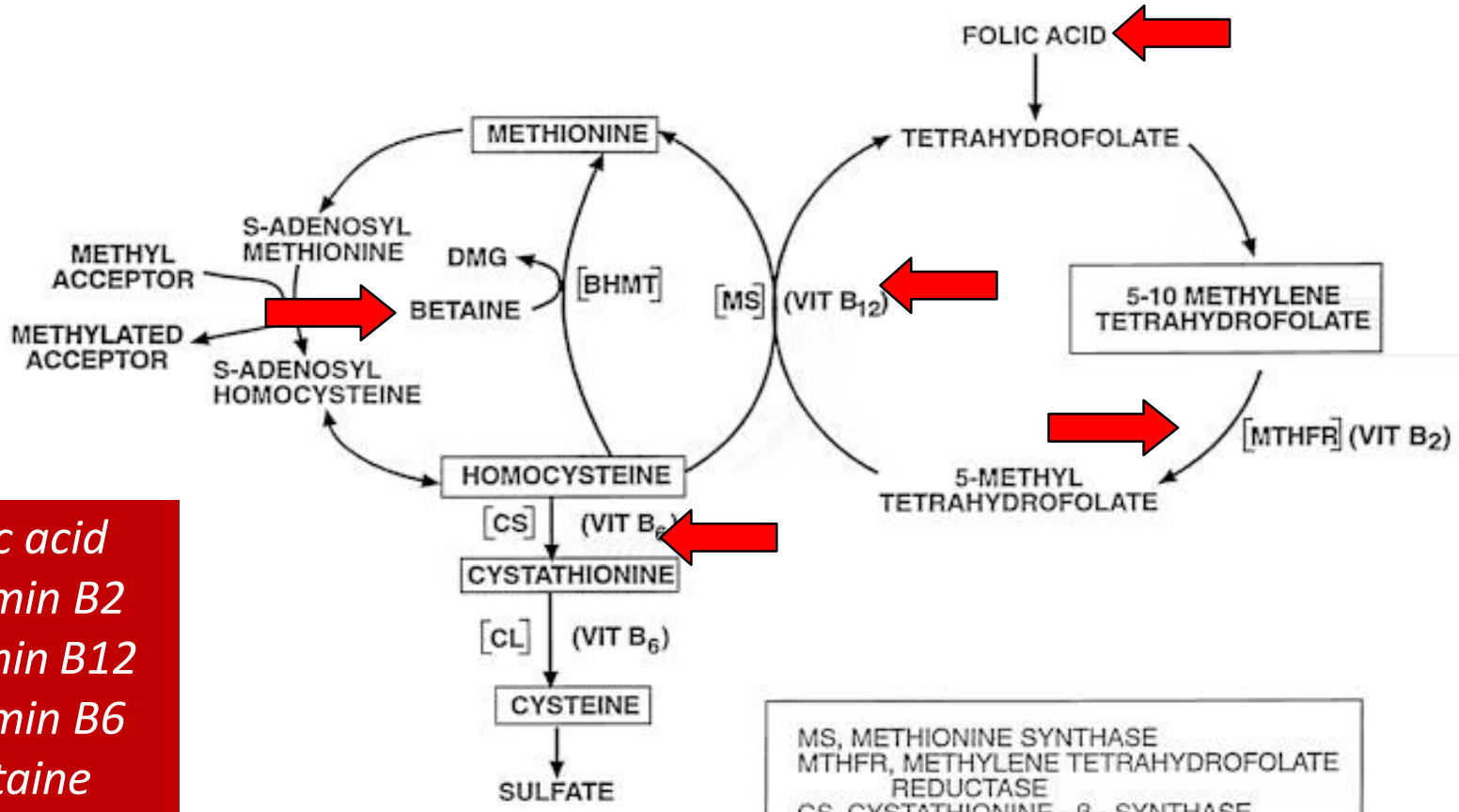
The BIGGEST SNP in Functional Medicine!



Adapted from the Neurological Research Institute's Diagram and simplified by April Ward-Hauge MS, NP

The Yasko Hypothesis of neurological & autoimmune disorders

Your health depends on the transfer of a 1-carbon unit and this transfer depends on nutrients!



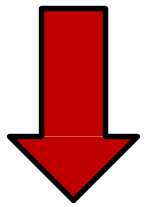
Folic acid
Vitamin B2
Vitamin B12
Vitamin B6
Betaine

Disease risk is most pronounced for the homozygous genotype for C677T

Copy of the MTHFR gene:

- C677T

Wild type	-/-	Full strength of the enzyme
Heterozygous	+/-	Enzyme reduced 30-40%
Homozygous	+/+	Enzyme reduced 60-70%

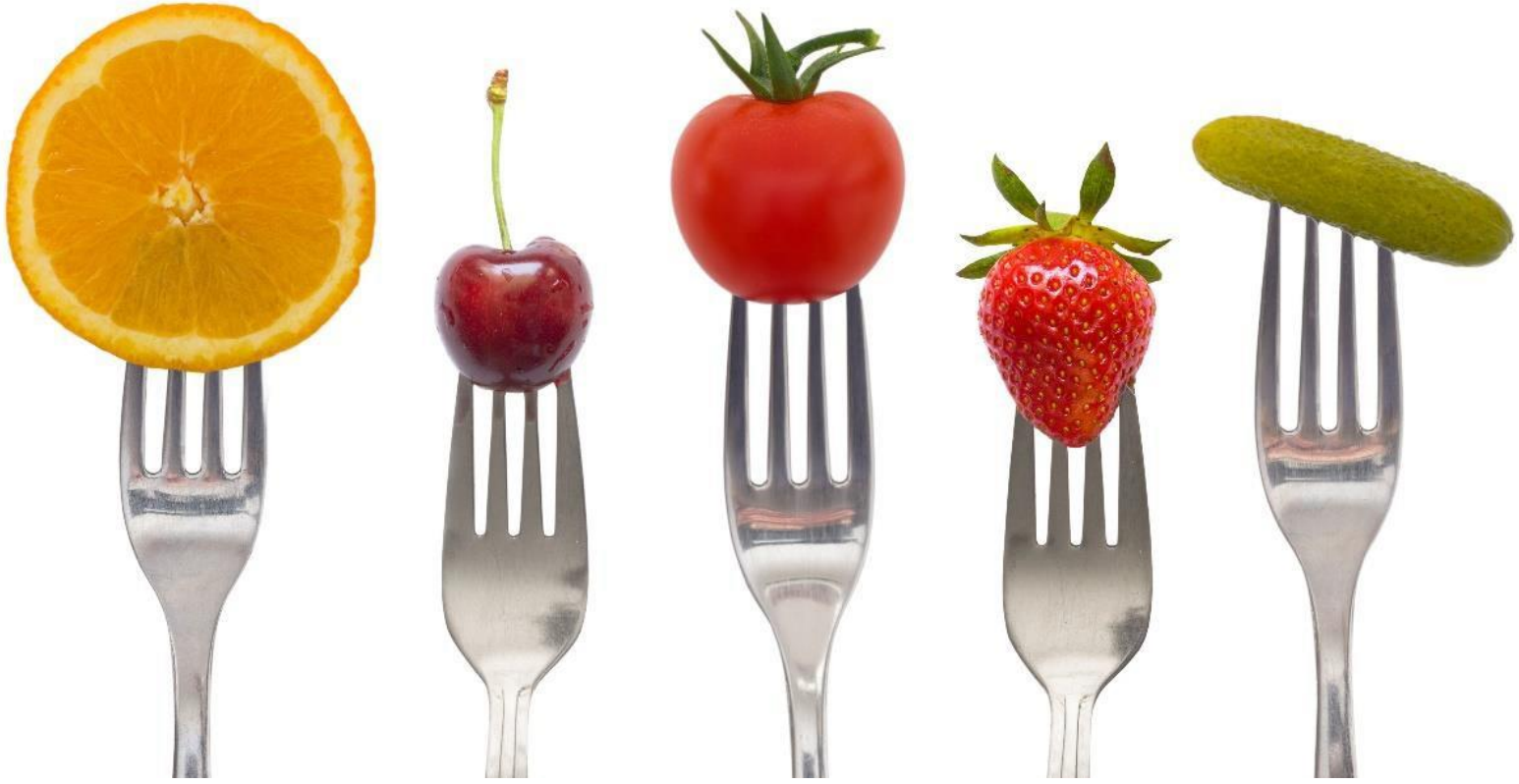


Reduced methylation

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

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

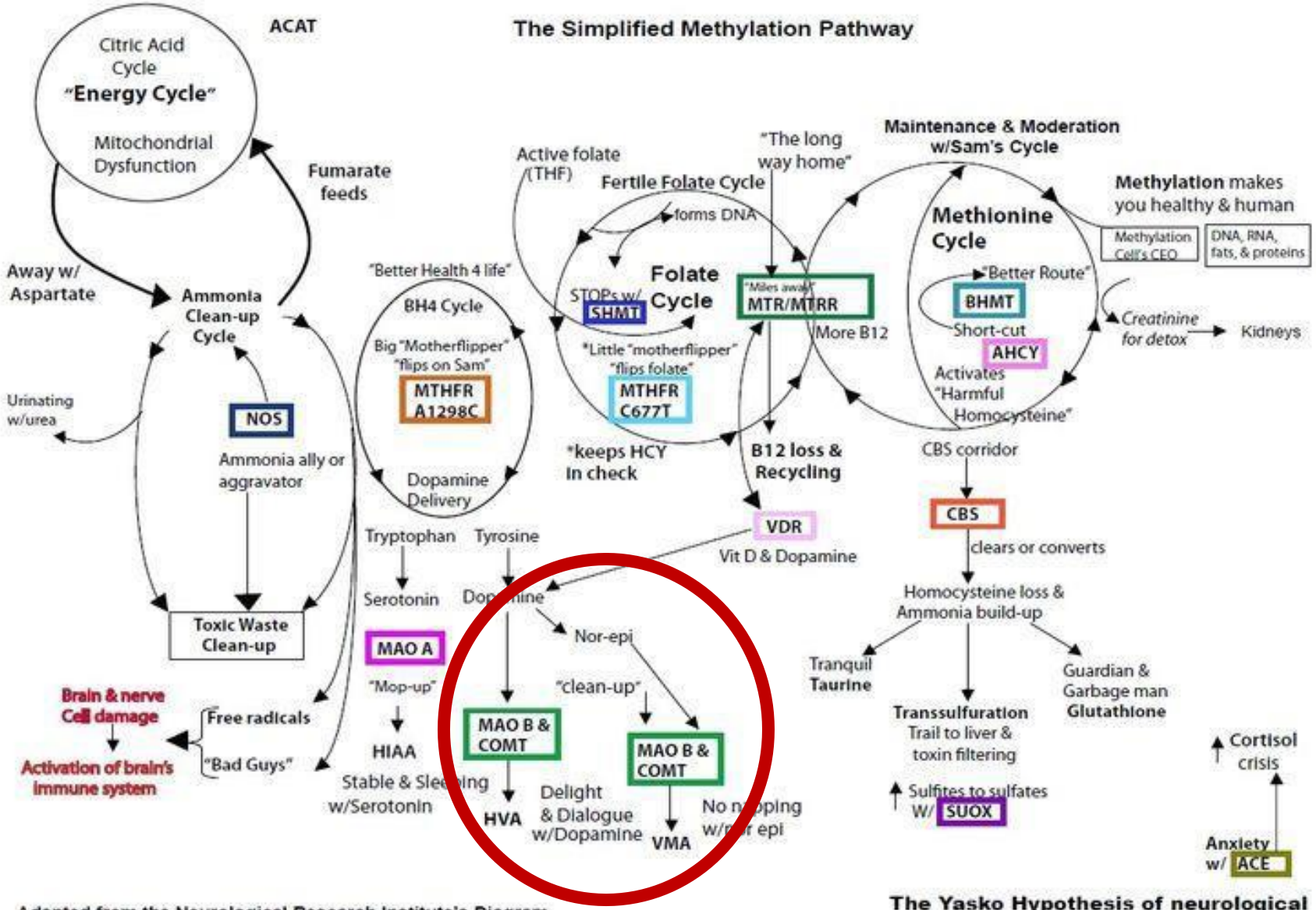
Catechol-O-Methyltransferase:

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


The Simplified Methylation Pathway
ACAT
Citric Acid Cycle
"Energy Cycle"
Mitochondrial Dysfunction
Fumarate feeds
Away w/ Aspartate
Ammonia Clean-up Cycle
Urinating w/urea
NOS
Ammonia ally or aggravator
Toxic Waste Clean-up
Brain & nerve Cell damage
Free radicals
Activation of brain's immune system
"Bad Guys"
"Better Health 4 life"
BH4 Cycle
Big "Motherflipper" "flips on Sam"
MTHFR A1298C
Dopamine Delivery
Tryptophan
Tyrosine
Serotonin
Dopamine
MAO A
"Mop-up"
HIAA
Stable & Sleeping w/Serotonin
Nor-epi
"clean-up"
MAO B & COMT
HVA
Delight & Dialogue w/Dopamine
MAO B & COMT
VMA
No napping w/Nor epi
Active folate (THF)
Fertile Folate Cycle
forms DNA
"The long way home"
STOPs w/ SHMT
Folate Cycle
"Miles away"
MTR/MTRR
More B12
*Little "motherflipper" "flips folate"
MTHFR C677T
*keeps HCY in check
B12 loss & Recycling
VDR
Vit D & Dopamine
Maintenance & Moderation w/Sam's Cycle
Methionine Cycle
"Better Route"
BHMT
Short-cut
AHCY
Activates "Harmful Homocysteine"
CBS corridor
CBS
clears or converts
Homocysteine loss & Ammonia build-up
Tranquil Taurine
Transsulfuration Trail to liver & toxin filtering
Guardian & Garbage man Glutathione
Sulfites to sulfates W/ SUOX
Methylation makes you healthy & human
Methylation Cell's CEO
DNA, RNA, fats, & proteins
Creatinine for detox
Kidneys
Cortisol crisis
Anxiety w/ ACE
Adapted from the Neurological Research Institute's Diagram and simplified by April Ward-Hauge MS, NP
The Yasko Hypothesis of neurological & autoimmune disorders

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)

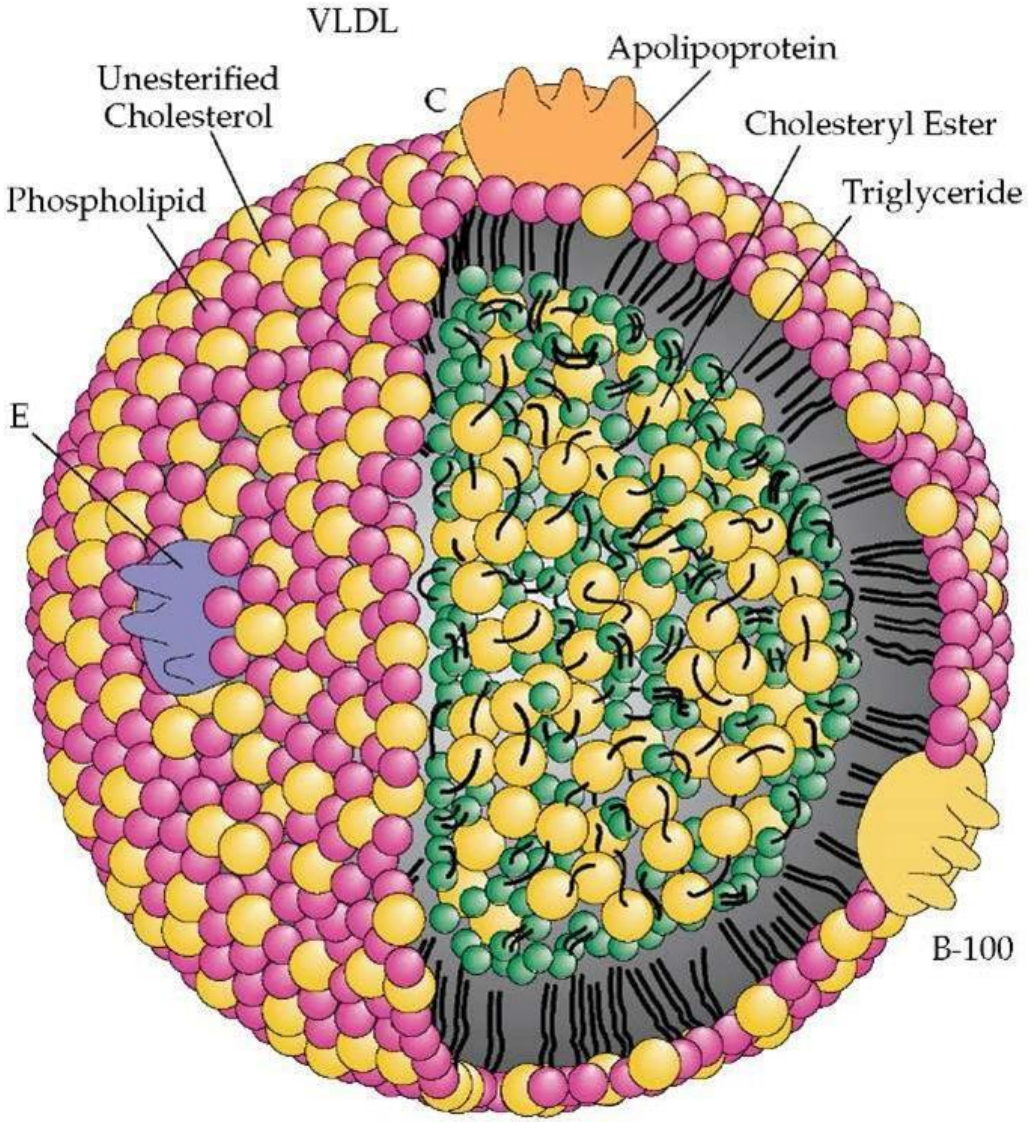
COMT 158V→M
+ + (Homozygous, most impaired)

Treatment Options

- **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


VLDL
Apolipoprotein
Unesterified
Cholesterol
C
Cholesteryl Ester
Triglyceride
Phospholipid
E
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

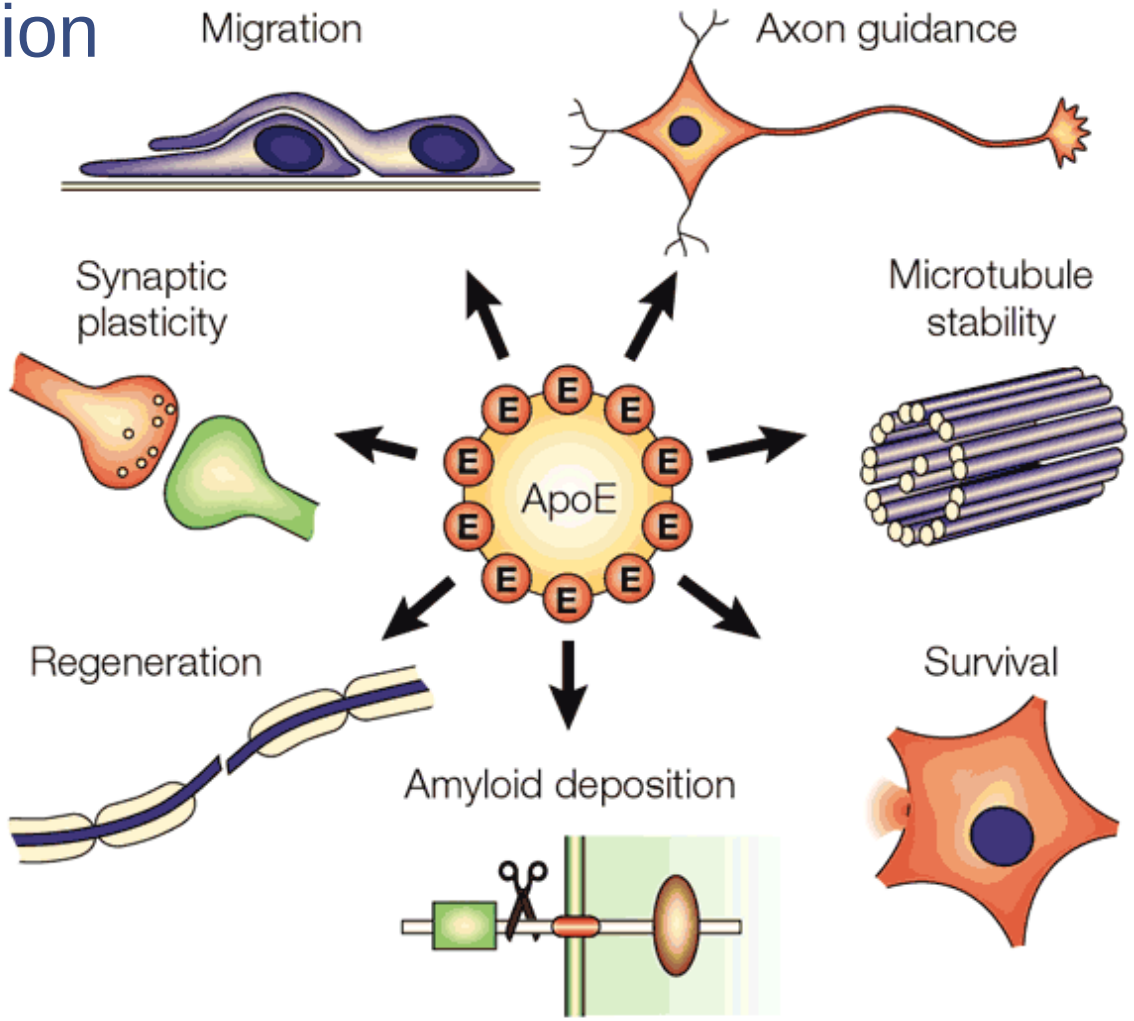
Apolipoprotein E:

Gene Variant Possibilities

- E2/E2
- E2/E3
- E2/E4
- E3/E3
- E3/E4
- E4/E4

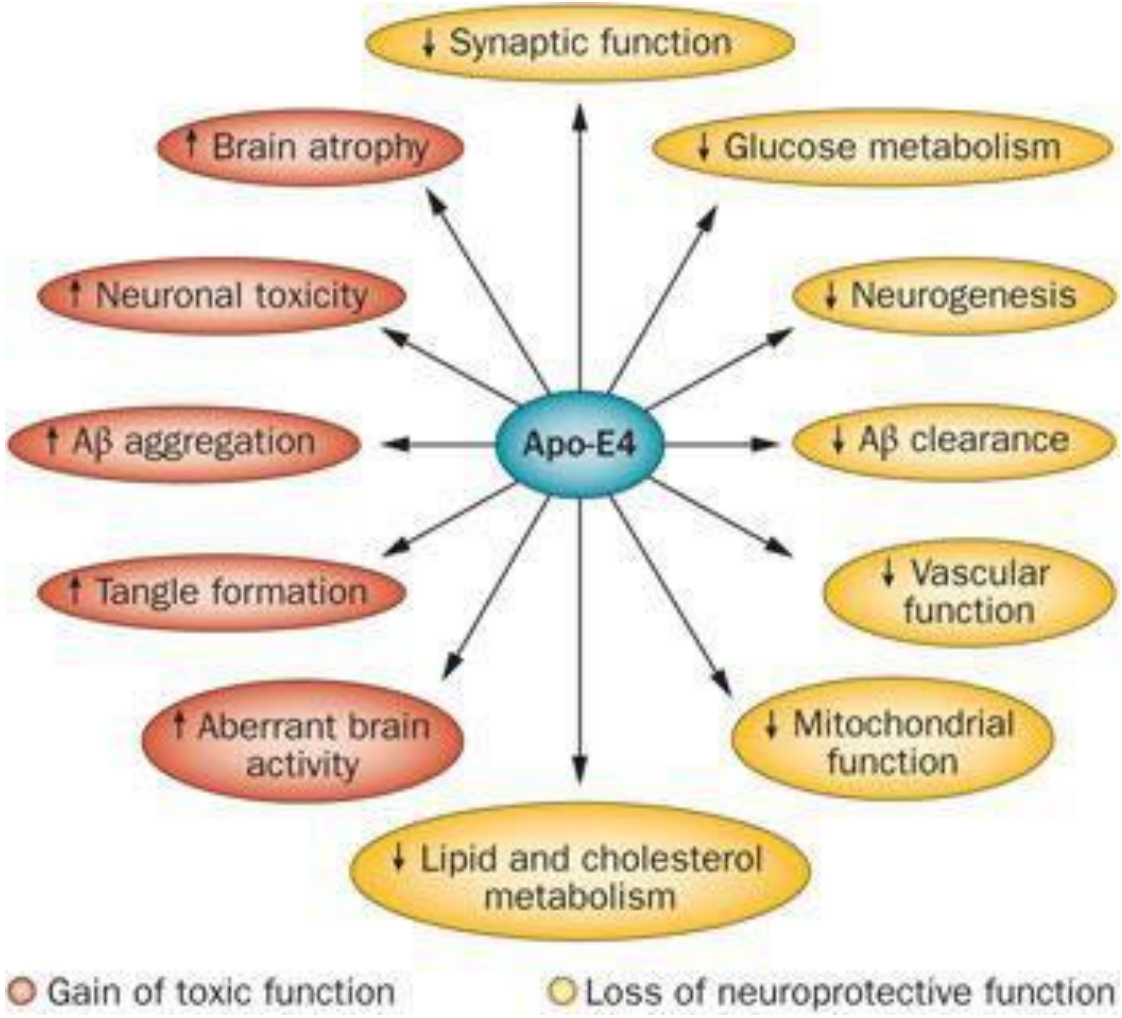
Apolipoprotein E:

Role in neuroscience & cognition



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

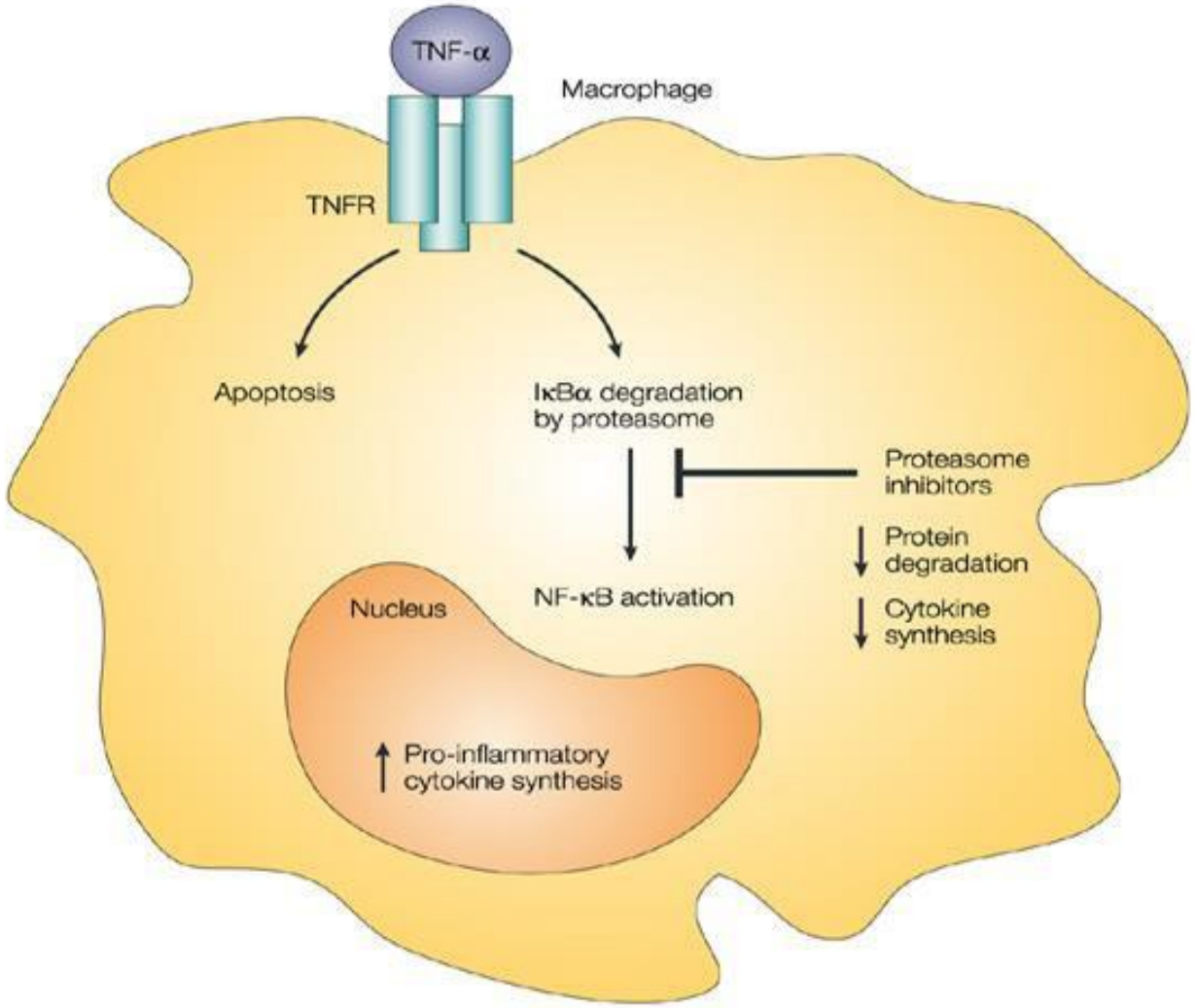
Treatment Options

- **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)

Tumor Necrosis Factor-alpha:

The inflammation ‘monster’

TNF-alpha



Nature Reviews | Drug Discovery

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

TNF-alpha:

Genetic variability

- **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

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

ویژگی های فردی:

- ژنوم و پروتئین های فرد
- میزان متابولیسم پایه و مصرف انرژی
- سیستم های هورمونی/آنزیمی
- فلور طبیعی دستگاه گوارش
- PH خون و عوامل موثر بر آن

سیستم های طبقه بندی فردی

از دیدگاه طب مکمل(طب ایرانی) بر اساس معیارهایی (طبع، مزاج...) افراد دارای ویژگیهای فردی اختصاصی هستند

تطابقی در این خصوص؟

Proteomics of hot-wet and cold-dry temperaments proposed in Iranian traditional medicine: a Network based Study

- Lack of molecular biology evidence has led clinical success of alternative and complementary medicine (CAM) to be marginalized. In turn, a large portion of life Science researchers could not communicate and help to develop therapeutic potential laid in these therapeutic approaches. In this study, we began to quantify descriptive classification theory in one of the CAM branches i.e. Iranian traditional medicine (ITM). Using proteomic tools and network analysis, the expressed proteins and their relationships were studied in mitochondrial lysate isolated from PBMCs from two different temperaments i.e. Hot-wet (HW) and Cold-dry (CD).

- The 82% of the identified proteins are over- or under-represented in distinct temperaments. Also, our result showed the different protein-protein interaction networks (PPIN) represented in these two temperaments using centrality and module finding analysis.

- Following the gene ontology and pathway enrichment analysis, we have found enriched biological terms in each group which are in conformity with the physiologically known evidence in ITM.

- In conclusion, we argued that the network biology which naturally consider life at the system level along with the different omics data will pave the way toward explicit delineation of the CAM activities.

Scientific **Reports** | 6:30133 | DOI: 10.1038/srep30133

Hot and Cold natures and some parameters of neuroendocrine and immune systems in traditional Iranian medicine: a preliminary study

- The purpose of this study was to assess differences in persons of a Hot or Cold nature (according to traditional Iranian medicine), in terms of changes in their neuroendocrine and immune systems.
- It can be deduced that the persons of a Hot nature had more sympathetic nervous system activity, less adrenal sympathetic, adrenal corticosteroid, and parasympathetic nervous system activities and more deviation of the immune system toward T-helper (Th)2 responses than the persons of a Cold nature. Moreover, the activity of the sympathetic nervous system was increased and adrenal sympathetic was decreased with an increasing Warmth/Coldness ratio. Furthermore, when the person's nature veered toward extreme Warmth or extreme Coldness, the deviation of the immune system toward Th2-like responses was greater, but this increased deviation was much more marked when veering toward extreme Warmth than toward extreme Coldness.

•PMID: 18307392 DOI: 10.1089/acm.2007.0693

The Evaluation of basic and neurohormonal parameters in hot or cold temperament person proposed in Iranian Traditional Medicine: an observational study

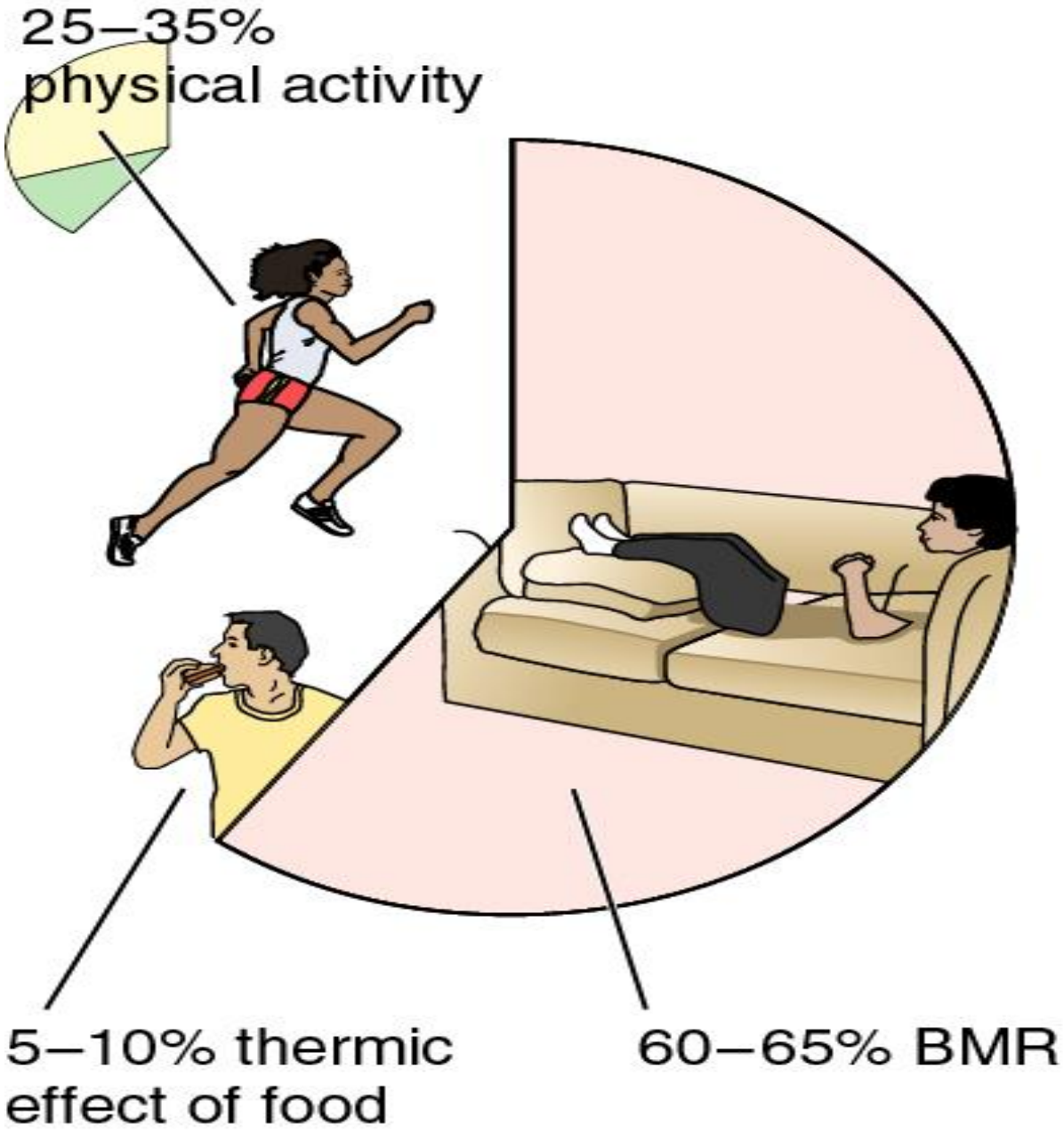
Table 1. **Factors affecting BMR & temperament.**

Variable	Basal metabolic rate	Temperament
Age	*High in the first and second year of life *Reduces 2–3% in each decade of life[1, 2]	*Hot temperament is more common in children and the young[4] *Temperament heat starts to decrease after the age of 35–40[5]
sex	In the same weight and height, BMR in women, is 5–10% less than men[1, 2]	Female temperament is colder than the male[5]
Body composition	*Muscle mass is the most significant determinant of BMR. *It increases BMR[1, 2]	*High muscle mass indicate shot temperament *High fat mass indicates cold temperament[4]
Body surface	Those who have more body surface, have higher BMR[1]	*High muscle mass indicate shot temperament *High fat mass indicates cold temperament[4]
The sympathetic-parasympathetic nervous system	Stimulating the sympathetic nervous system (e.g. stress) raises the BMR[1]	Some emotional conditions can increase body's hot temperament[6]
Thyroid function	*Hypothyroidism reduces the BMR *Hyperthyroidism increases the BMR[1, 3]	*Individuals with cold temperament feel cold more than others & tolerate heat better than the cold in normal conditions. *In hot temperament individuals, it is reversed[4, 6]

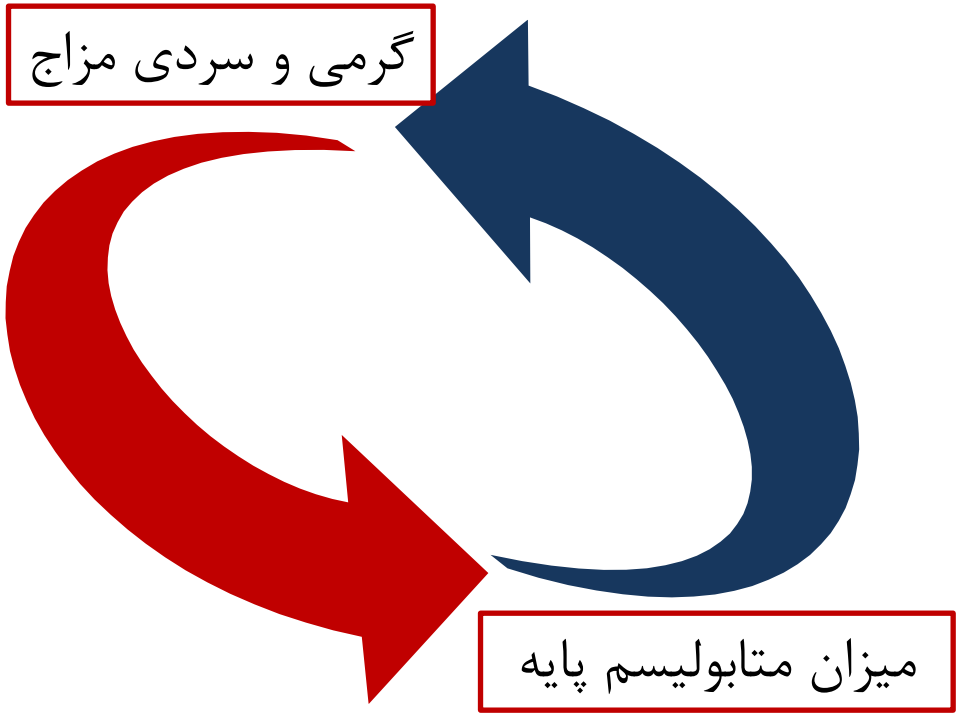
J Contemp Med Sci|Vol. 6, No. 4, July-August 2020: 176–180

Basal Metabolic Rate:

Components of Energy Expenditure

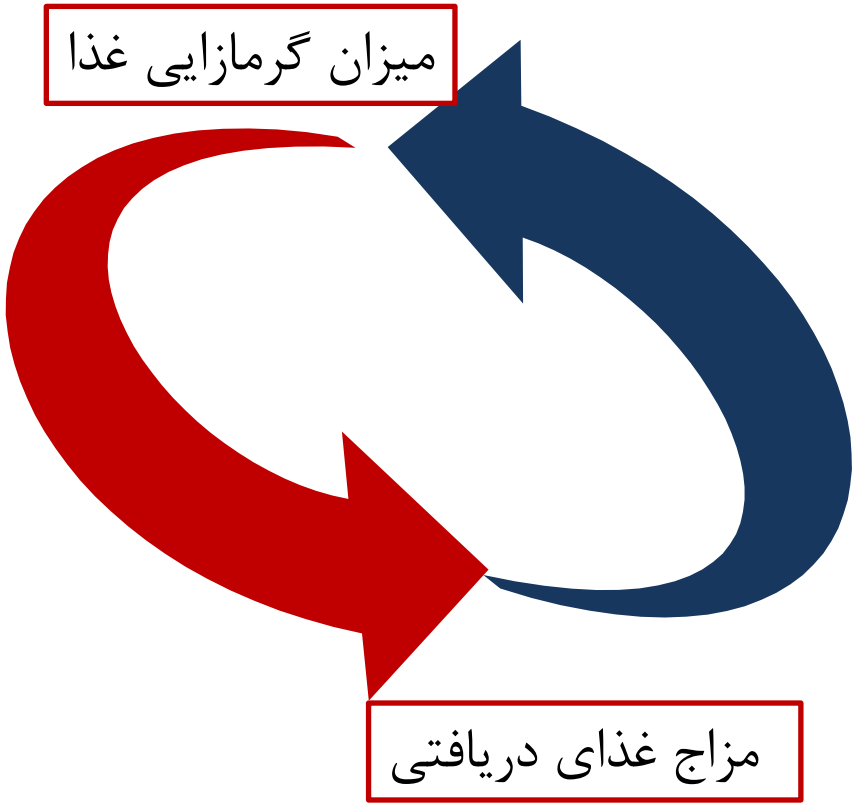


عوامل موثر بر میزان متابولیسم پایه و تطابق آن با تظاهرات گرمی و سردی مزاج


گرمی و سردی مزاج
میزان متابولیسم پایه

ارتباط گرمازایی غذا با مزاج غذا از دیدگاه طب سنتی

- ✓ اثر گرمازایی غذا (میزان متابولیسم فرد متعاقب خوردن غذا)
- ✓ مزاج غذا (تاثیر بر روی مزاج فرد)



The Evaluation of basic and neurohormonal parameters in hot or cold temperament person proposed in Iranian Traditional Medicine: an observational study

Table 2. **Body composition of participants based on temperament.**

Variable	Temperament of individuals				Significance statistical test*P value
	Cold temperament n=22		Hot temperament n=21		
	Mean	Standard deviation	Mean	Standard deviation	
Body fat mass(kg)	13.41	2.15	18.35	4.15	.001>
Body fat percentage	21.73	5.47	27.98	7.37	.003
Skeletal muscle mass (kg)	30.99	7.15	27.03	5.48	.047
Fate free mass percentage	78.49	5.72	72.02	7.37	.003
Body water (Lt)	36.14	8.34	37.41	6.47	.579
Body water percentage	55.70	6.23	56.11	462	.807

J Contemp Med Sci|Vol. 6, No. 4, July-August 2020: 176–180

The Evaluation of basic and neurohormonal parameters in hot or cold temperament person proposed in Iranian Traditional Medicine: an observational study

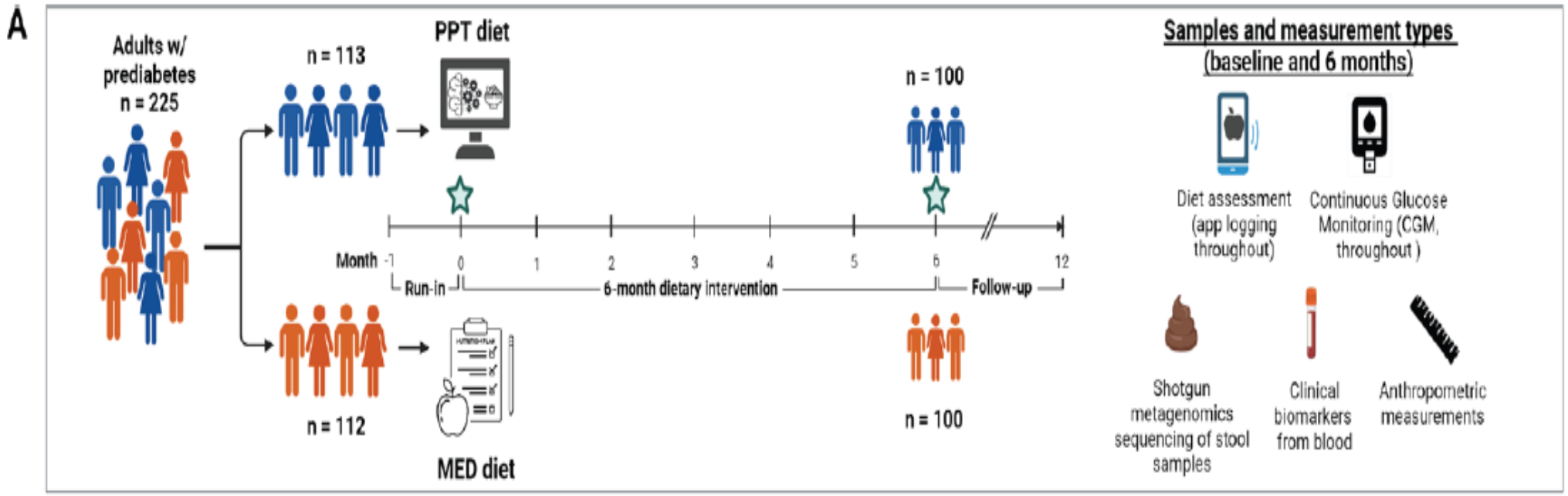
Table 3. **BMR and T3, T4, TSH levels based on temperament.**

Variable	Temperament of individuals				Significance statistical test*P value
	Cold temperament n=22		Hot temperament n=21		
	Mean	SD	Mean	SD	
Basal metabolic rate/Kgbw(kcal)	25.74	2.96	28.76	3.88	.006
Basal metabolic rate(kcal)	1664.09	252.04	1909.48	442.40	.030
T3	1.26	.13	1.40	.18	.008
T4	7.19	.48	7.29	.34	.457
TSH	1.99	1.24	1.38	.63	.049

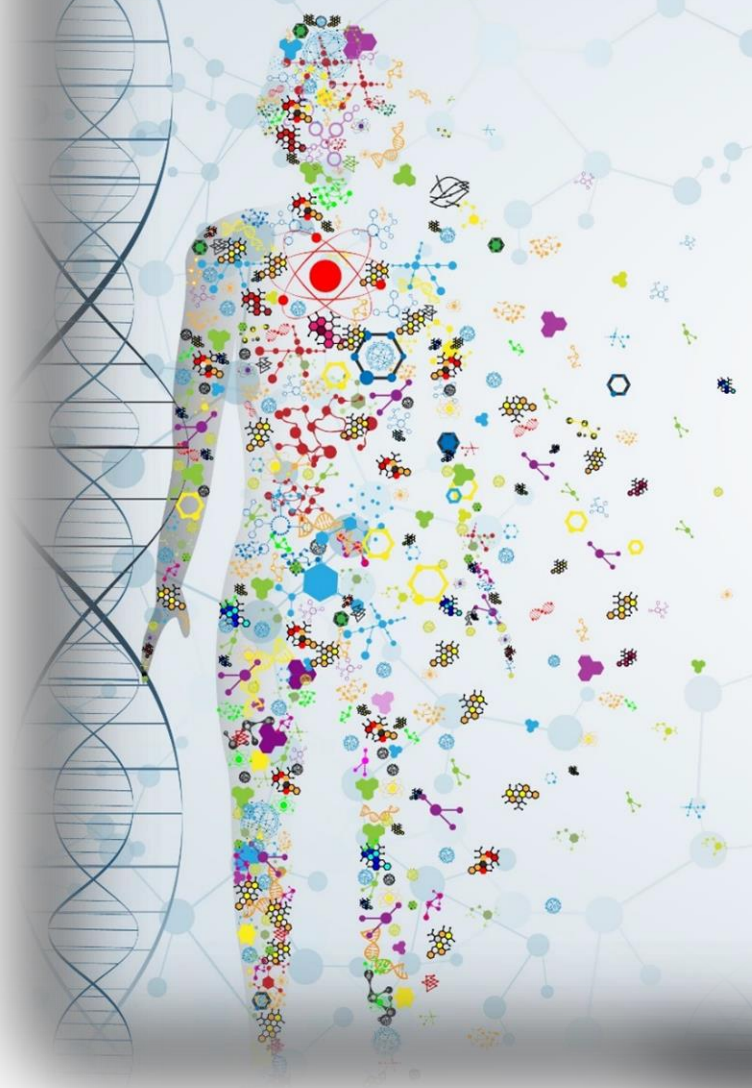
J Contemp Med Sci|Vol. 6, No. 4, July-August 2020: 176–180

Gut microbiome modulates the effects of a personalised postprandial-targeting (PPT) diet on cardiometabolic markers: a diet intervention in pre-diabetes

Gut microbiota



Ben-Yacov O, et al. Gut 2023;**72**:1486–1496. doi:10.1136/gutjnl-2022-329201



Personalized Nutrition (Healthcare)

Targeted dietary prescriptions for the individual based on genetics and lifestyle

جمع بندی

- بصورت فردی نگاه کردن به سلامت هر فرد(پیشگیری و درمان)
- توجه به کل نگری در خصوص سلامت فردی در هر فرد
- استفاده از معیارهایی در این خصوص (طب ایرانی ...)

از توجه شما سپاسگزارم

- بین مزاج فرد و میزان متابولیسم پایه ارتباط وجود دارد
- افراد گرم مزاج متابولیسم پایه بالاتر و سرد مزاج پایین تری دارند
- فعالیت تیروئید در افراد گرم مزاج بیشتر از سرد مزاج است
- فعالیت سمپاتیک می تواند در افراد گرم مزاج بیشتر از سرد مزاج باشد
- مزاج غذای دریافتی افراد می تواند در میزان متابولیسم و عملکرد سیستم عصبی فرد تاثیرگذار باشد .

عوامل موثر بر میزان متابولیسم پایه و تطابق آن با تظاهرات گرمی و سردی مزاج

✓ سن

متابولیسم پایه در سالهای اولیه زندگی بالا و پس از آن، در هر دهه از عمر حدود 2 الی3 در صد از میزان آن کم می شود

حرارات مزاج کودکان و جوانان بیشتر از حرارت مزاج کهول و مشایخ بوده و پس از سن 35 الی 40 سالگی شروع به کاهش می کند

✓ جنس

میزان متابولسم پایه در زنان 8 الی 10 درصد کمتر از مردان با همان وزن و قد است

مزاج زنان نسبت به مردان سردتر است

✓ ترکیب بدن

توده عضلانی بیشتر و چربی کمتر باعث افزایش متابولیسم پایه می گردد

کثرت و زیادتی لحم، دلالت بر حرارت (گرمی مزاج) و کثرت شحم و سمین دلالت بر برودت (سردی مزاج) می کند

عوامل موثر بر میزان متابولیسم پایه و تطابق آن با تظاهرات گرمی و سردی مزاج

✓اندازه بدن

کسانی که سطح بدنی بیشتری داشته باشند میزان متابولیسم بیشتری دارند
سعت و گشادگی صدر (وسعت و گشادگی قفسه سینه)، عظم اطراف (بزرگی اندام ها) و ظهور مفاصل (بزرگی و برجستگی مفاصل) دلالت بر گرمی مزاج بدن می نماید

✓محیط

میزان متابولیسم پایه کسانی که در آب و هوای گرمسیری زندگی می کنند، 5 تا 20 درصد بالاتر از افرادی است که در مناطق معتدل زندگی می کنند
تماس با گرم کننده های غیر شدید از قبیل هوای گرم سبب گرمی مزاج می شود
ورزش کردن بار متابولیکی را تا حدود 5 درصد زیاد می کند
حرکت معتدل که شامل ریاضات، دلک و غمز معتدل است، از جمله مسخنات (گرم کنده ها) بدن شمرده می شوند

عوامل موثر بر میزان متابولیسم پایه و تطابق آن با تظاهرات گرمی و سردی مزاج

✓ درجه حرارت بدن

تب میزان متابولیسم را تا حدود 7 درصد به ازای هر درجه افزایش دمای بدن بالا می برد
عفونت از اسباب گرم کننده بدن بوده و خاصیت آن ایجاد حرارت غریبه است

✓ سیستم عصبی

تحریک سیستم عصبی سمپاتیک (برای مثال در هیجان های احساسی یا استرس) سبب افزایش متابولیسم پایه می گردد
خشم و شادی معتدل از اسباب گرم کننده بدن شمرده می شوند و در مقابل آن اندوه و حتی شادی و لذت عظیم، سبب سرد شدن بدن می گردند

✓ هورمون ها (هورمون تیروئید)

در کم کاری تیروئید میزان متابولیسم پایه فرد کاهش و در پرکاری تیروئید، افزایش می یابد

- The Evaluation of basic and neurohormonal parameters in hot or cold temperament person proposed in Iranian Traditional Medicine: an observational study

https://www.jocms.org/index.php/jcms/article/view/709/421