Queen Mary
University of London
Medicine and Dentistry

Nutrition as the key to improved health: Nutrigenetics, nutraceuticals, and functional foods

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Outline

- What are the different dietary patterns and what is a food pyramid
- Which are the nutritional factors related to common diseases
- What is the planetary diet
- Which are the nutraceuticals and functional foods
- What is nutrigenetics and why investigate it
- How might gene-by-environment interactions modulate disease risk

Food groups

Vegetable

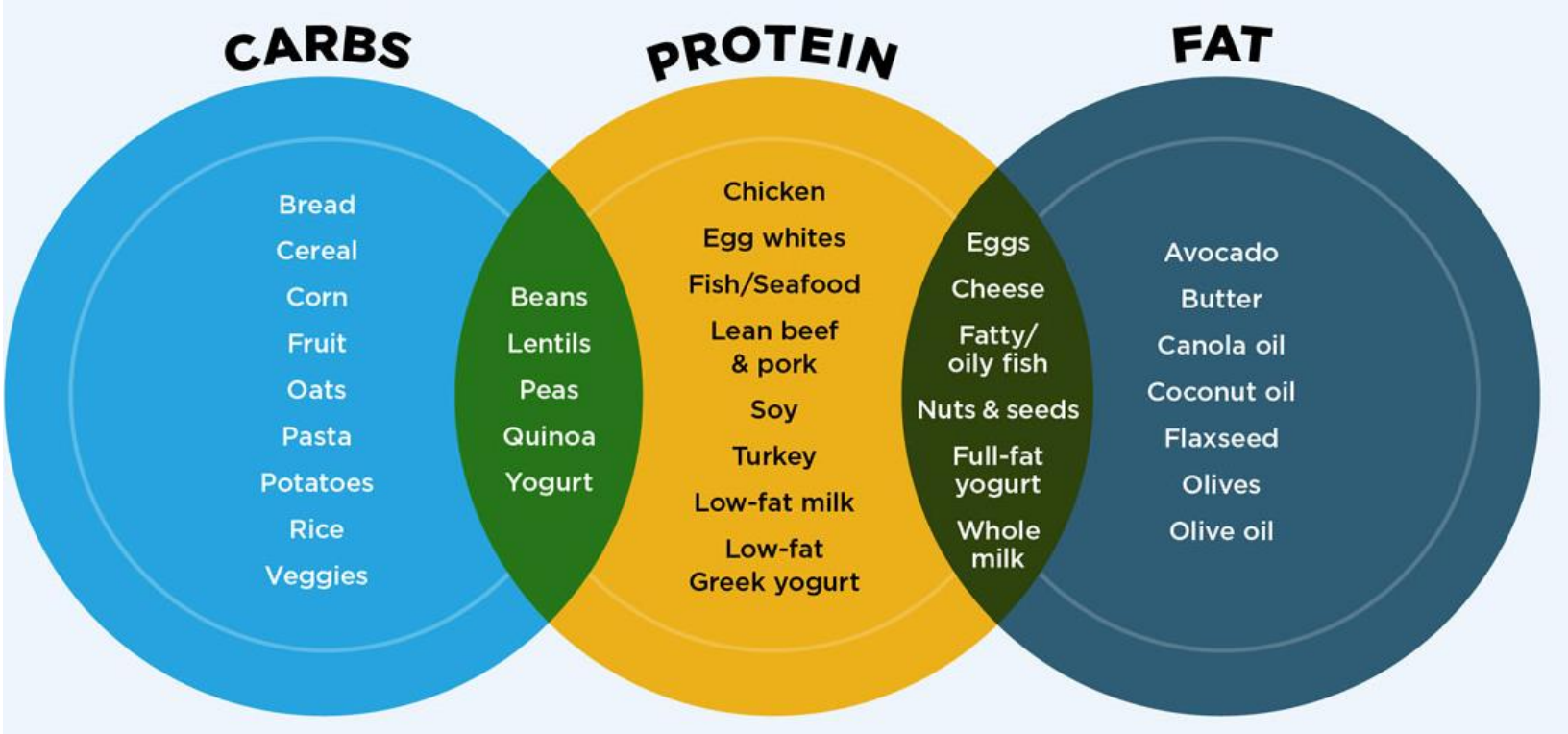
Dairy

Grains

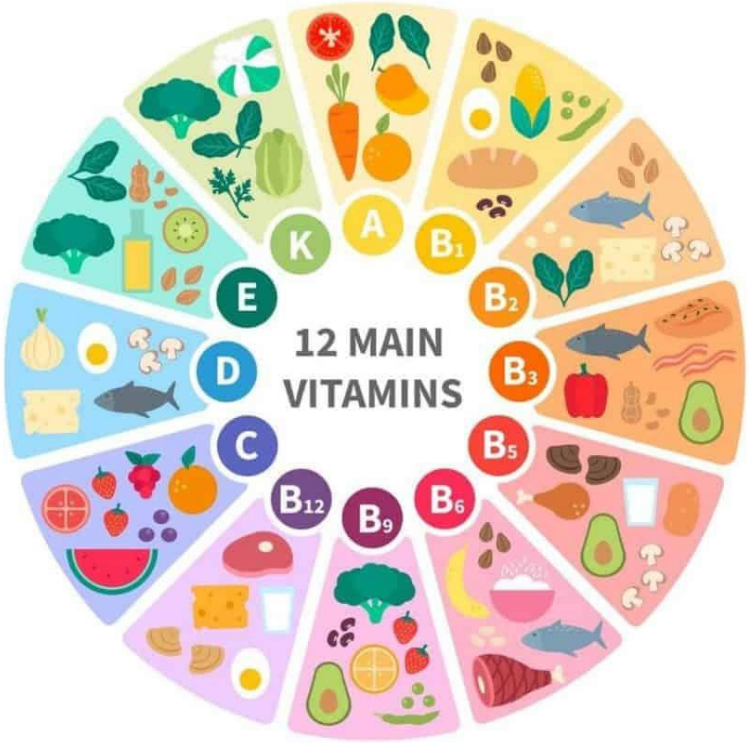
Protein (meat, eggs, fish, legumes)

Fruits

Nutrients - Macronutrients



Nutrients - Micronutrients



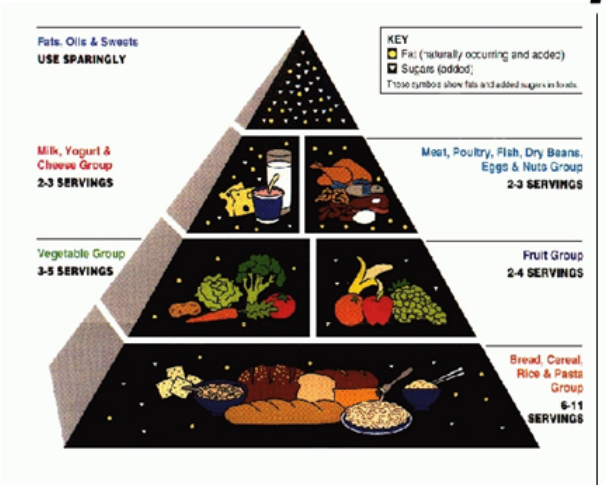
Dietary patterns

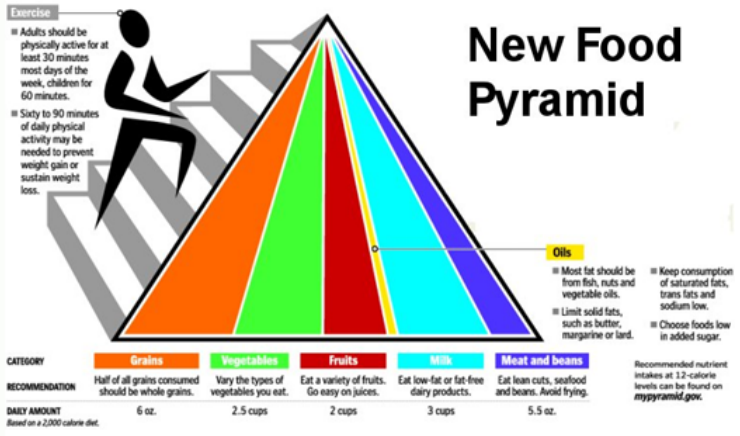
- The quantity, variety and combination of different foods and beverages in a diet and the frequency with which they are consumed:
  - Cooking and preparation methods
  - Cultural and regional influences

- Well-known dietary patterns:
  - Mediterranean diet
  - DASH diet
  - Plant-based/vegan/vegetarian diets
  - Western diet

Food pyramids

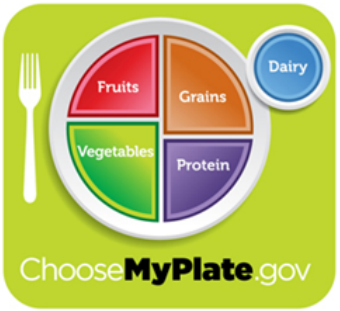
USA Food Guide Comparision Through the Years





- Fats, Oils, & Sweets have a surprisingly large visual portion compared to later food guides
- Specific food categories, highly descriptive

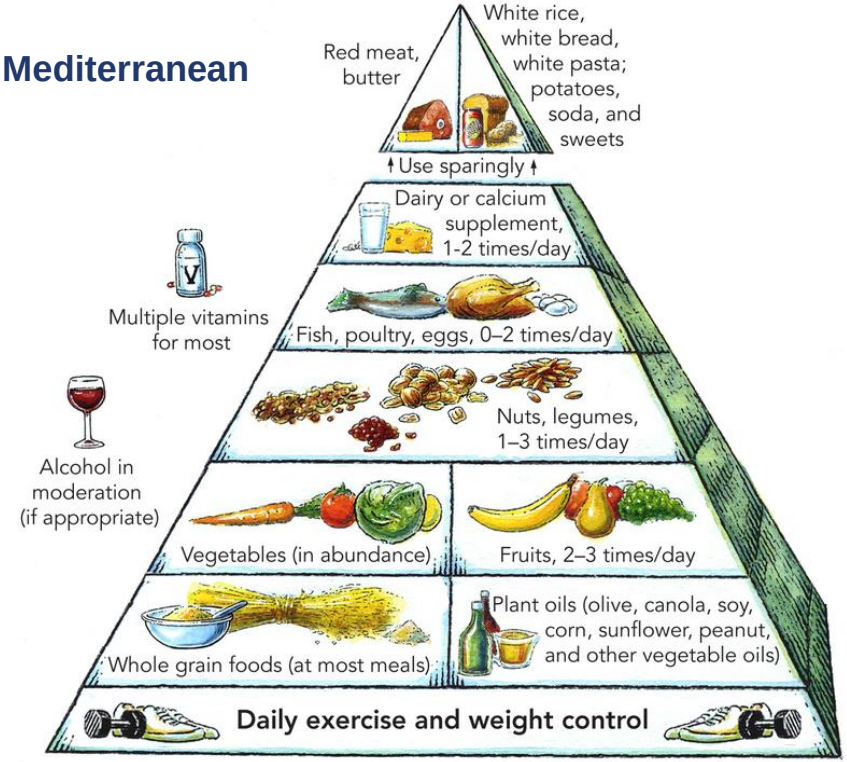
- Exercise Added to Food Guide
- Oils became smaller visual representation



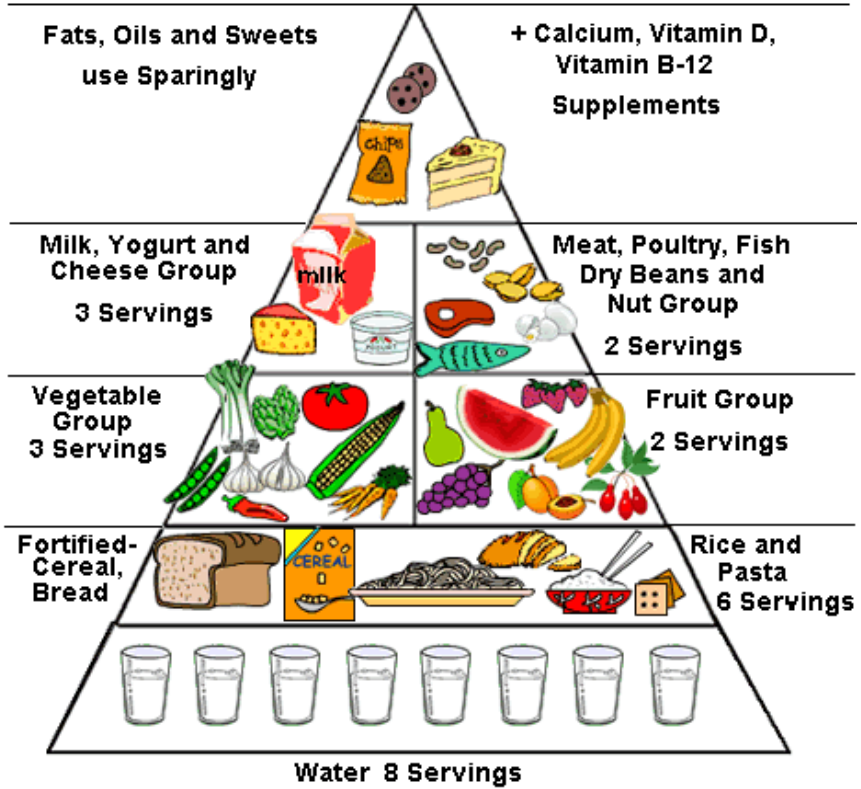
- Simplified Graphic Approach
- No Exercise in graphic
- Meat & Beans change to a more generic "protein" category
- Milk became "dairy"
- Elimination of serving size in exchange for proportion compared to other items on plate

Food pyramids

Mediterranean

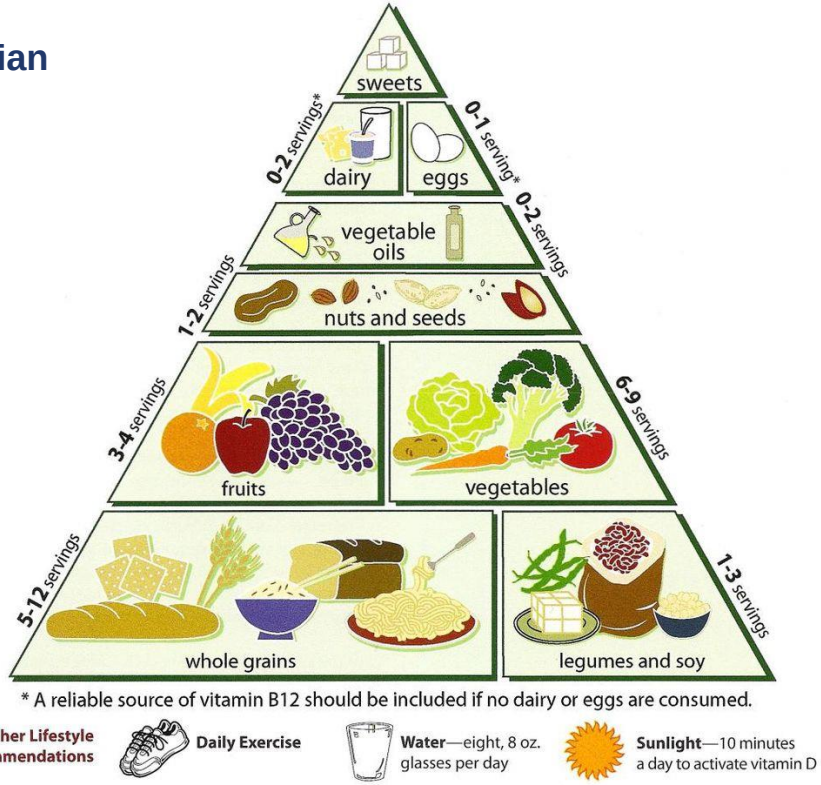


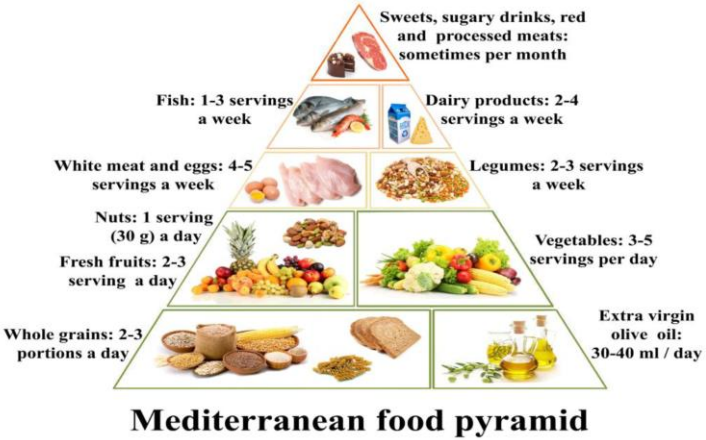
DASH



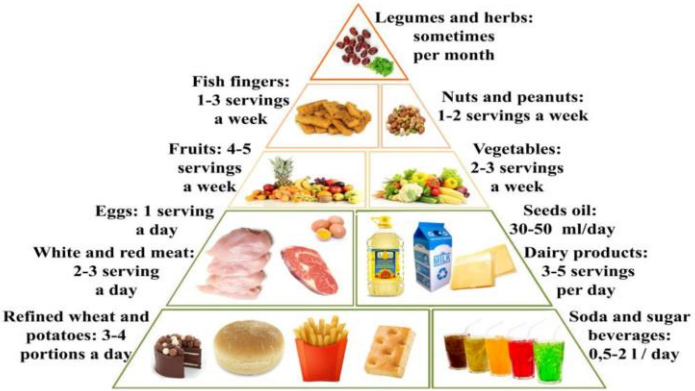
Food pyramids

Vegetarian





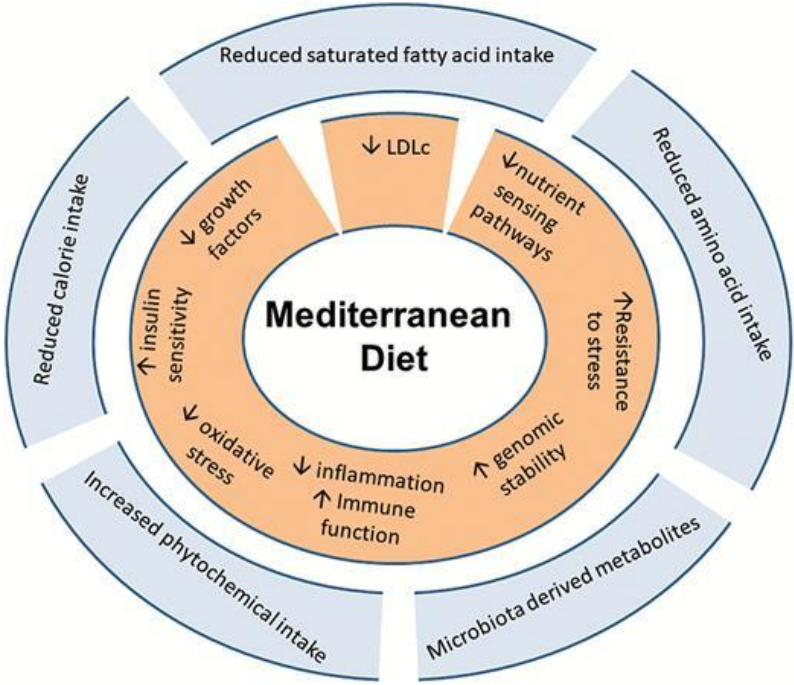
Mediterranean food pyramid



Western-style food pyramid

Mediterranean Diet and the risk for chronic disease

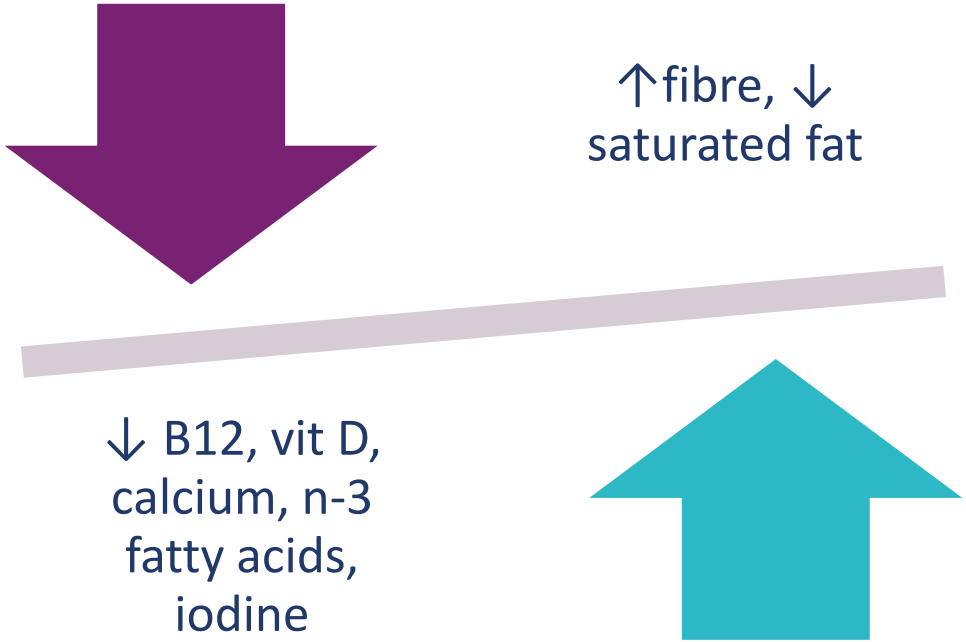
- Recent umbrella review meta-analysis:
  - ↑ adherence to the MedDiet -> ↓ incidence of T2D, ↓ incidence/mortality of CVD and cancer
  - An inverse dose–response association of whole grains with cancer mortality
  - A non-linear dose–response model that ↓ intake of total dairy products could be protective against cancer-related deaths
  - Processed meat has been particularly associated with an ↑ risk of colorectal and stomach non-cardia cancers
  - Fruits, nuts, whole grains are associated with ↓ risk of colorectal cancer



Galbete C *et al. Eur J Epidemiol,* 2018
https://doi.org/10.1007/s10654-018-0427-3

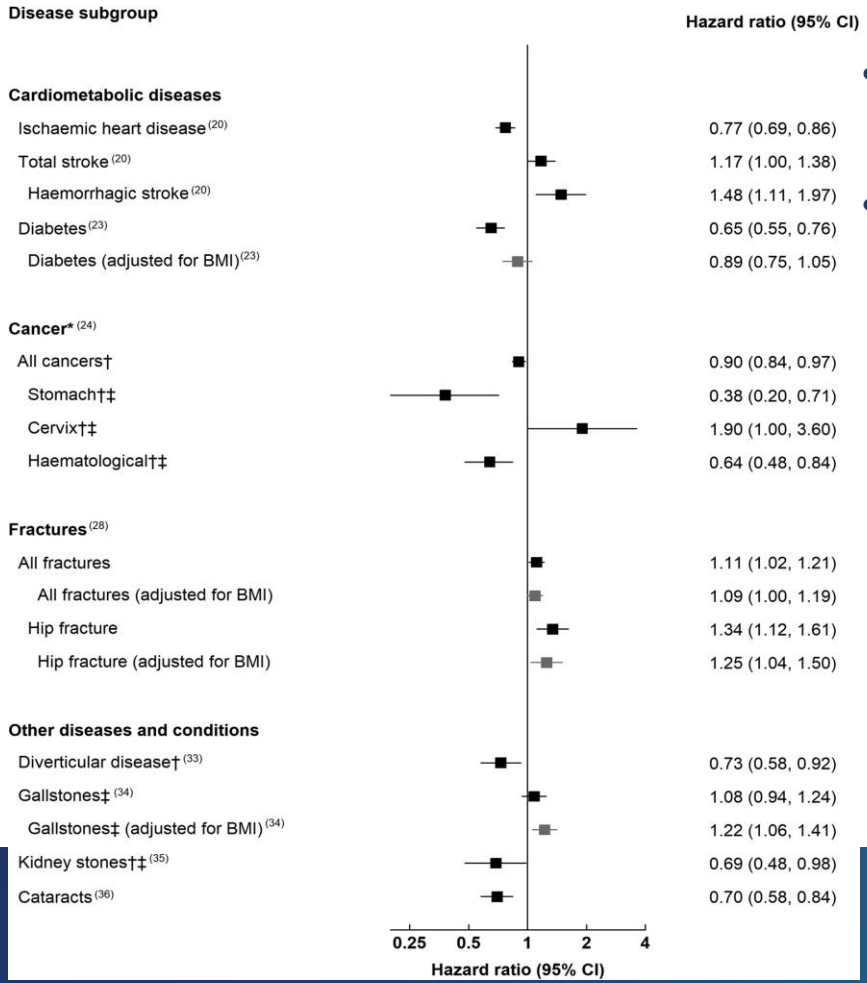
Vegetarian Diet and the risk for chronic disease

- Large-scale studies comparing vegan/vegetarian people vs meat-eaters
- Similar nutrient intakes; nutritionally adequate
- Vegan/vegetarians vs meat-eaters:
  - ↓ BMI
  - ↓ serum LDL cholesterol
  - ↓ blood pressure
  - ↓ bone mineral density



Key T *et al. Proceedings of the Nutrition Society, 2022*
https://doi.org/10.1017/S0029665121003748

Vegetarian Diet and the risk for chronic disease



- Large-scale studies comparing vegan/vegetarian people vs meat-eaters
- Vegetarians:
  - ↓ risk of IHD
  - ↓ risk diabetes
  - ↓ risk diverticular disease
  - ↓ risk kidney stones
  - ↓ risk cataracts
  - ↓ risk cancers
  - ↑ risk of stroke (principally haemorrhagic stroke)
  - ↑ risk bone fractures

Key T *et al. Proceedings of the Nutrition Society, 2022*
https://doi.org/10.1017/S0029665121003748

Vegetarian Diet – Environmental Sustainability

- Climate and environment benefits
- Assumption: the diet does not comprise large quantities of processed foods
- Current trend towards eating more highly processed plant-based convenience foods:
  - Public health concerns
  - Missing the targets of reducing GHG emissions
- Availability of plant-based convenience foods:
  - Shift towards eating a more plant-based diet
  - Willingness to eat less meat if it is attractive, convenient and accessible

- Ensure that dietary changes to reduce meat consumption will improve health and it does not become the vehicle for high fat, sugar and salt foods, which could also create more environmental damage

Macdiarmid J, *Proceedings of the Nutrition Society, 2022*
https://doi.org/10.1017/S0029665121003712

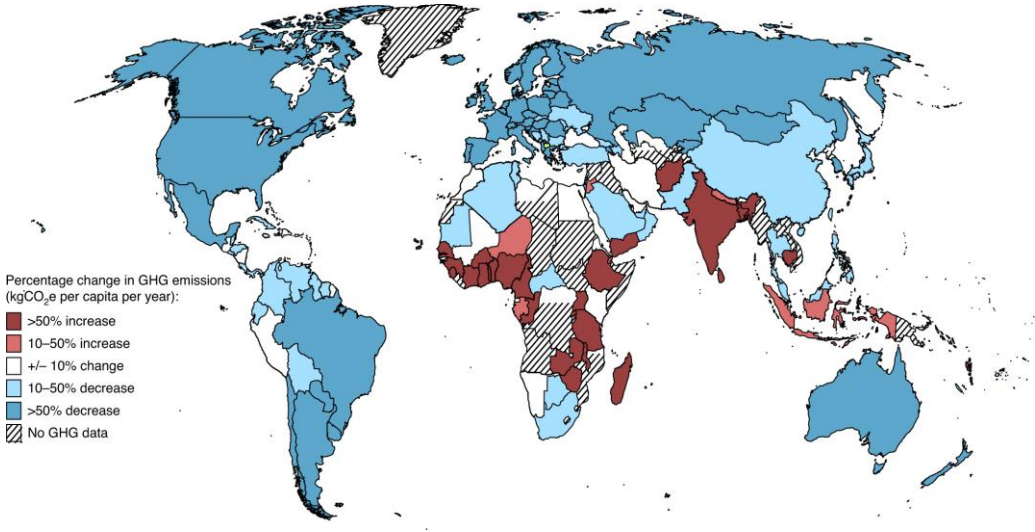
The planetary diet - Overview

- EAT–*Lancet* Commission -> planetary health diet: restricted intake of highly processed foods and animal source foods globally
- The planetary diet is designed to address the challenges of providing a nutritious and sustainable diet for the world's growing population while mitigating the environmental impacts of food production
- Recommended intake for adults: 2500 kcal per day
- While this amount will vary based on age, gender, activity levels and health profiles, overconsumption is a waste of food with both health and environmental costs

https://eatforum.org/eat-lancet-commission/the-planetary-health-diet-and-you/

The planetary diet – Greenhouse gas emissions

- Plant-based diets could reduce expected emissions by up to 80%
- The effect on country-specific GHG emissions low-income regions not well characterized
- Modelling study:
  - In 14 countries (0.3 billion) -> small change in GHG emissions
  - Reduce per capita GHG emissions for ~60% of the world's population
  - Increase per capita GHG emissions for the remaining ~40%

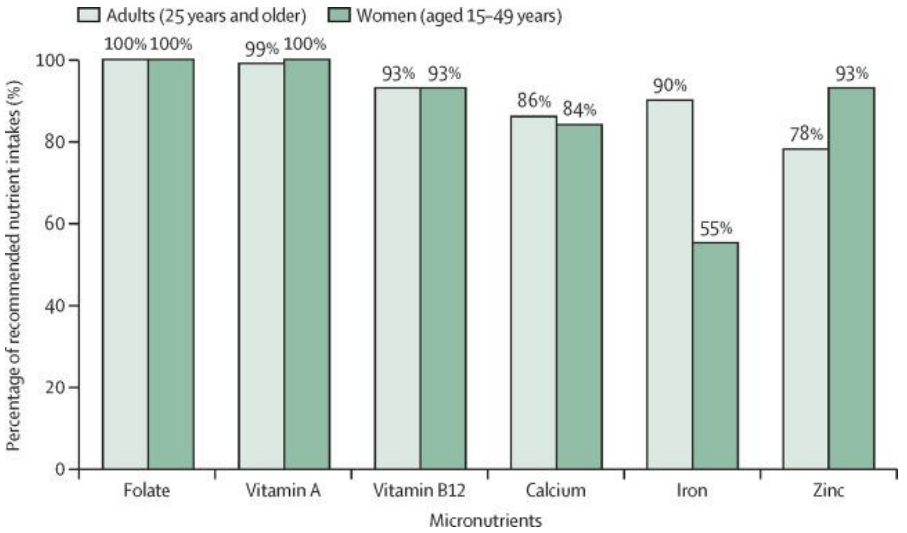


Replacement of an unhealthy diet with a high GHG footprint in high-income countries
Replacement of an insufficient diet limited in diversity and nutrients with a low GHG footprint in LMICs

Semba RD, *et al.* *Nat Food,* 2020
https://doi.org/10.1038/s43016-020-0128-4

The planetary diet – Nutritional content

- A planetary health diet consisting mostly of minimally processed, healthy plant source foods that is low in animal source foods
- Nutritionally adequate? -> insufficient intake of minerals such as iron (especially for women of reproductive age), calcium and zinc
- Adequate dietary nutrient intake:
  - Increased quantities of nutrient-dense foods such as fish, shellfish, seeds, eggs, and beef and reduced quantities of foods high in phytate such as whole grains, pulses and nuts -> # planetary diet
  - Need for fortified foods or use of supplements -> environmentally sustainable?

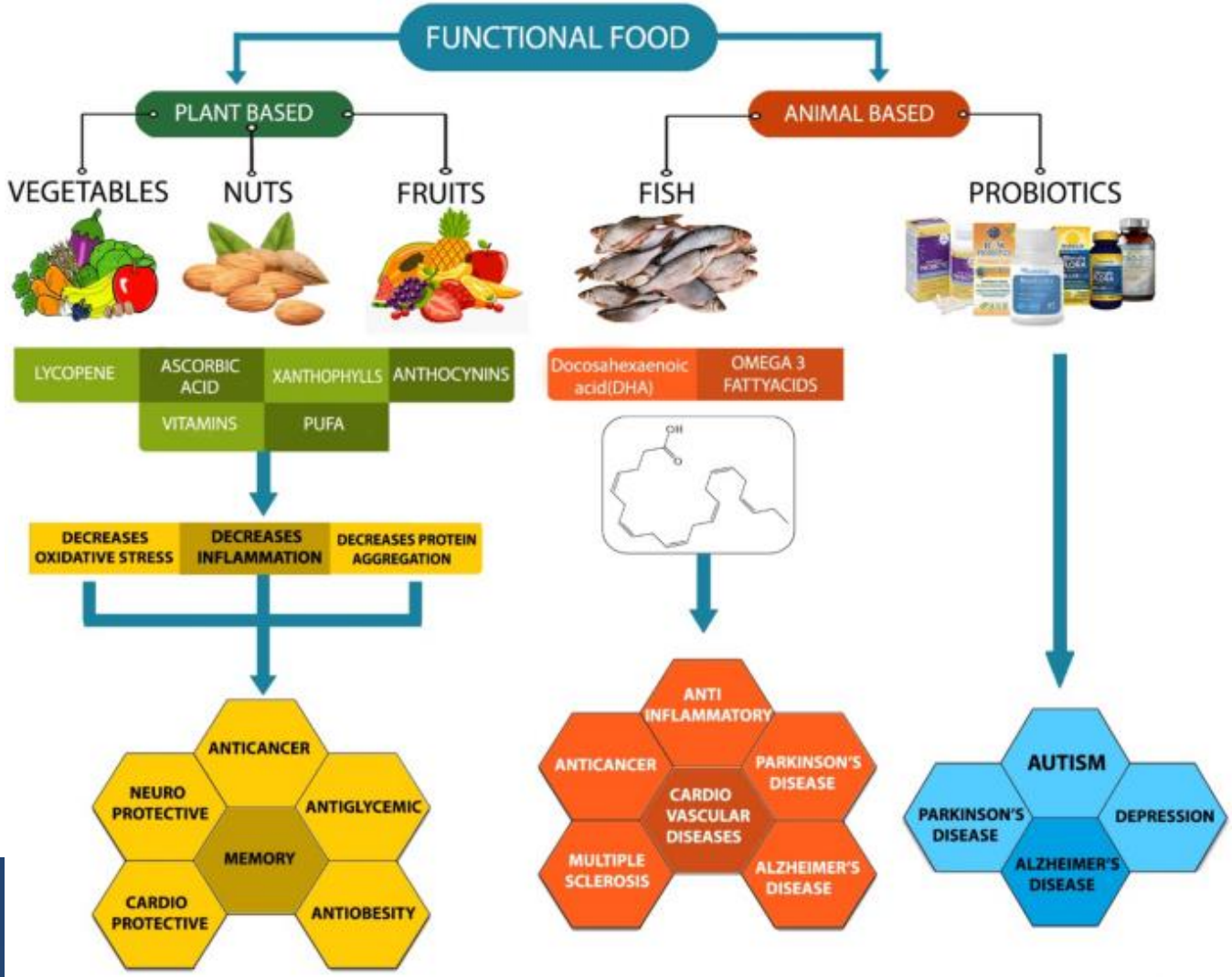


Beal T *et al. The LANCET Planetary Health, 2023*
https://doi.org/10.1016/S2542-5196(23)00006-2

Functional foods

- A common definition is that they contain substances “beyond basic nutrition” -> confusing as many foods could fall under this definition
- “Functional foods are novel foods that have been formulated so that they contain substances or live microorganisms that have a possible health-enhancing or disease-preventing value, and at a concentration that is both safe and sufficiently high to achieve the intended benefit. The added ingredients may include nutrients, dietary fiber, phytochemicals, other substances, or probiotics”
- Functional foods:
  - Conventionally used food
  - Fortified foods (i.e. orange juice with vit C, margarine with n-3 fatty acids)
  - Foods with added probiotics and/or prebiotics

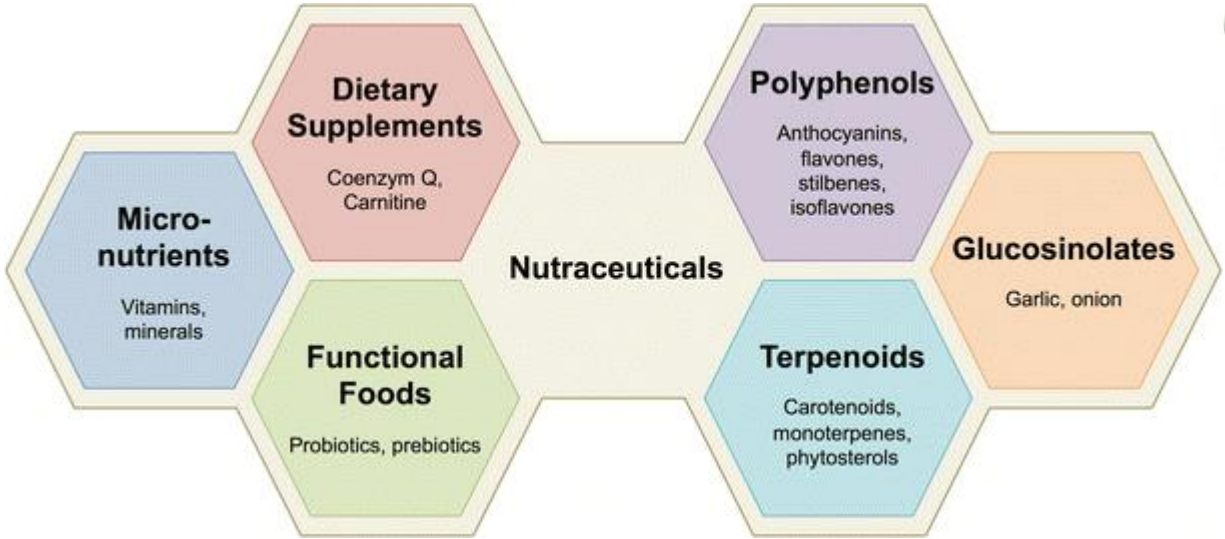
Functional foods



Essa MM *et al. J Food Sci Technol* 2023
https://doi.org/10.1007/s13197-021-05193-3

Nutraceuticals

- “A product isolated or purified from foods that is generally sold in medicinal forms not usually associated with food and is demonstrated to have a physiological benefit or provide protection against chronic disease”



Functional food vs Nutraceuticals

Functional foods

- Whole foods or food products that naturally contain bioactive compounds or have been fortified with specific nutrients or ingredients to provide additional health benefits
- Regulated as food products and may have specific labelling and marketing requirements
- Whole foods or food products that are consumed as part of the regular diet
- Less processed or minimally processed and retain their natural composition along with the added nutrients or bioactive compounds
- Marketed as food products that can be easily incorporated into the regular diet
- Regulated as food products and are subject to food safety regulations and guidelines

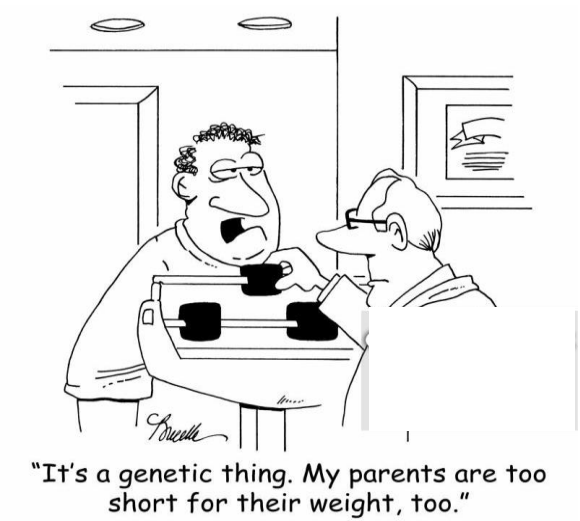
Nutraceuticals

- Isolated compounds or extracts, such as vitamins, minerals, or herbal extracts, that are used as dietary supplements or formulated into specific products
- Regulated and marketed as dietary supplements or medicinal products in many countries
- Capsules, tablets, powders, or liquids and are typically consumed as supplements alongside the regular diet
- Highly processed to isolate or extract specific compounds or ingredients
- Marketed and sold as standalone products, usually in the form of dietary supplements
- Subject to specific regulatory requirements and guidelines for safety, quality control, labelling, and marketing claims

Conclusions [1]

- Nutrition is an important factor of health and disease
- Dietary patterns low in saturated fat and processed food and high in fruits and vegetables, fibre and PUFA is protective against many disease
- Functional foods and nutraceuticals are special categories of food with enhanced health properties
- Vegan/vegetarian diets have health benefits, but caution must be paid over potential nutrient deficiencies
- The planetary diet is proposed as the ideal diet for both human health and environmental sustainability

Disease heritability



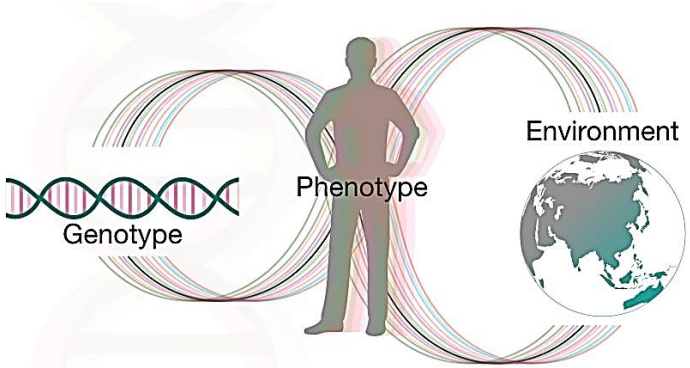
- Disease heritability is the proportion of the disease variability that is due to genetic differences

- A phenotype can be modelled as the sum of genetic and environmental effects

Phenotype (P) = Genotype (G) + Environment (E)

- All phenotypic traits (including diseases) are a product of gene-by-environment interactions to varying degrees

P = G + E + G x E



Tenesa et al. *Nat Rev Genet,* 2013
https://doi.org/10.1038/nrg3377

GWAS Catalog associations

~72K published genetic variant-trait associations with p-value ≤ 5.0×10^{-8}

http://www.ebi.ac.uk/gwas/

Complex traits and the missing heritability

- Large GWAS studies have identified numerous variants/loci associated with complex traits

- Yet explaining only a fraction of the traits' heritability:
  - BMI ~5% out of the 80% of heritability
  - Blood lipids ~15% out of the 50% of heritability
  - Coronary Artery Disease ~30% out of the 40-50% of heritability

Missing heritability: Lost and Found or Forever Lost?

- Gene-by-environment interactions:
  - Might explain a portion of the missing heritability
  - Studies would need very large sample sizes
  - Reliability issues (what is a representative set of environmental factors and how to assess this?)
- Microbiota interactions:
  - The microbiome is associated with many diseases and encodes a high number of genes
  - Human genotypes interact with the microbiome
  - Microbial genetic composition is heritable

How do we define gene-by-environment interactions?

- Interplay between genes and environmental factors
- Gene-by-nutrient interactions

- Interaction effects ≠ Joint effects

A different effect of an environmental exposure on disease risk in people with different genotypes

A different effect of a genotype on disease risk in people with different environmental exposures

Why investigate gene-by-environment interactions?

Disease risk prediction

- Obtain a better estimate of the genetic and environmental risk by accounting for their joint interactions

Environmental impact

- Determine environmental risk factors for disease
- Examine environmental risk factors in genetically susceptible individuals

Dissect disease mechanisms

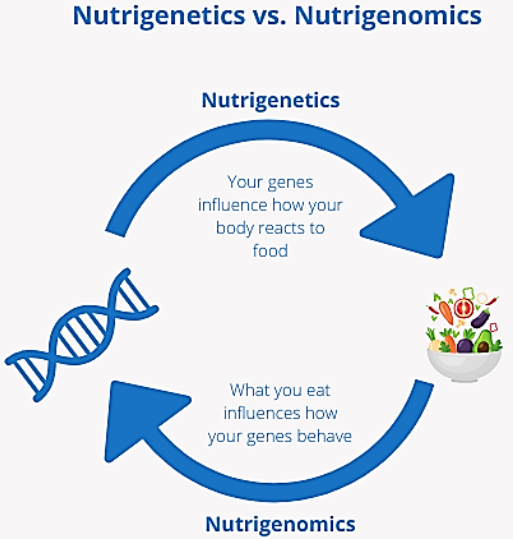
- Identify the biological pathways most relevant to disease
- Identify the environmental factors most relevant to the pathways

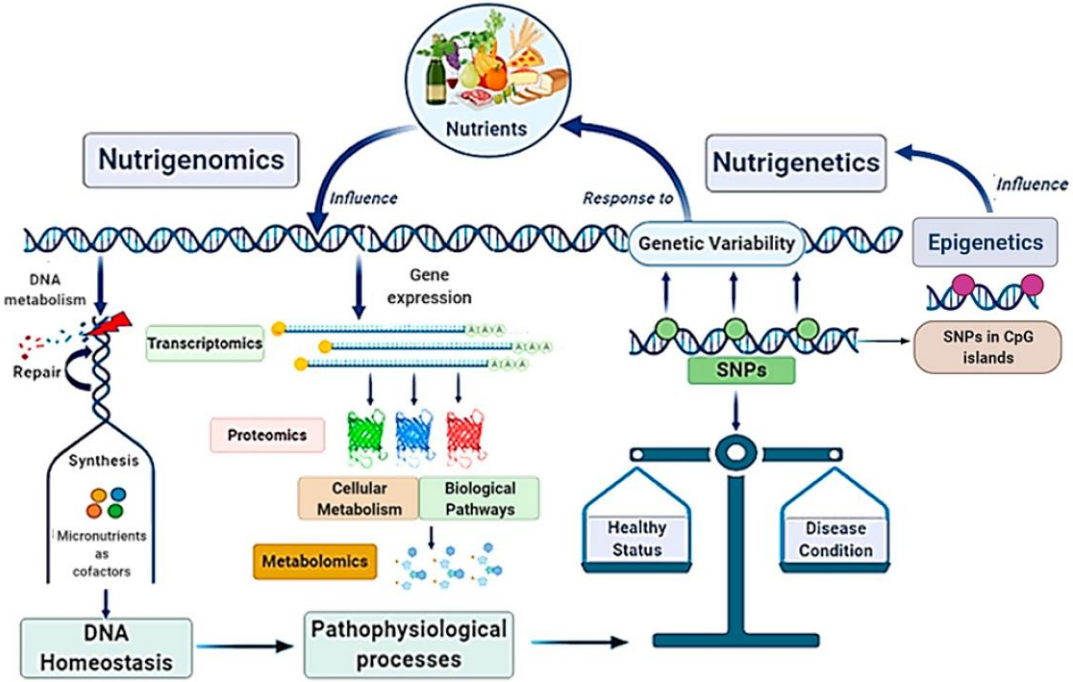
Novel preventive and therapeutic strategies

- Tailored preventive advice & pharmaceutical treatment based on genomic stratification of patients and disease phenotype

Nutrigenetics/Nutrigenomics

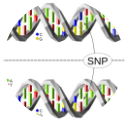
- The science that explores the specific interactions between genes and nutrients and relating this variation to human health and to variable disease states





Dallio et al. *Nutrients*, 2021
https://doi.org/10.3390/nu13051679

Analytical approaches



Single Genetic Variant

Genetic Risk Score

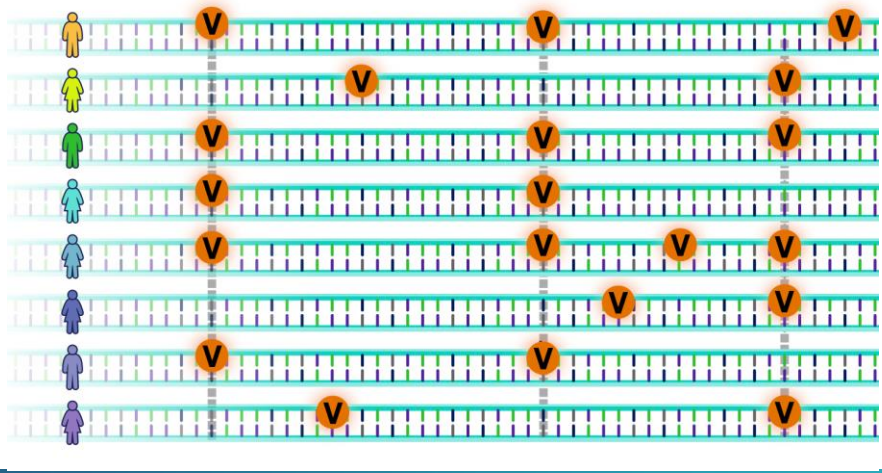
Single nutrients of food items

Food groups or food patterns

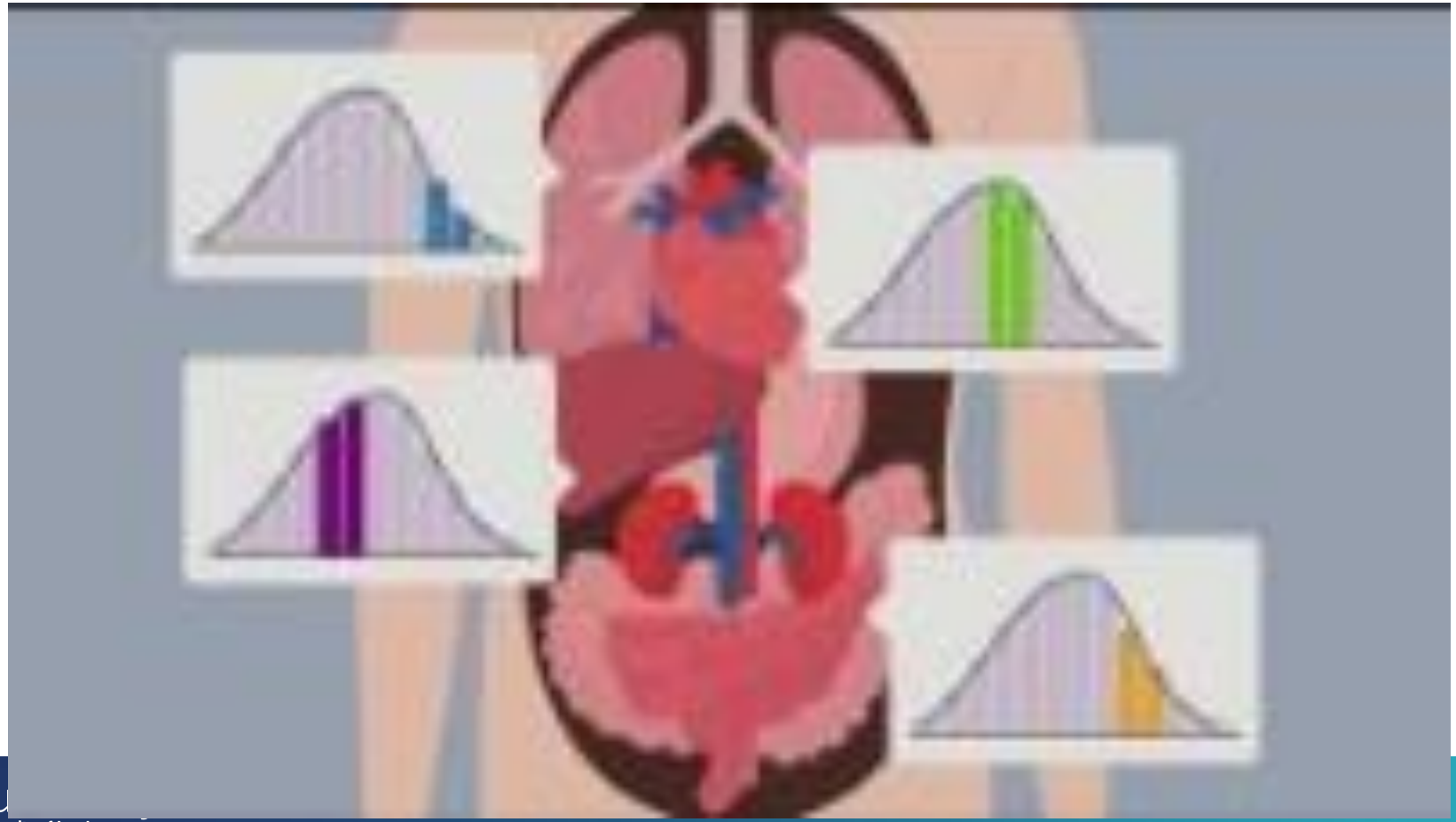
Lifestyle

Health lifestyle score

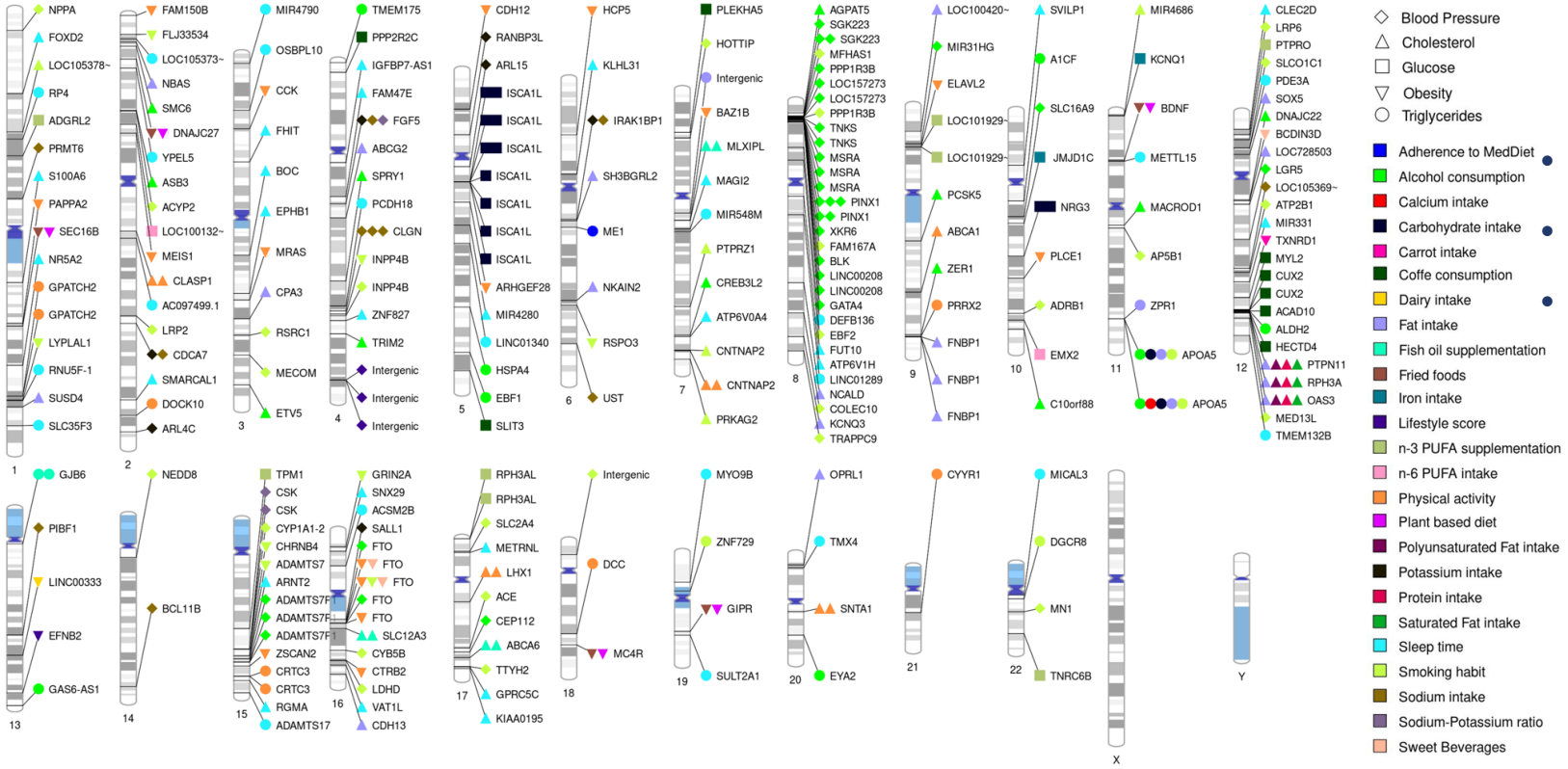
- Genetic risk score:
  - an estimate of an individual's genetic risk for some trait
  - the weighted sum of the risk increasing alleles (0, 1, or 2) per SNP for each individual



Polygenic Risk Scores

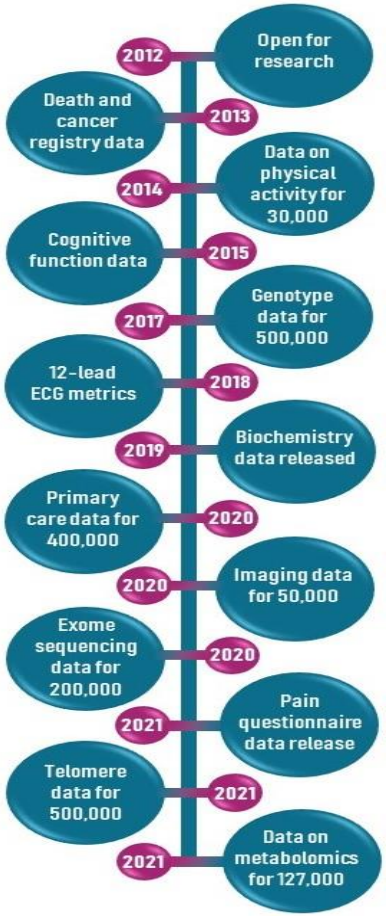


Gene-by-Environment Association Studies



- 42 studies
- 300 genes
- Cardiometabolic disease

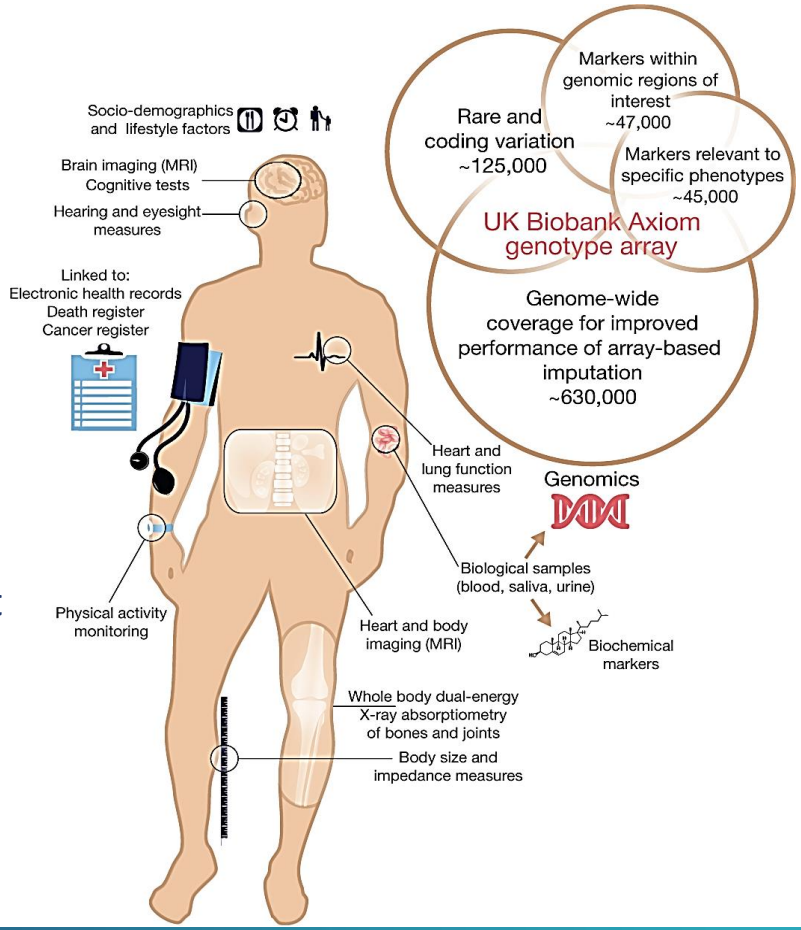
San-Cristobal et al. *Curr Nutr Rep*, 2022
https://doi.org/10.1007/s13668-022-00430-3



biobank uk

Improving the health of future generations

- Recruitment of 500,000 people aged 40-69 from 2006-2010 from UK
- The participants consented to:
  - Participate in the measurements
  - Supply a sample of saliva, blood and urine
  - Give detailed information about themselves
  - Allow access to their EHR



Extended Data Table 1 | Types and dates of data collection in UK Biobank

	Type of data	Date of data collection	Number of participants
			Anticipated
Questionnaire and interview	Sociodemographic data	Recruitment: 2006-2010[a]	500,000
	Family history and early life		500,000
	Psychosocial factors		500,000
	Lifestyle		500,000
	Medical history		500,000
	Cognitive function		500,000
Physical measures	Blood pressure	Recruitment: 2006-2010	500,000
	Hand grip strength		500,000
	Anthropometry		500,000
	Spirometry		500,000
	Heel bone density		500,000
	Arterial stiffness		200,000
	Hearing test		200,000
	Cardiorespiratory fitness plus ECG		100,000
	Eye measures		100,000
Web-based questionnaires	Diet	2011-2012	210,000[b]
	Cognitive function	2014	120,000
	Occupational history	2015	120,000
	Mental health	2016	150,000
	Irritable bowel syndrome	2017	150,000
Enhancements	Physical activity monitor	2013-2014[c]	100,000
	Biochemistry markers[d]	2006-2010	500,000
	Genotyping	2006-2010	500,000
	Multi-modal imaging[e]	2014-2022	100,000[f]
Electronic medical records	Death registry	2006-current	14,000
	Cancer registry	1971-current	79,000
	Hospital inpatient data	1996-current	400,000
	Primary care data	Birth-current	pending

biobank uk
Improving the health of future generations

Queen Mary University of London
Medicine and Dentistry

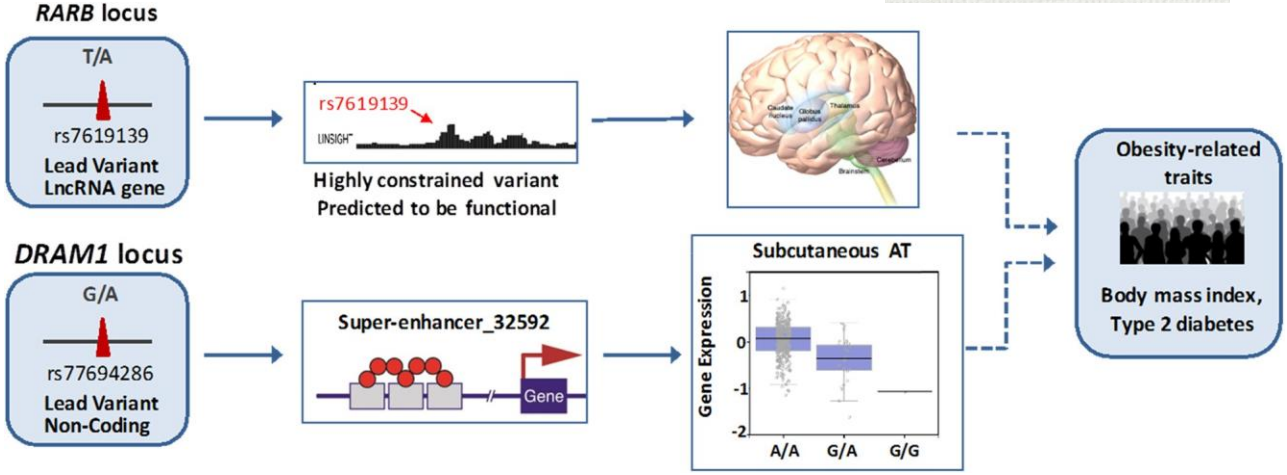
https://tinyurl.com/5n7nas3u

Bycroft et al. *Nature*. 2018
https://doi.org/10.1038/s41586-018-0579-z

Nutrigenetics in Obesity

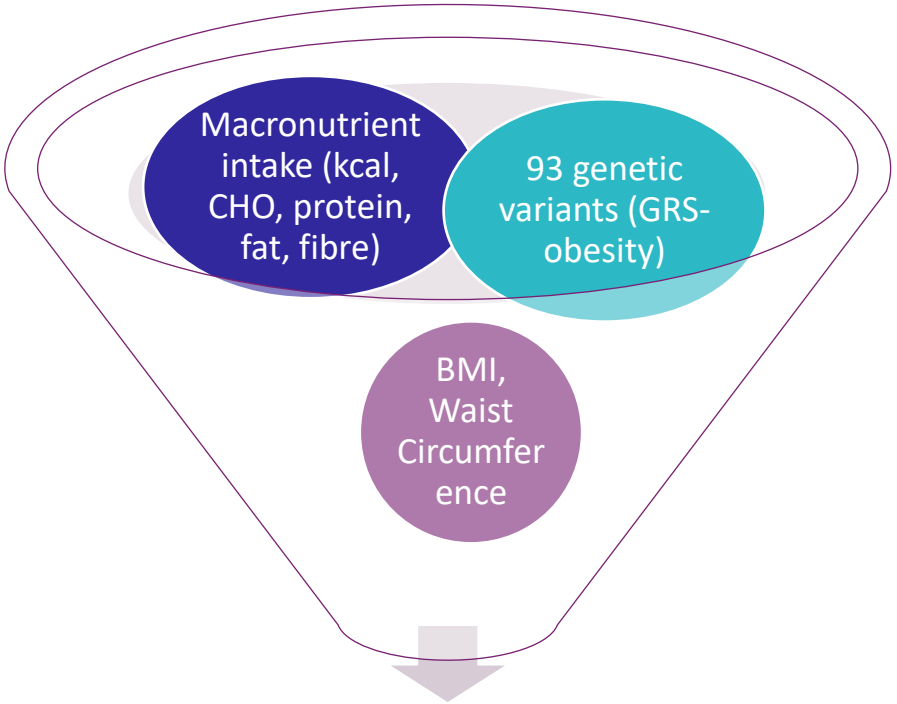
- ~941 genetic loci associated with BMI:
  - genes expressed in the hypothalamus (CNS)
  - genes involved in feeding and fasting pathways

- Obesogenic genes found to be associated with dietary intake:
  - *RARB* -> ↑ CHO intake
  - *DRAM1* -> ↑ protein intake
  - *FGF21* -> ↑ CHO & ↓ fat & protein intake
  - *FTO* -> ↑ protein intake



Locke et al. *Nature*, 2015 https://doi.org/10.1038/nature14177 Merino et al. *Mol Psychiatry*, 2019 https://doi.org/10.1038/s41380-018-0079-4

Nutrigenetics in Obesity – GRS x Macronutrients



Interactions in ~50K adults

- People with the highest GRS score (high genetic risk for obesity) and high consumption of SFA had 1.8kg/m^2 higher BMI and 3.7cm higher WC compared to those with low SFA consumption but same GRS
- Similar results for total fat and total energy intake
- **Identifying people susceptible to obesity -> offering customised dietary advice -> reduce the risk for obesity**

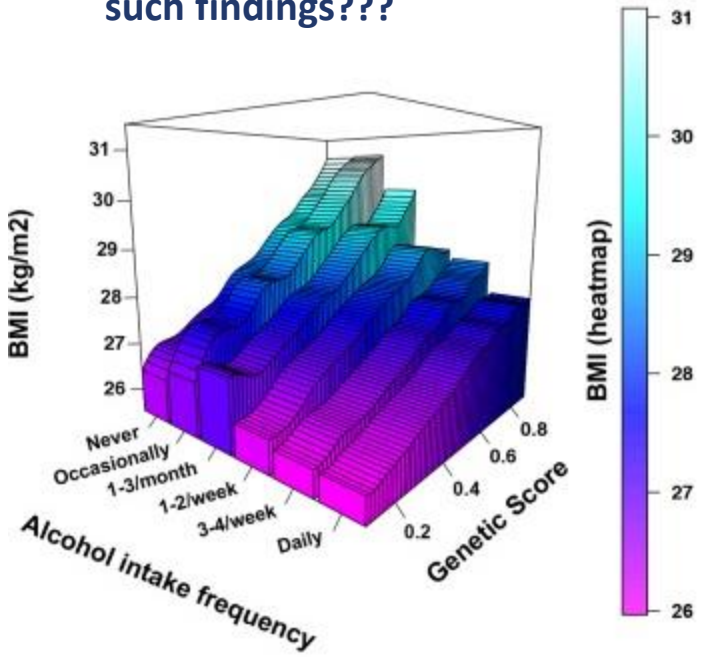
Celis-Morales et al. *Int J Obes*, 2017
https://doi.org/10.1093/ije/dyw186

Nutrigenetics in Obesity – Gene-by-lifestyle interactions

- 362K Europeans from UK Biobank
- Genetic risk score for obesity -> 94 genetic markers associated with BMI
- 131 lifestyle factors-> diet, smoking, alcohol, physical activity, socioeconomic status, mental health, sleeping patterns, menopause, childbirth

- **15 lifestyle factors had significant interaction with the genetic risk score -> alcohol intake frequency, walking pace, Townsend deprivation index**

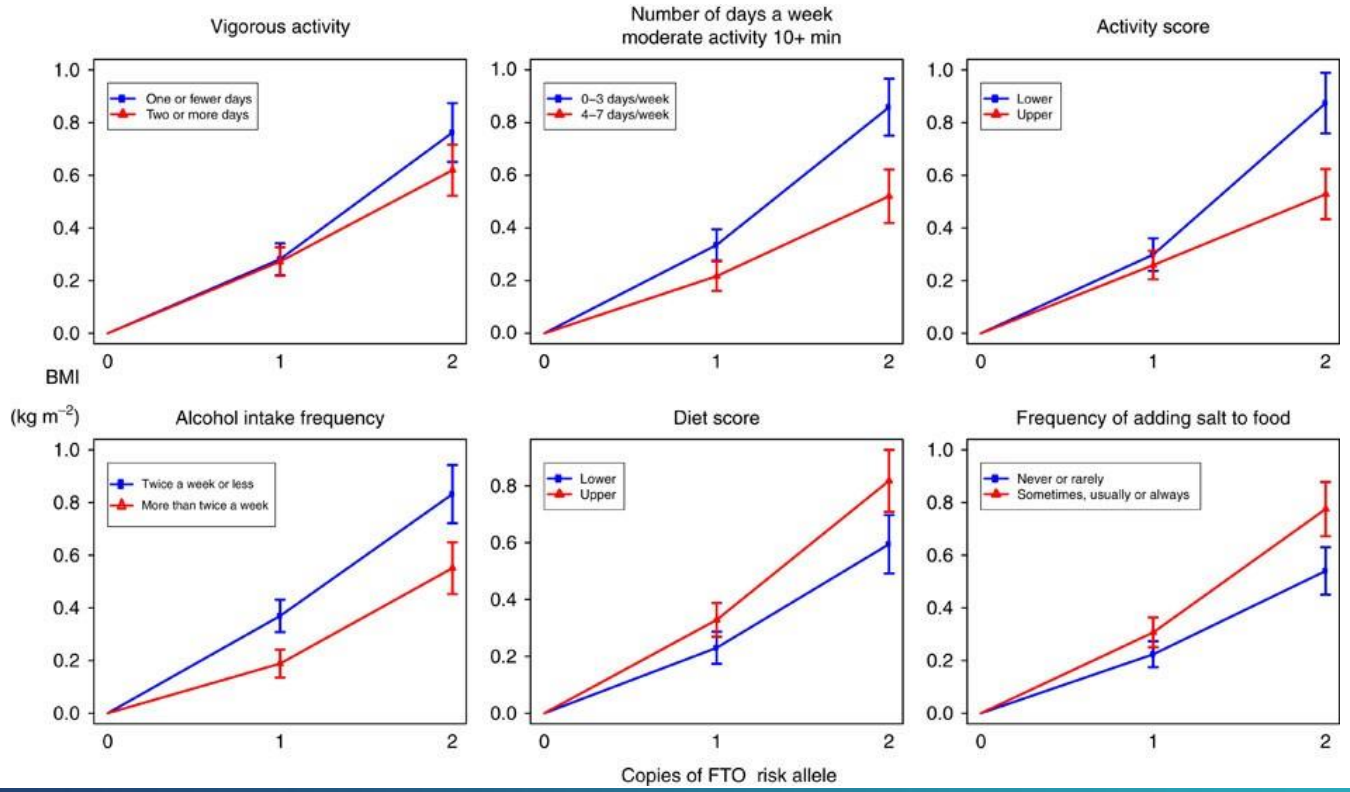
How do we interpret such findings???



Rask-Andersen et al. *PLoS Genet*, 2017
https://doi.org/10.1371/journal.pgen.1006977

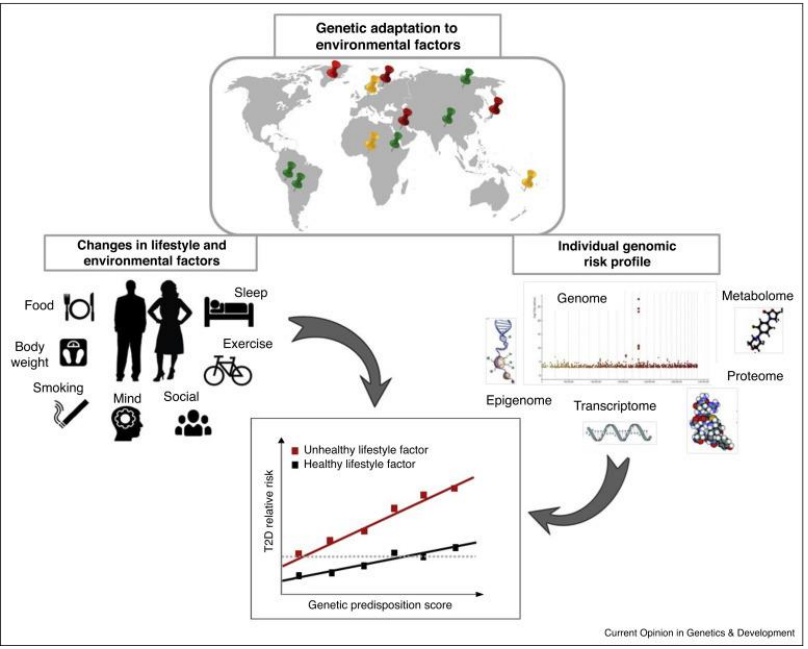
Nutrigenetics in Obesity – *FTO*

- First identified obesity gene
- 200K UK Biobank participants
- *FTO* variant rs1421085
- 1.17% BMI increase per risk allele



Young AI et al. *Nat Commun*, 2016
https://doi.org/10.1038/ncomms12724

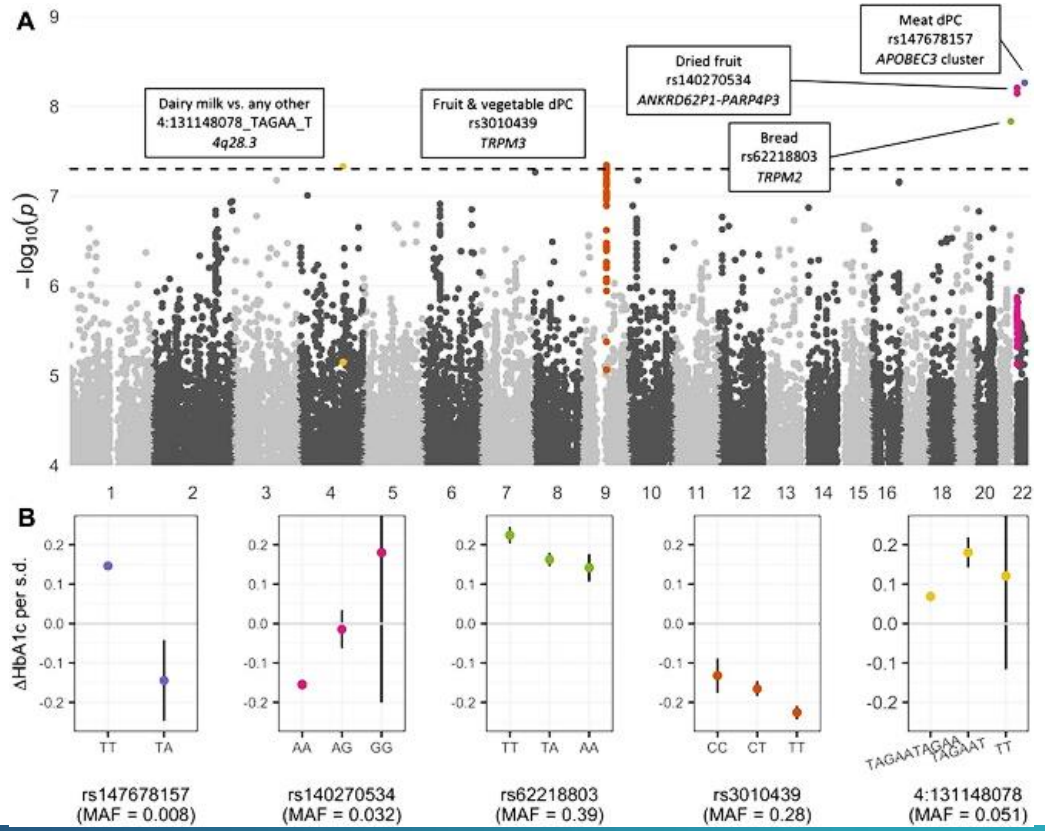
Nutrigenetics in T2D



T2D locus	Lifestyle	Interaction effect	Study
PPARG	Dietary fat	Insulin levels	Isle of Ely Study; Nurses' Health Study
HNF1B	Physical activity	T2D incidence	16K Swedish individuals during 25y
GIPR	Dietary fat Dietary CHO	T2D incidence	Malmo Diet and Cancer Cohort
TCF7L2	Dietary fibre	T2D incidence	Malmo Diet and Cancer Cohort
GCKR SLC30A8	Dietary whole grains Dietary zinc	Insulin levels	CHARGE
FBN1	Smoking	T2D incidence	Meta-analysis (Africans)
C2orf63	Smoking	T2D incidence	Meta-analysis (Europeans)

Nutrigenetics in T2D – HbA1c

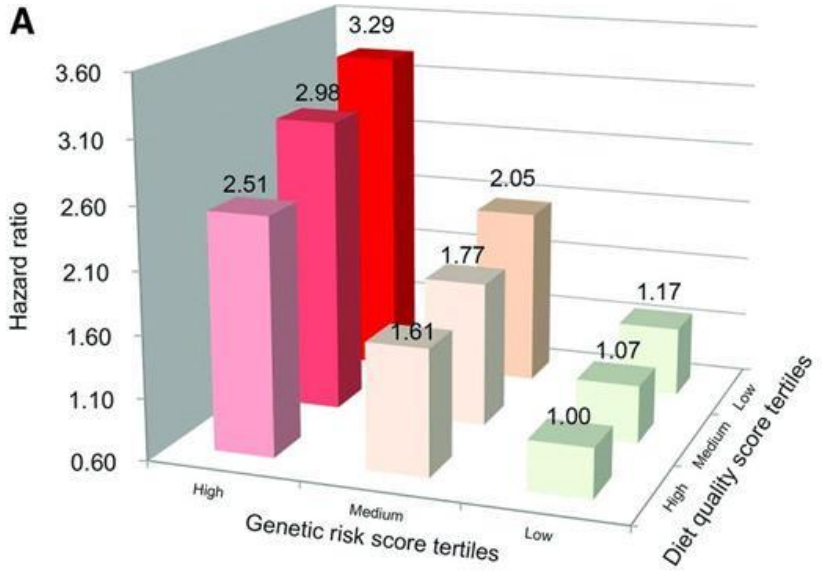
- Gene x Diet Interaction in 340K UK Biobank participants without T2D
- HbA1c prognostic & diagnostic marker for T2D
- TRPM2 & TRPM3 known to affect insulin secretion by beta-cells
- Effect sizes of interaction meaningful for personalized nutrition interventions



Westerman KE et al. *Hum Mol Genet*, 2021
https://doi.org/10.1093/hmg/ddab109

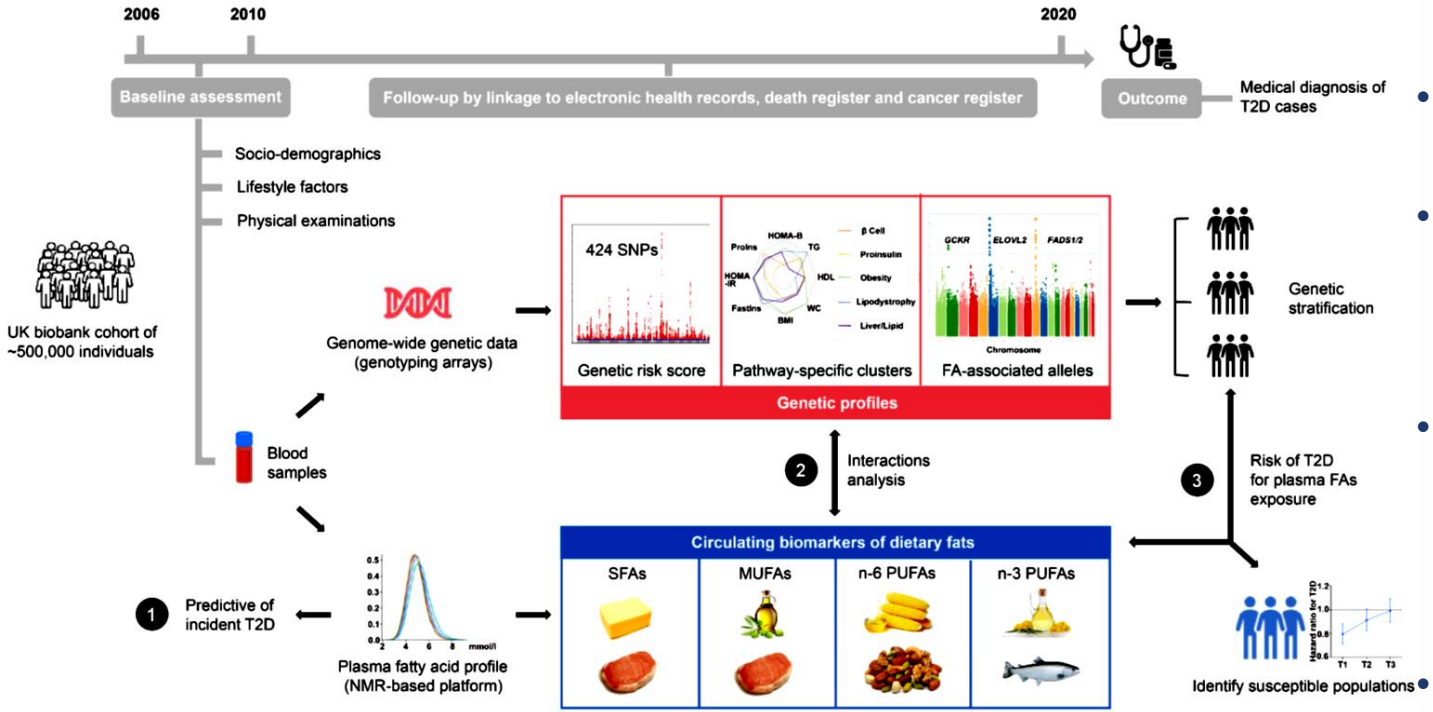
Nutrigenetics in T2D – HbA1c

- 350K UK Biobank participants, incl. 5,633 with T2D
- T2D GRS (424 genetic variants)
- Healthy diet score
- The GRS was associated with a 54% higher risk of T2D
- The diet quality score was associated with a 9% lower risk of T2D
- A 1-SD increment in the diet quality score in the high GRS group (GRS >95%) was associated with a ↓ 23% in T2D risk
- A strong negative interaction between the GRS and the diet quality score on the blood HbA1c level



Zhuang P et al. *Diabetes Care*, 2021
https://doi.org/10.2337/dc21-1051

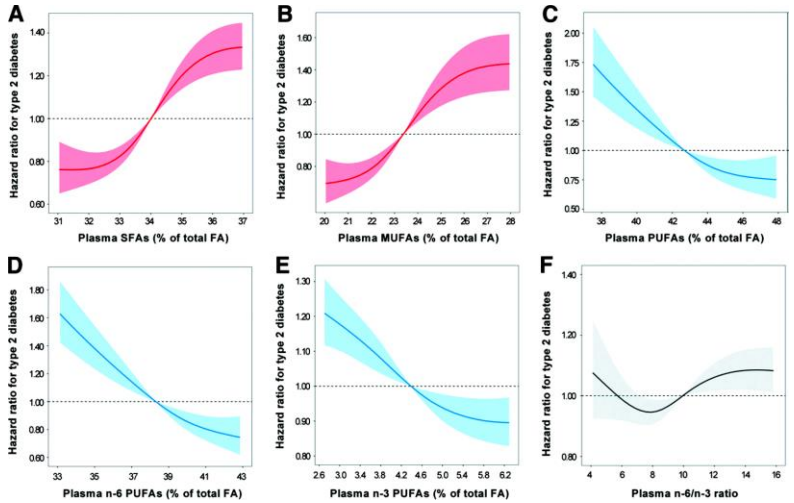
Nutrigenetics in T2D – Fatty acids

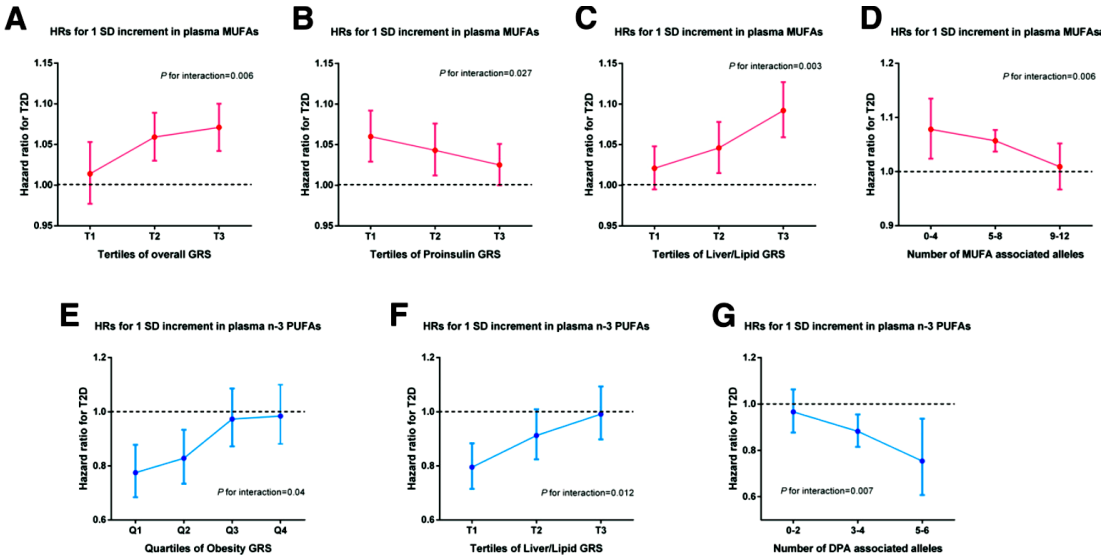


- NMR metabolites -> fatty acids
- Risk of T2D incidence ~ Plasma fatty acid levels (95,854 participants)
- Risk of T2D incidence ~ Genetic risk score x Plasma fatty acid levels (89,955 participants)
- 3,052 incident T2D cases in 11.6 years

Zhuang P et al. *Diabetes Care*, 2022
https://doi.org/10.2337/dc21-2048

Nutrigenetics in T2D – Fatty acids



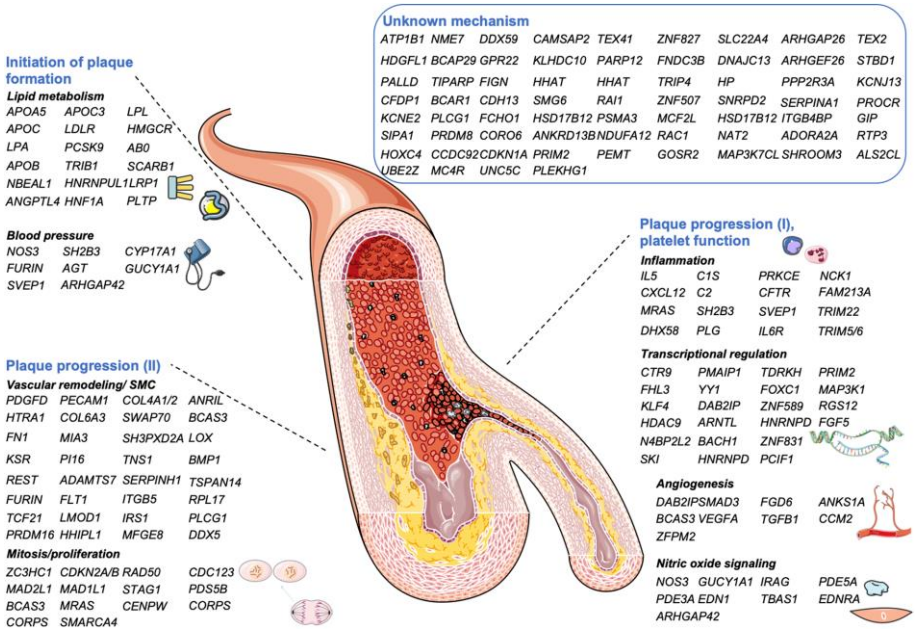


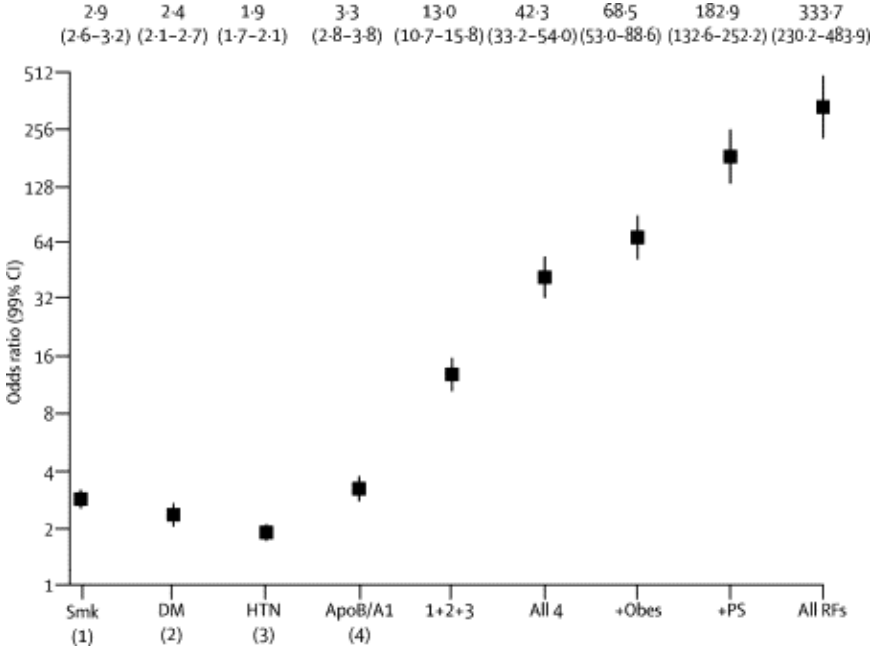
biobank uk
Improving the health of future generations

Zhuang P et al. *Diabetes Care*, 2022
https://doi.org/10.2337/dc21-2048

Nutrigenetics in CVD

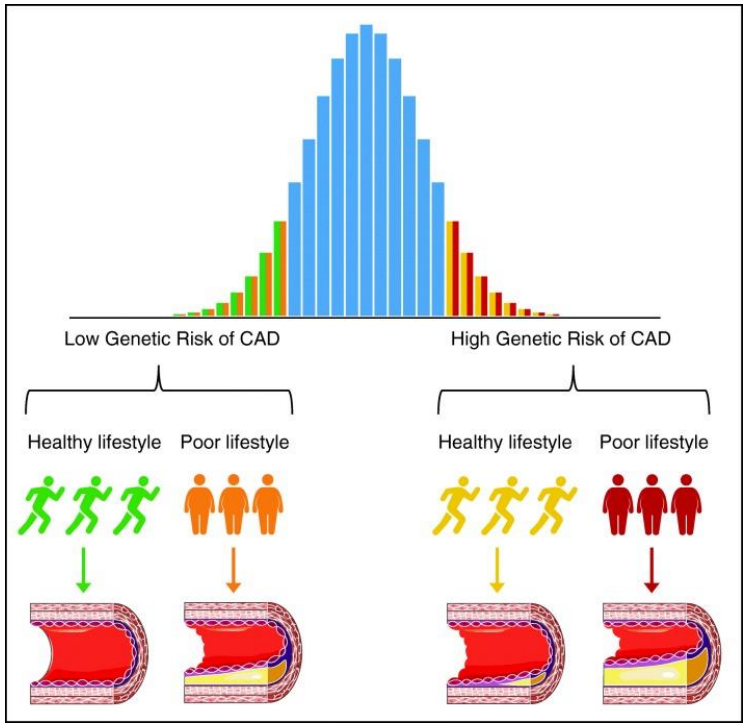
- INTERHEART study in 52 countries





Yusuf S et al. *Lancet*, 2004
https://doi.org/10.1016/S0140-6736(04)17018-9

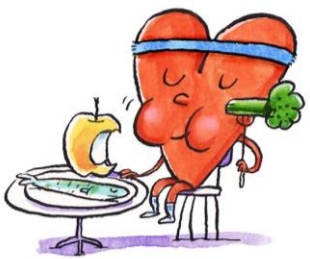
Nutrigenetics in CVD – GRS & Lifestyle



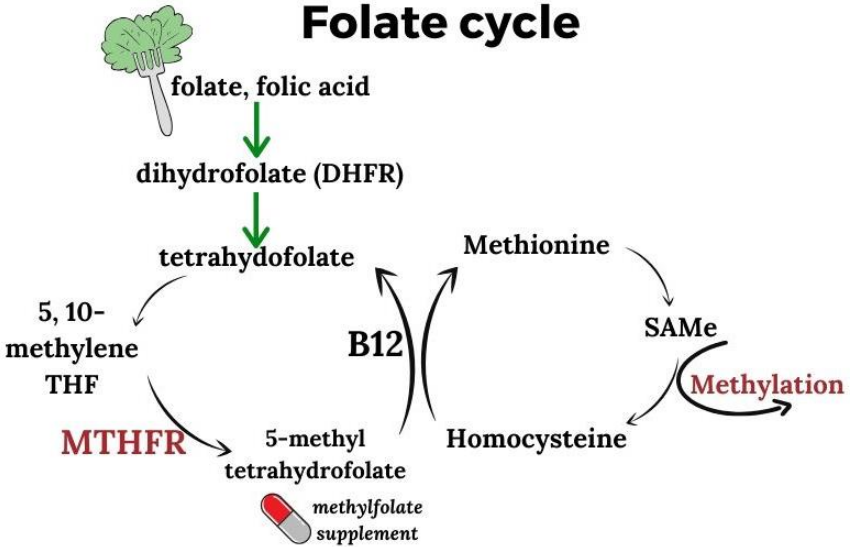
- 4 studies ~55K participants
- CAD GRS (50 genetic variants)
- Healthy lifestyle: smoking, obesity, physical activity, healthy diet patterns
- Healthy lifestyle was associated with reduced CAD risk across all levels of genetic risk
- **Among participants at high genetic risk, a healthy lifestyle was associated with a 46% lower relative risk of coronary events than an unhealthy lifestyle**
- Similar results in another study in UK Biobank

Khera AV et al. *New Engl J Med*, 2016
https://doi.org/10.1056/NEJMoa1605086

Nutrigenetics in CVD – *MTHFR* x Folate

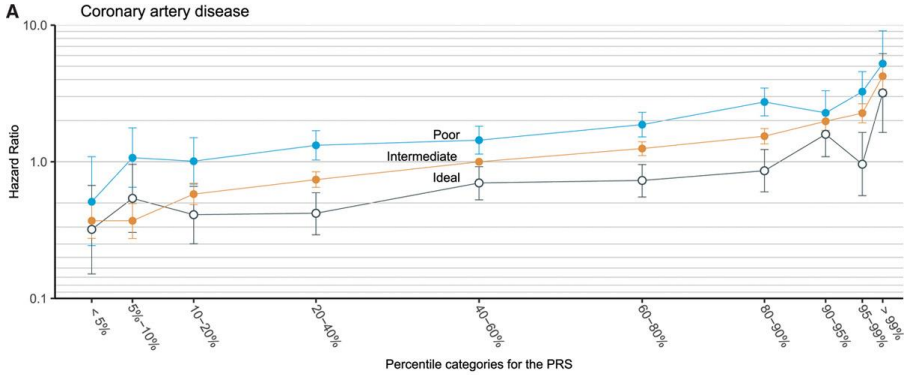


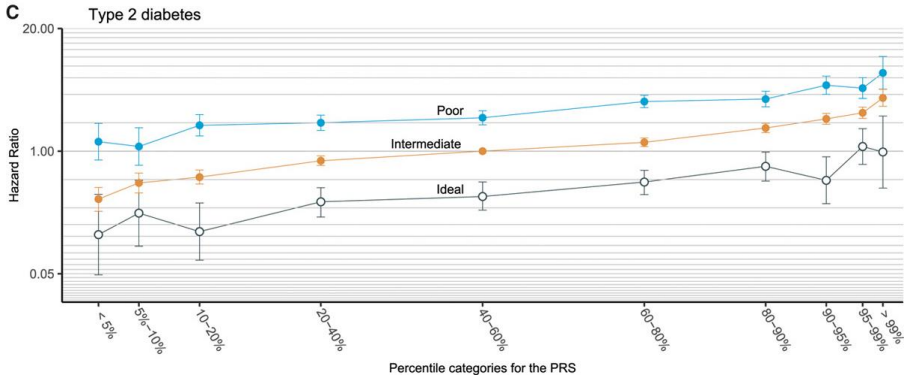
- Homocysteine levels (tHcy) are a CVD risk factor -> inflammation pathways
- tHcy is modulated by folate intake and alcohol consumption
- C677T polymorphism in *MTHFR* is associated with ↑ tHcy & ↓ folate levels
- TT genotype is also associated with ↓ folate supplementation response



Colson NJ et al. *Eur J Nutr*, 2017
https://doi.org/10.1007/s00394-015-1076-x

Nutrigenetics in CVD – Genetic risk score-by-Lifestyle risk score



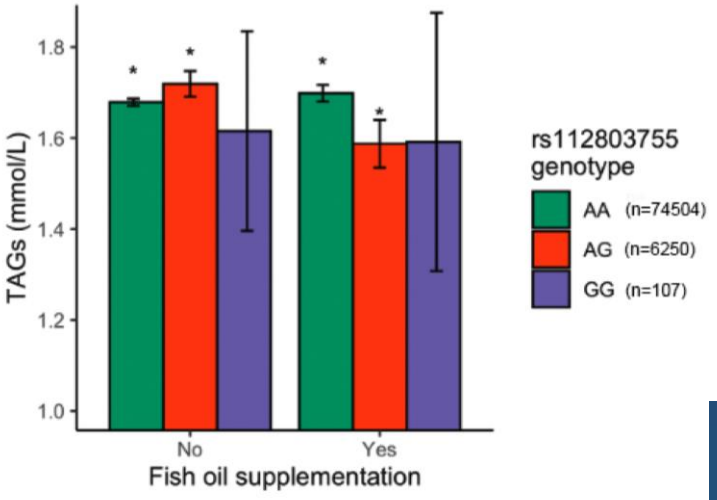


- 276,096 European participants from UK Biobank
- Compared with the group of individuals with mid-range genetic risk (40%–60% PRS) and intermediate combined lifestyle:
  - The group of individuals having the top 1% PRS while leading poor lifestyles increased to 5.23 for CAD and 6.67 (95%, 4.55–10.1) for T2D
  - For individuals with similarly high genetic risk (top 1% PRS) but healthy lifestyle, the corresponding risk decreased to 3.19 for CAD and 0.98 for T2D
- **Benefit of leading a healthy lifestyle even for this extremely high genetic risk group**

Ye Y et al. , *Circ Genom Precis Med*, 2021
https://doi.org/10.1161/CIRCGEN.120.003128

Nutrigenetics in CVD – Fish oil supplements

- 73,962 participants from UK Biobank and validation in 7.284 participants from the ARIC study
- Blood lipid levels ~ Genetic variants + Fish oil supplements + **Genetic variants x Fish oil supplement**
- **Significant interaction for rs112803755 and triglyceride levels -> gene complex *GJB6-GJB2-GJA3***



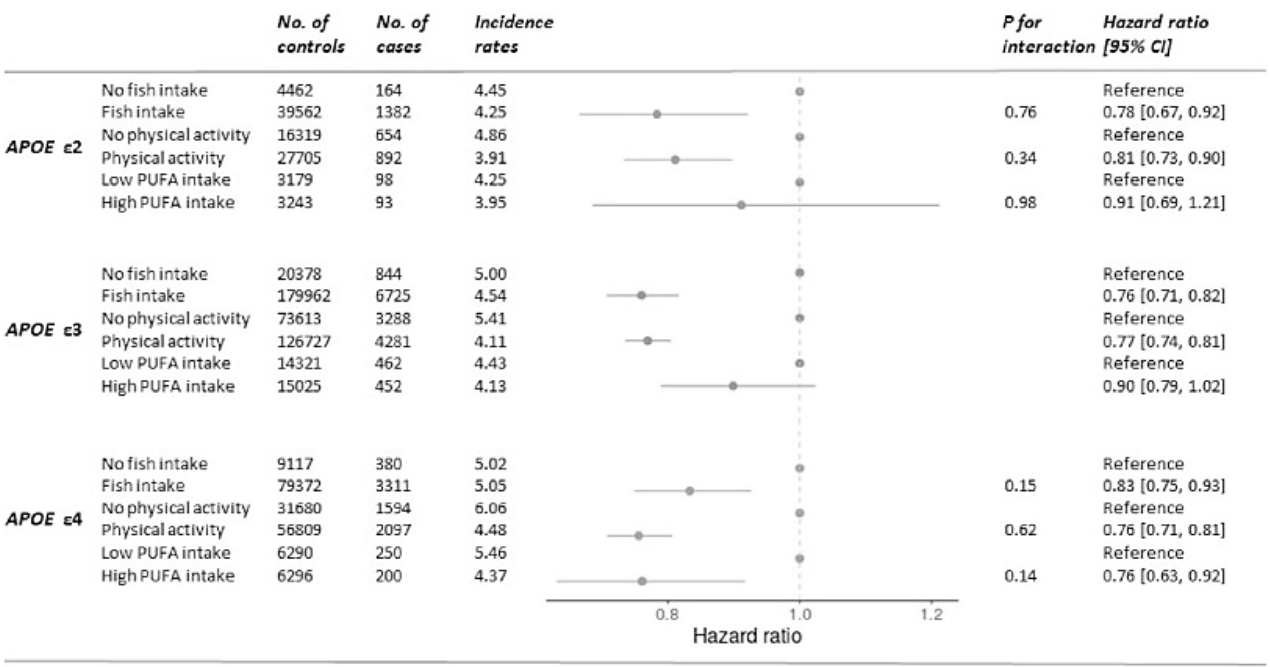
- The genetic region is associated with ↑expression of *GJB2* (connexin 26) in adipose tissue
- The gene's connexin function is modulated after exposure to n-3 fatty acids

Francis M et al. , *PLoS Genet,* 2021
https://doi.org/10.1371/journal.pgen.1010735

Nutrigenetics in CVD – *ApoE*

- 345,659 participants from UK Biobank with no history of coronary artery disease
- Interaction with the ApoE haplotypes with:
  - Physical activity
  - Oily fish consumption
  - PUFA

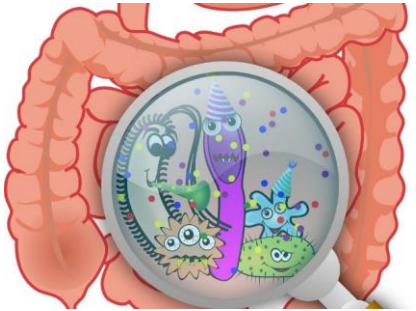
Risk for coronary artery disease (12,806 incident cases in 8.11 years)



Bos MM et al. , *Atherosclerosis,* 2021
https://doi.org/10.1016/j.atherosclerosis.2021.05.014

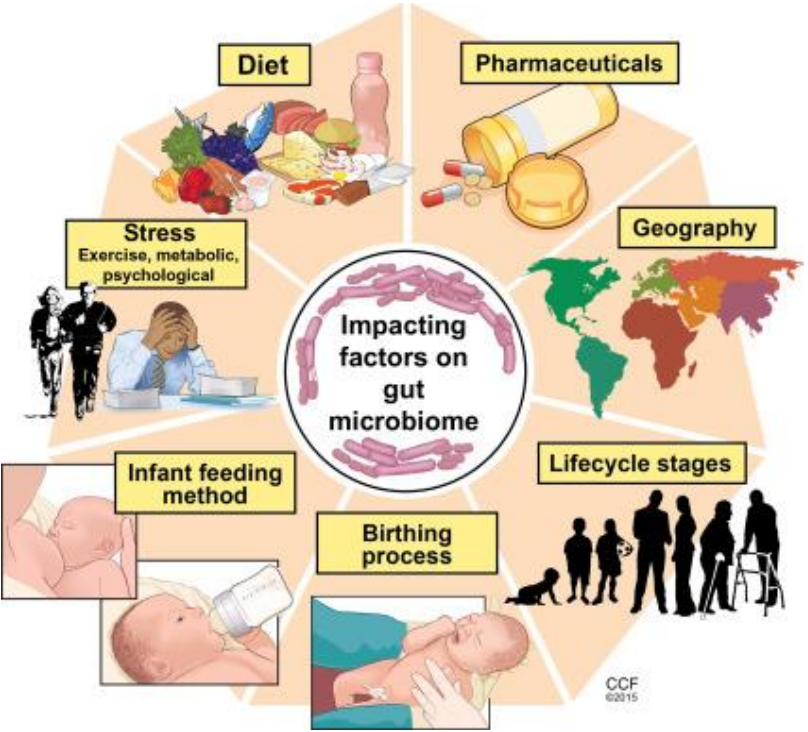
Human Microbiota

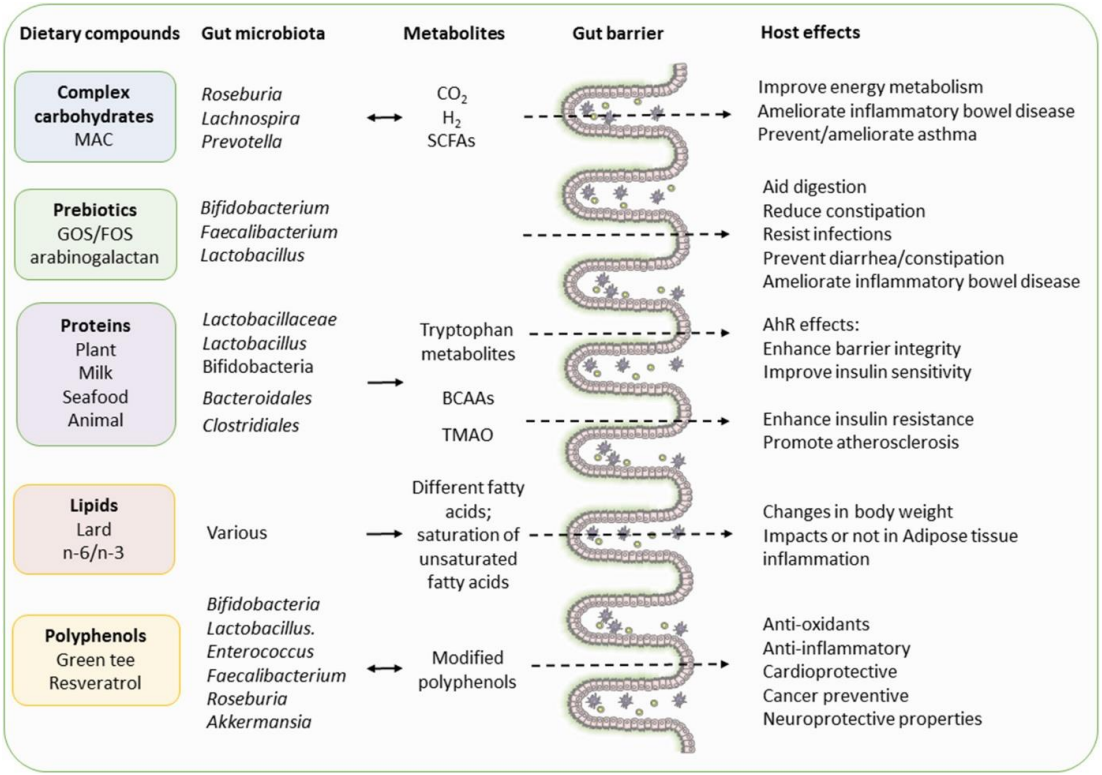
- The gut microbiota is highly variable from person to person
- Twin studies have revealed that human microbiota is heritable
- The microbes in our bodies collectively make up to 100 trillion cells (10x the number of human cells)



- Genetic potential of gut microbes:
  - Characterization of 3.3 million non-redundant microbial genes from faecal samples of 124 Europeans
  - The gene set ~150-fold larger than the human gene complement

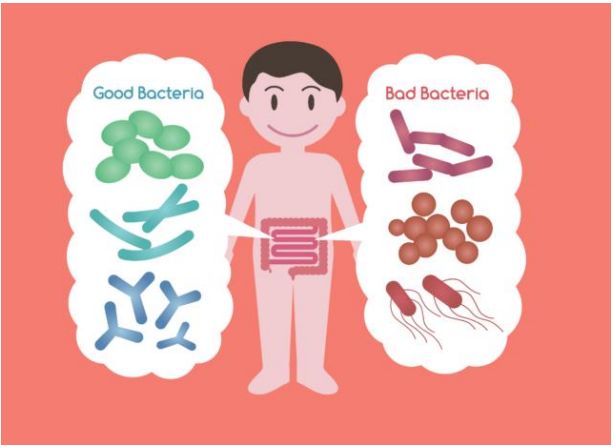
Human Microbiota





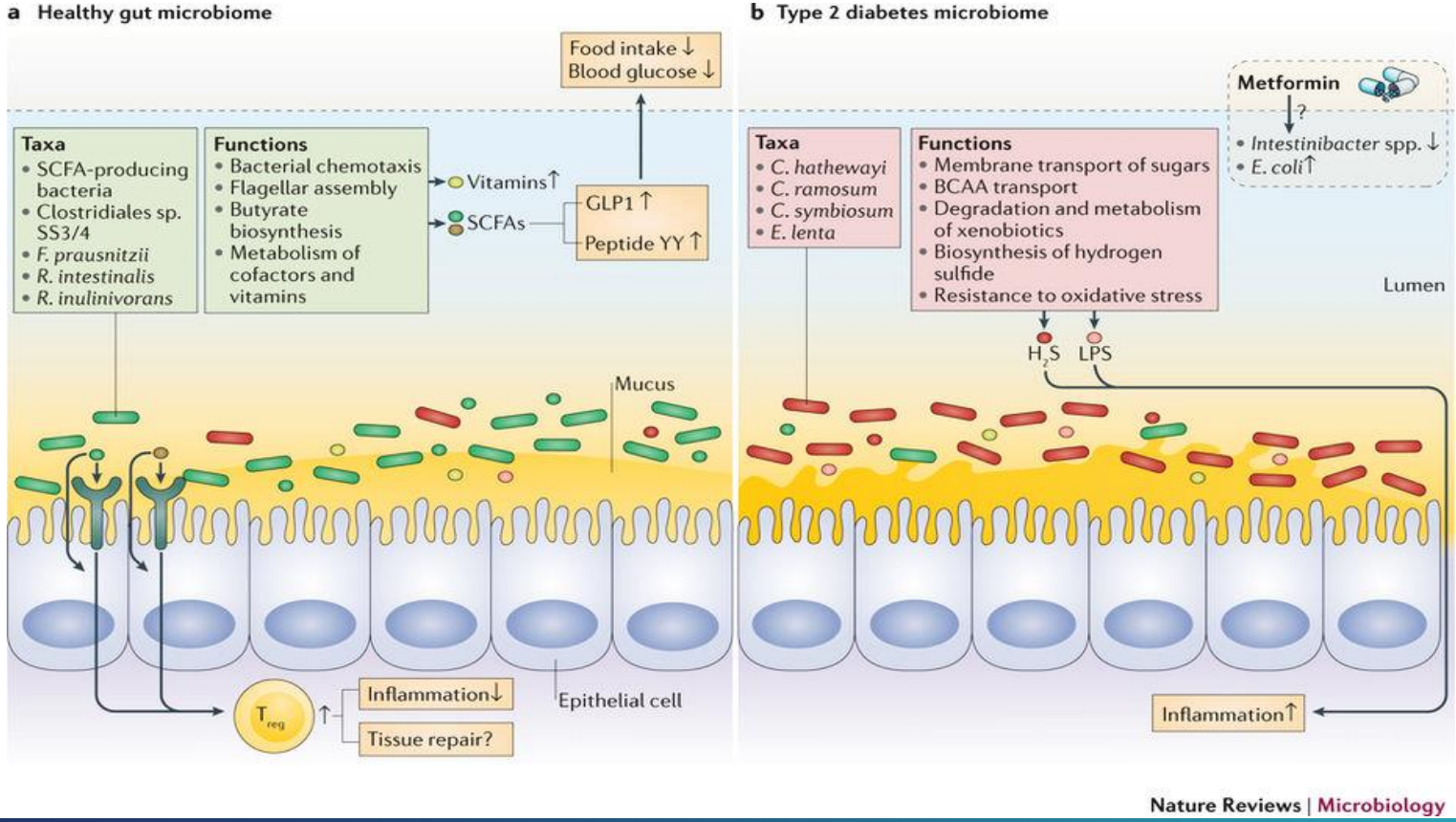
Danneskiold-Samsoe NB et al., *Food Res Int*, 2018
https://doi.org/10.1016/j.foodres.2018.07.043

Dysbiosis of the microbiome – Why is it important?



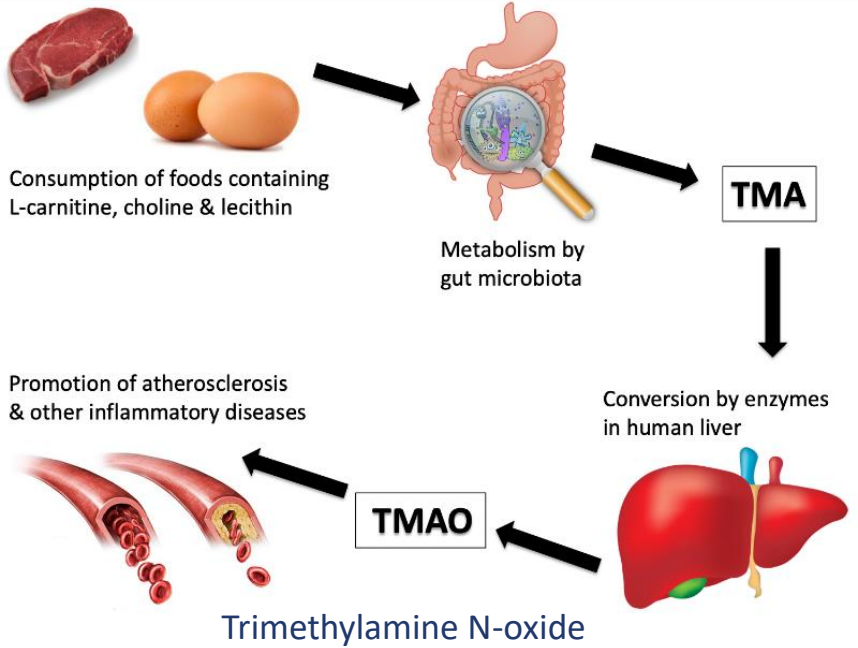
- Dysbiosis:
  - Loss of beneficial organisms
  - Excessive growth of potentially harmful bacteria
  - Loss of overall microbial diversity
- Changes in microbiota in chronic diseases
- **Is dysbiosis the cause or the consequence of disease?**

Metagenome-wide association in T2D



Wang J A et al., *Nat Rev Microbiol*, 2016
https://doi.org/10.1038/nrmicro.2016.83

Nutrigenetics in CVD – Dysbiosis of the gut microbiota



- Gut microbial metabolism of choline promotes atherosclerosis
- TMAO can lead to atherogenesis:
  - ↓ reverse cholesterol transport
  - ↓ cholesterol removal from peripheral macrophages
  - Affecting atheroprotective effects of HDL-C
- In a follow-up study of over 4,000 individuals: ↑TMAO associated with ↑ risk of major adverse cardiovascular events (death, MI and stroke)

Li XS et al. *Eur Heat J*, 2017
https://doi.org/10.1093/eurheartj/ehw582

Nutrigenetics – Vegetarianism

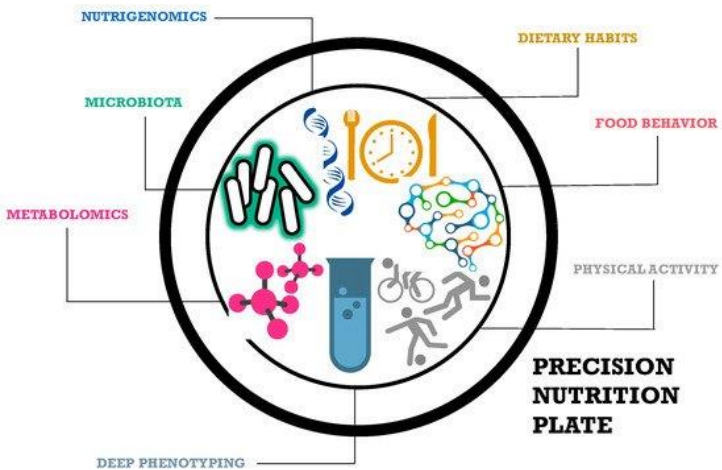
- Increase in adherence to plant-based diets (health benefits, ethical/environmental concerns)
- Is a vegan diet appropriate for everyone?

- Gene-vegetarianism interaction analyses for 30 biomarker traits (N = 147,253)
- rs72952628 (MMAA locus)-> the heterozygous genotype was associated with higher calcium in vegetarians
- Part of the B12 metabolism pathway; B12 has a high deficiency potential in vegetarian
- Gene-based analysis: *RNF168* with testosterone and *DOCK4* with eGFR
- Differences in genotype may moderate the vegetarian diet benefits

Franci M et al. *medRxiv 2022.10.21.22281358*, 2021
https://doi.org/10.1101/2022.10.21.22281358

What is precision nutrition?

- Precision nutrition at three levels:



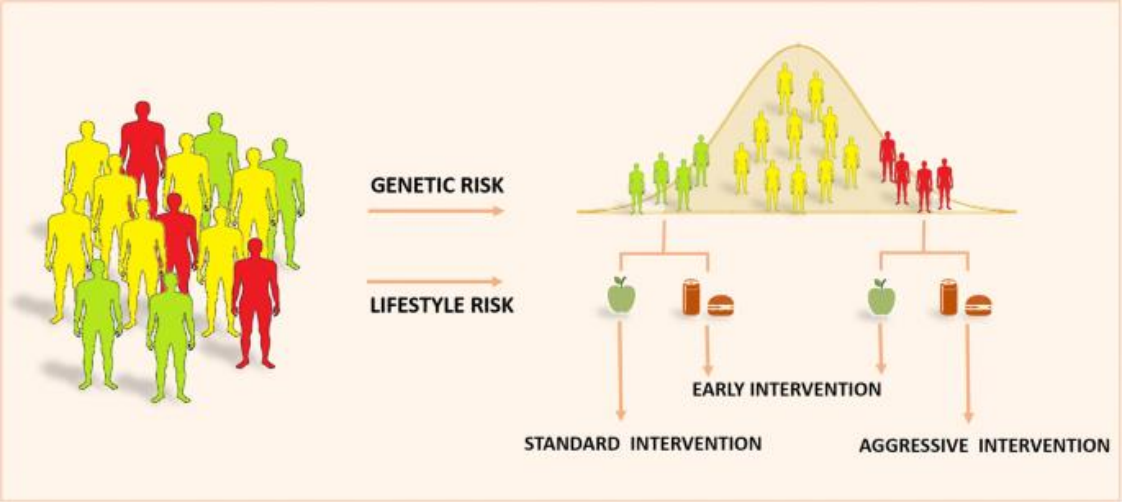
- Conventional nutrition based on general guidelines for population groups by age, gender and social determinants
- Individualized nutrition that adds phenotypic information about nutritional status, anthropometry, biochemical and metabolic analysis, physical activity
- Genotype-directed nutrition based on rare or common gene variation

Limitation in Nutrigenetics

- Accurate measurement of dietary intake
- Large sample sizes for robust results (expensive, less replication rates)
- Confounders and lack of causality evidence
- Commercial use of nutrigenetic test (ethics concerns, interpretability)
- Properly trained health professionals to assess such data and advice accordingly

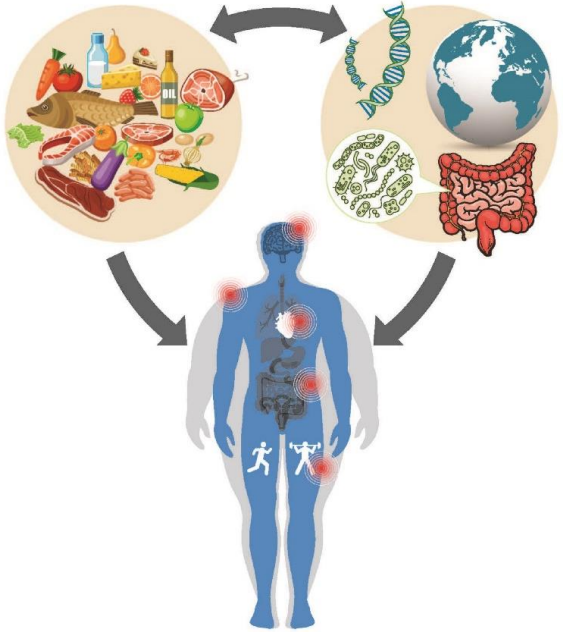
Implications of nutrigenetics

- Potential implications of nutrigenetics on public health:
  - Definition of personalized dietary requirement identification
  - Identification of nutrient intake combinations ideal for the homeostasis of specific genomic profiles
  - Better understanding of epidemiological data, clarifying the origin of the heterogeneous responses measured in populations after specific dietary intervention
  - Optimized intervention and prevention strategies



Conclusions [2]

- Complex human conditions could be modulated by the diet and lifestyle
- There is a strong genetic background
- Gene-by-environment interactions and the microbiome emerge as significant health factors
- Integrated omics approaches help identify relationships between diet and health
- New therapeutic approaches, targeted modification of dietary intake, pharmacotherapies, microbiome modulation



Further reading

- Ramos-Lopez O, et al: Guide for Current Nutrigenetic, Nutrigenomic, and Nutriepigenetic Approaches for Precision Nutrition Involving the Prevention and Management of Chronic Diseases Associated with Obesity. *J Nutrigenet Nutrigenomics* 2017;10:43-62. doi: 10.1159/000477729
- Marcum, J.A. Nutrigenetics/Nutrigenomics, Personalized Nutrition, and Precision Healthcare. *Curr Nutr Rep* **9**, 338–345 (2020). doi: 10.1007/s13668-020-00327-z
- Gul K, Singh AK, Jabeen R. Nutraceuticals and Functional Foods: The Foods for the Future World. Crit Rev Food Sci Nutr. 2016 Dec 9;56(16):2617-27. doi: 10.1080/10408398.2014.903384. PMID: 25629711
- Ojo O, Jiang Y, Ojo OO, Wang X. The Association of Planetary Health Diet with the Risk of Type 2 Diabetes and Related Complications: A Systematic Review. Healthcare (Basel). 2023 Apr 13;11(8):1120. doi: 10.3390/healthcare11081120. PMID: 37107955; PMCID: PMC10138355

Thank you

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