



Hannelore Daniel

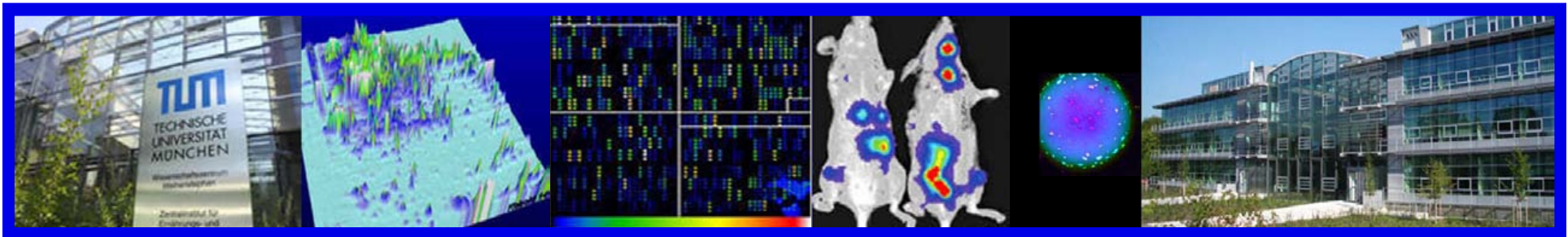


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Ernährungsforschung in den Lebenswissenschaften

Berlin, 15.12.2010

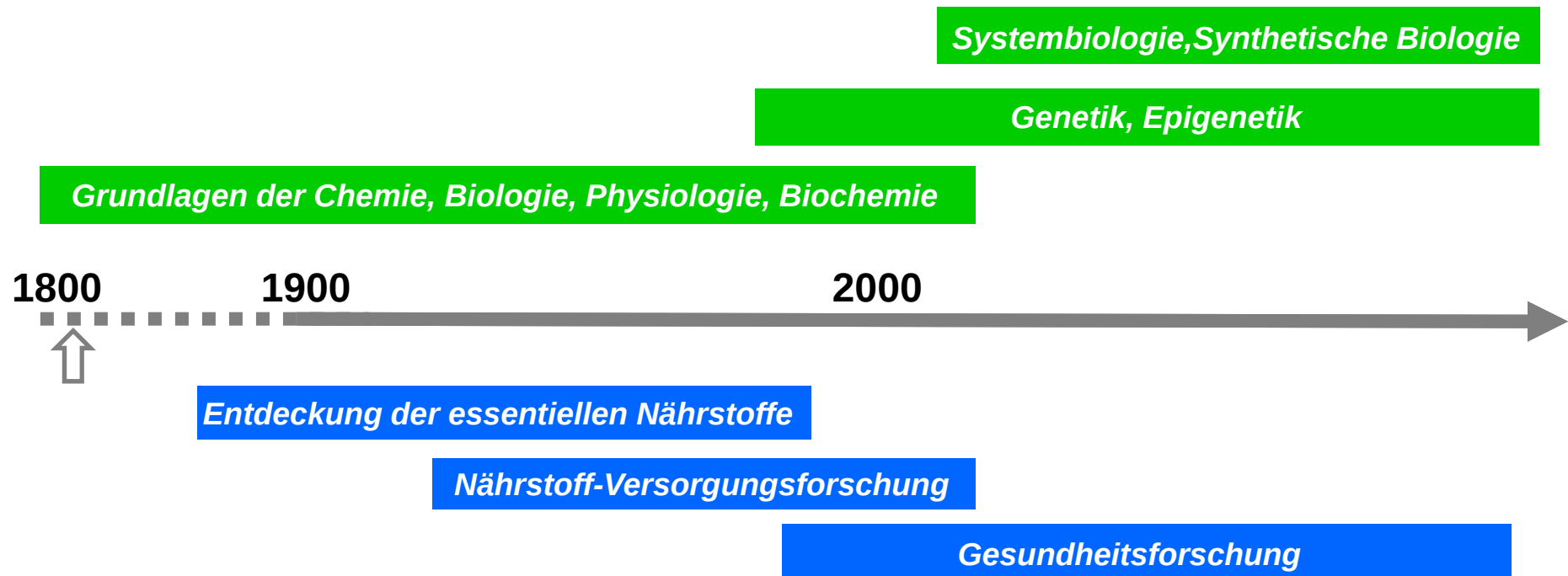


Odo Marquard

..... was keine Geschichte hat, hat keine *Zukunft!*



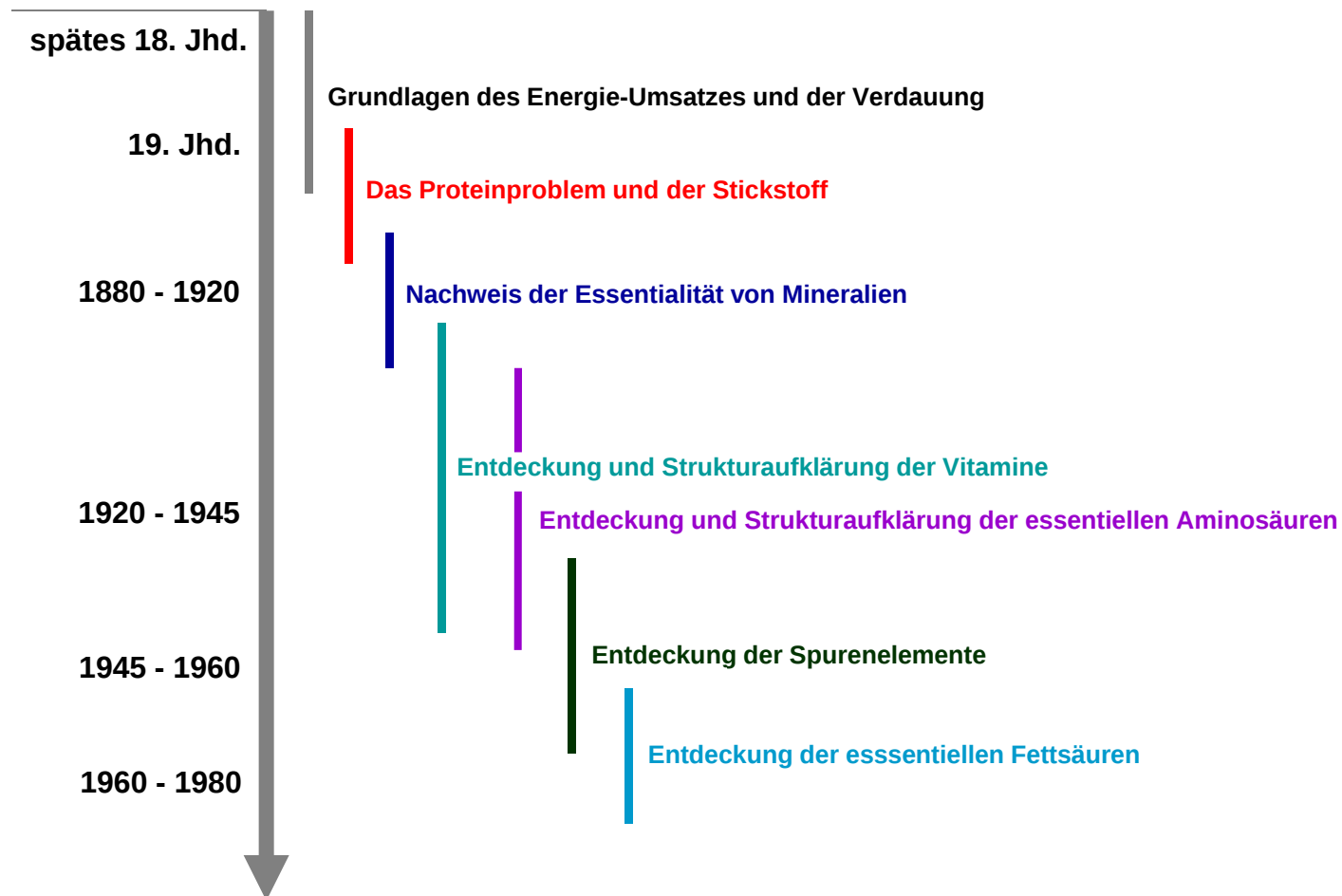
Die *EPOCHEN* der Ernährungsforschung





Die Zeit der *großen Entdeckungen* in der Ernährungsforschung

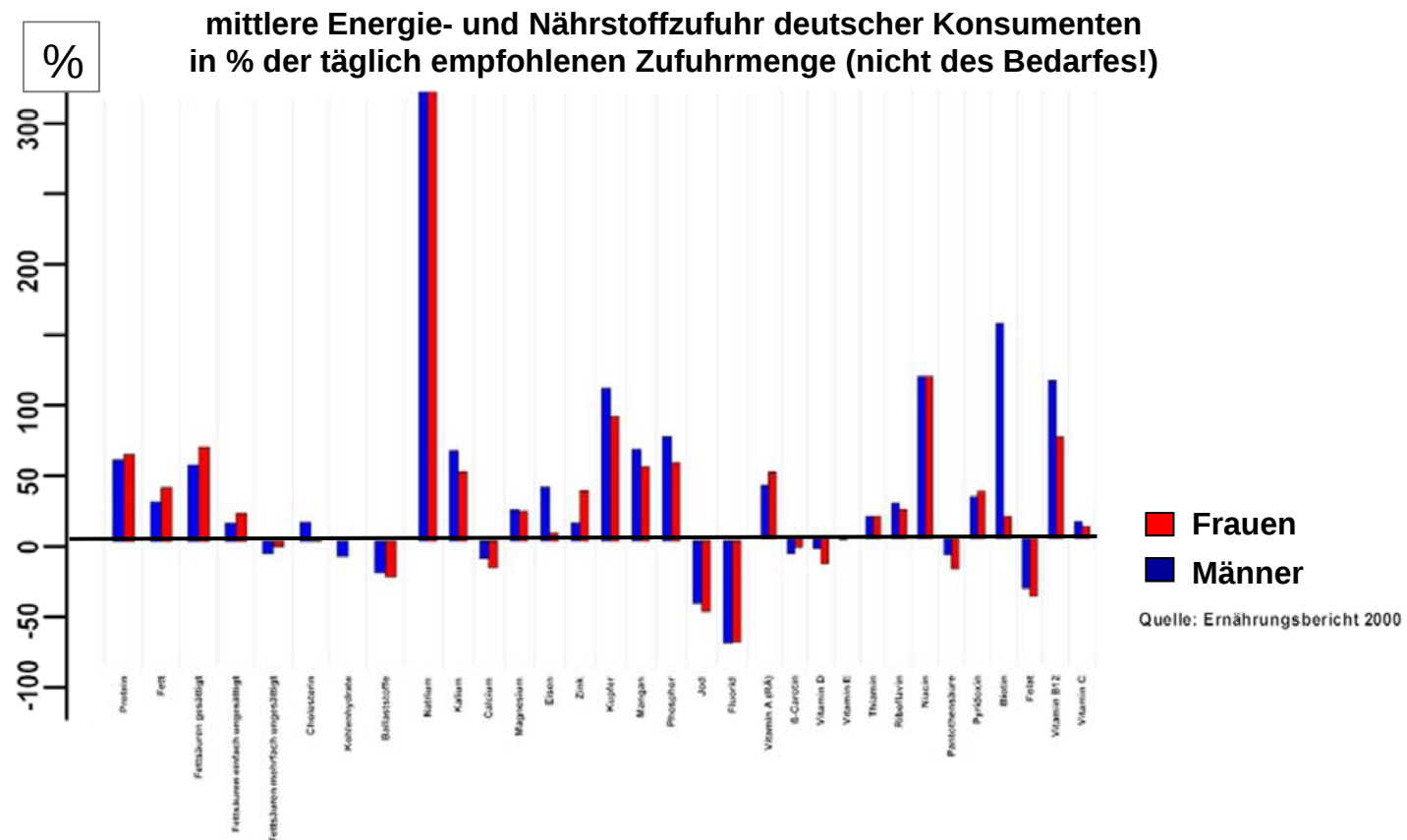
mit der Identifizierung der essentiellen Nährstoffe und ihrer Struktur





Der Nährstoff**MANGEL** als wissenschaftliches Leitmotiv

Generationen von Ernährungsforschern haben sich den Ursachen von Nährstoffmangelzuständen und ihrer Behebung gewidmet!





1960



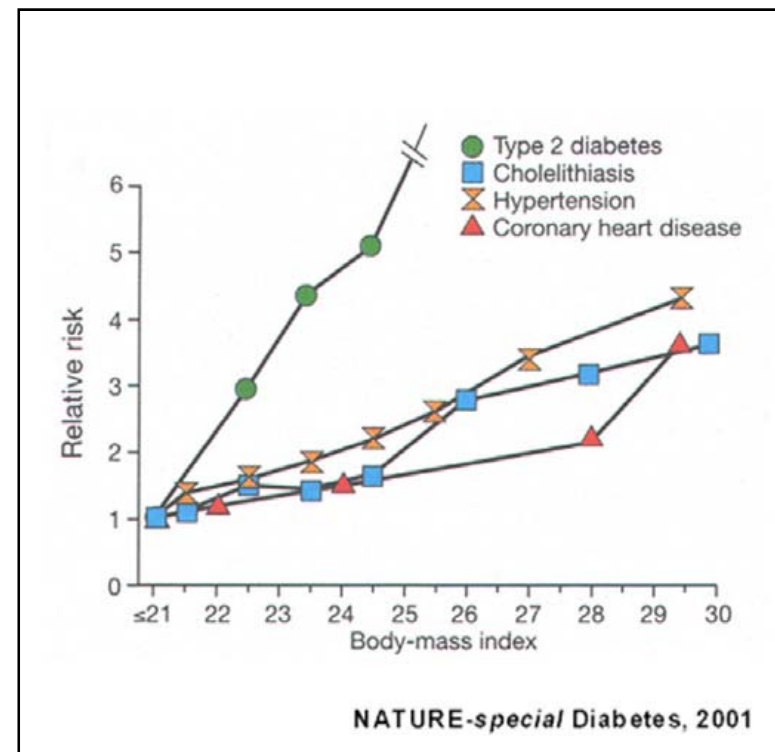
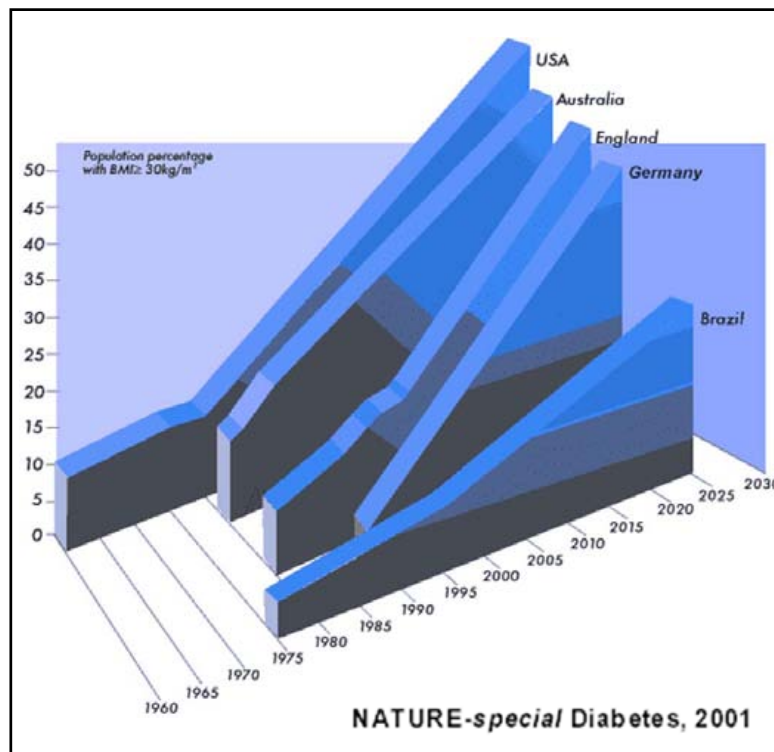
2010





Der Nährstoff**ÜBERFLUSS** als wissenschaftliches Leitmotiv

Generationen von Ernährungsforschern werden sich den Folgen der Überernährung und ihrer Behebung widmen!





The „new age“



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opportunities

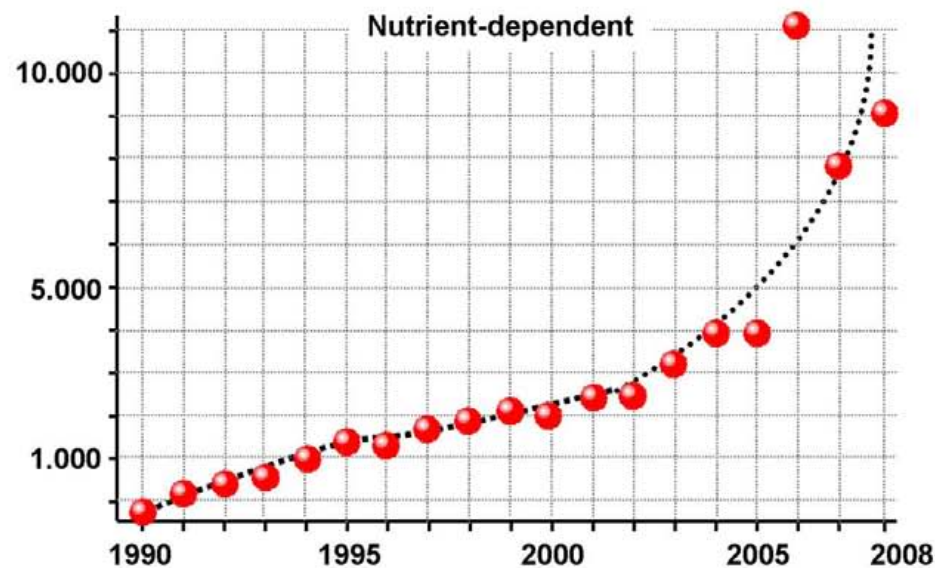
naturejobs
genomics special



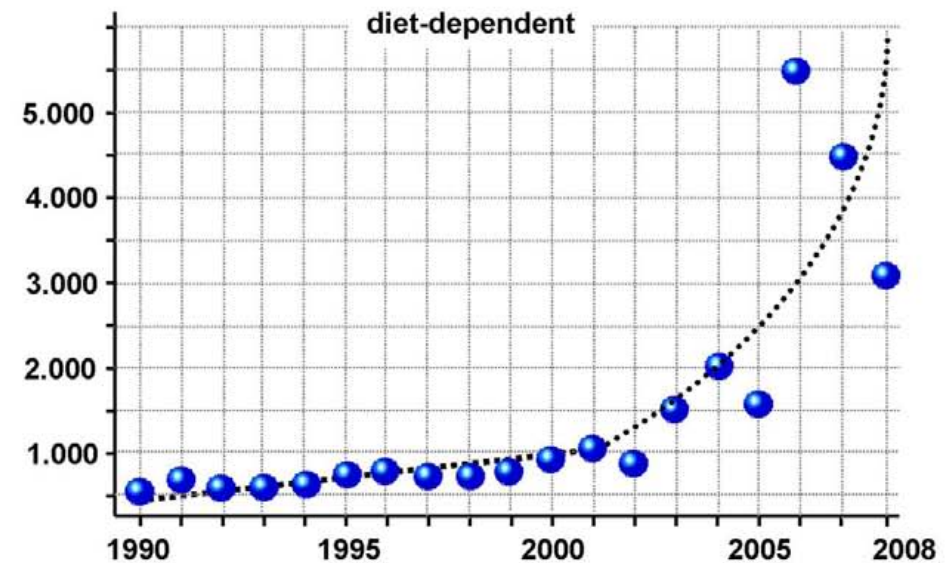
Ernährungsforschung - *en vogue*

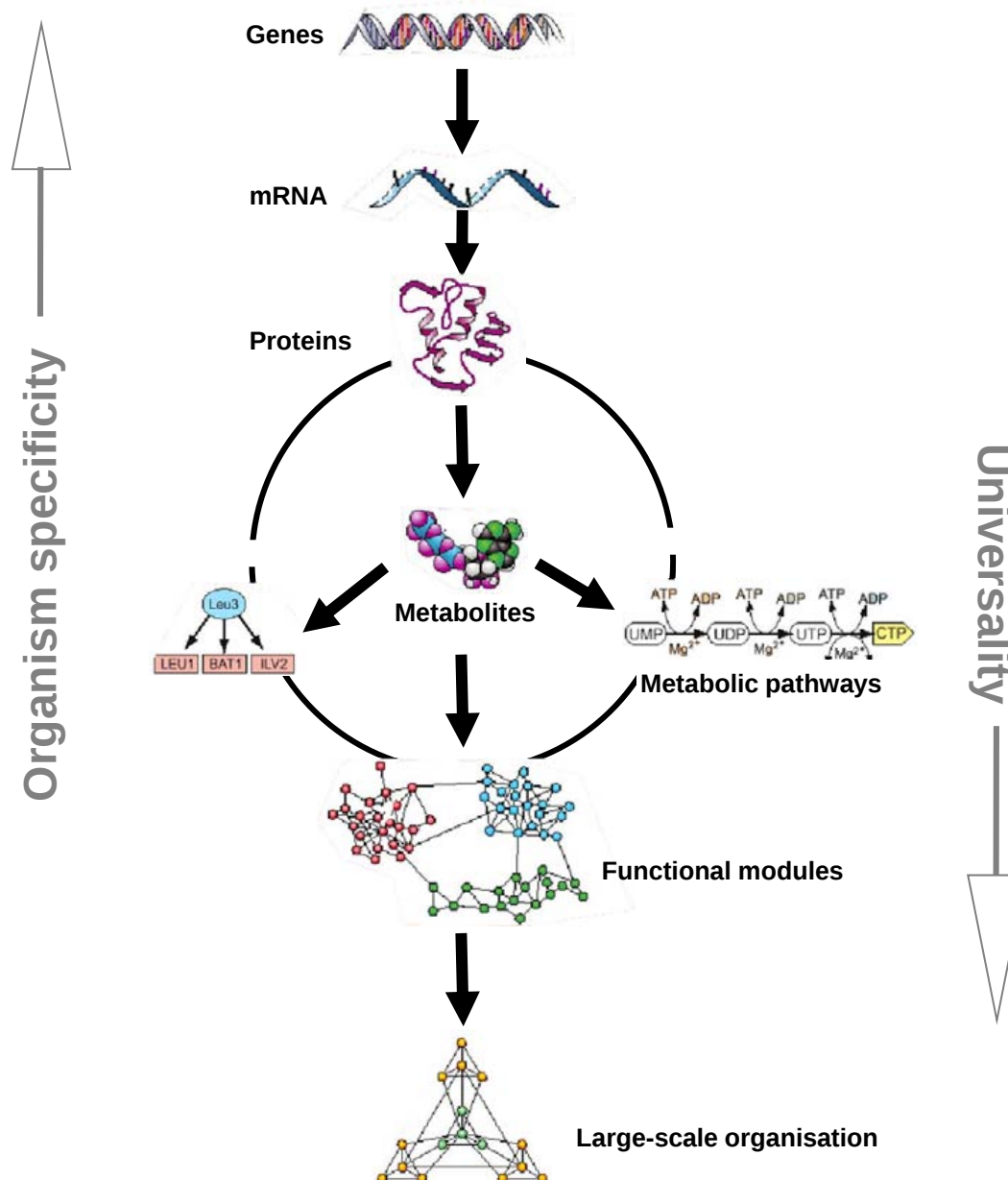
Analyse der Zahl der Publikation mit den Stichwörtern: nutrient or diet-dependent

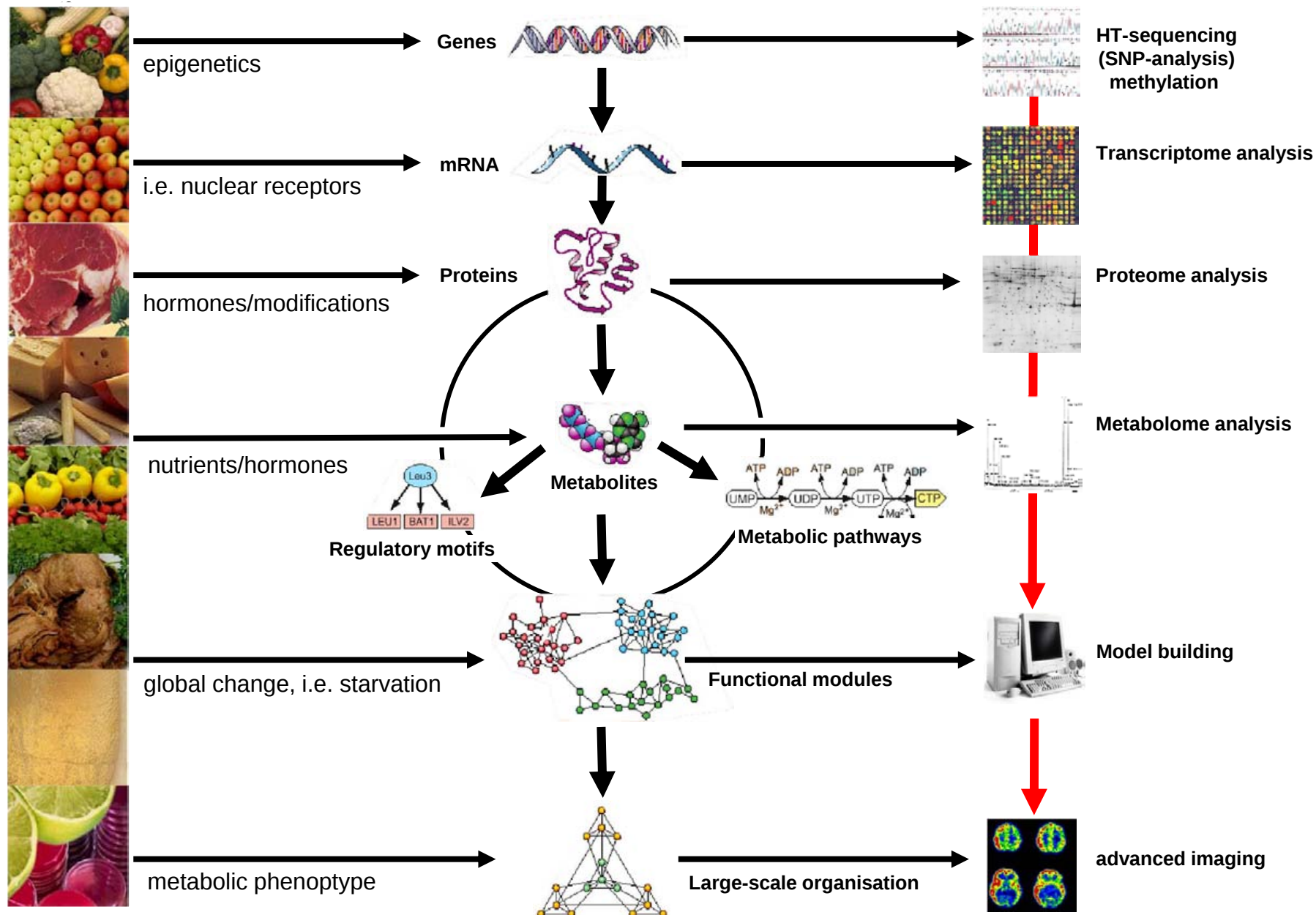
Pub-Med (number of citations per year) by key words



Pub-Med (number of citations per year) by key words

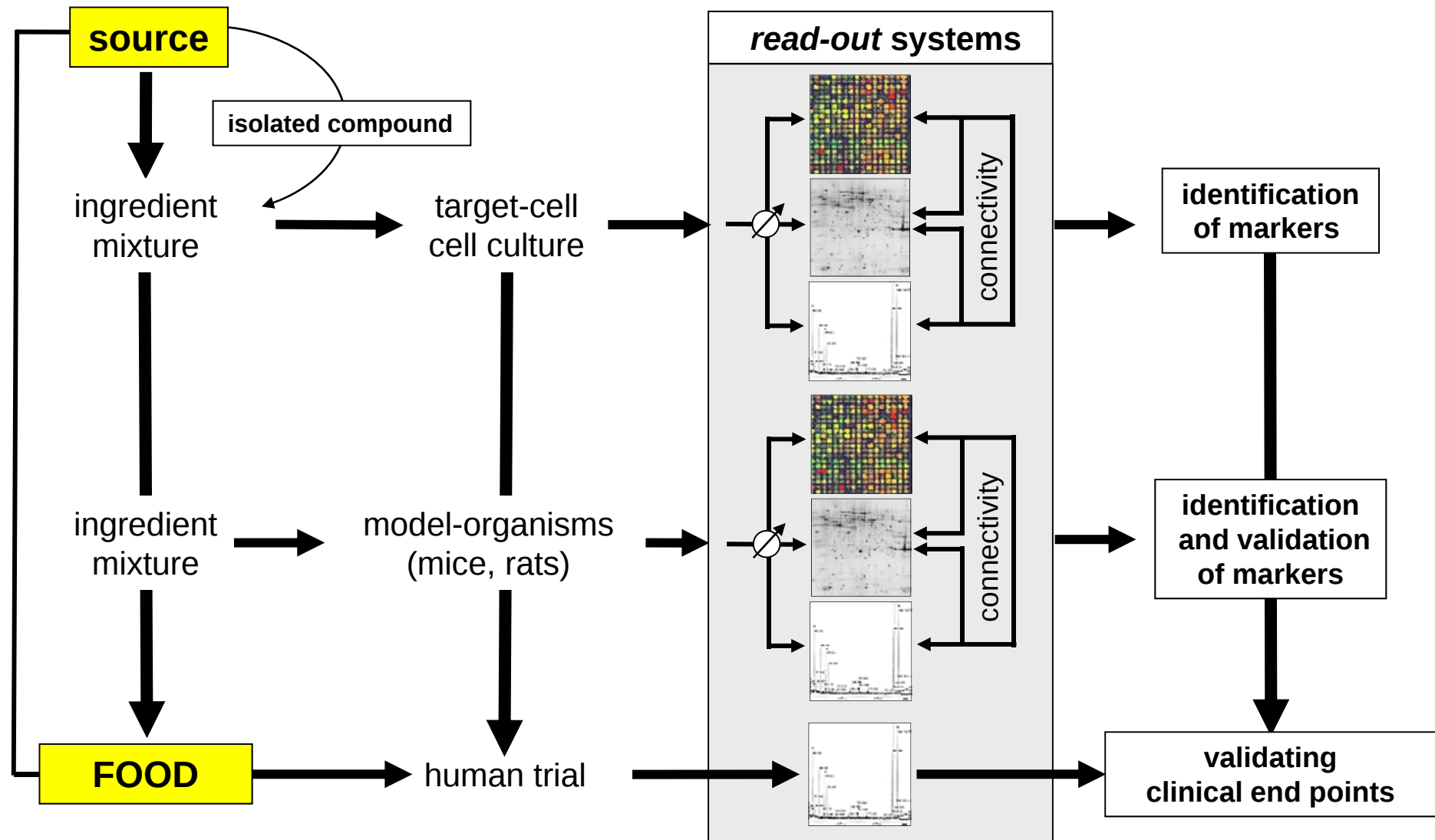






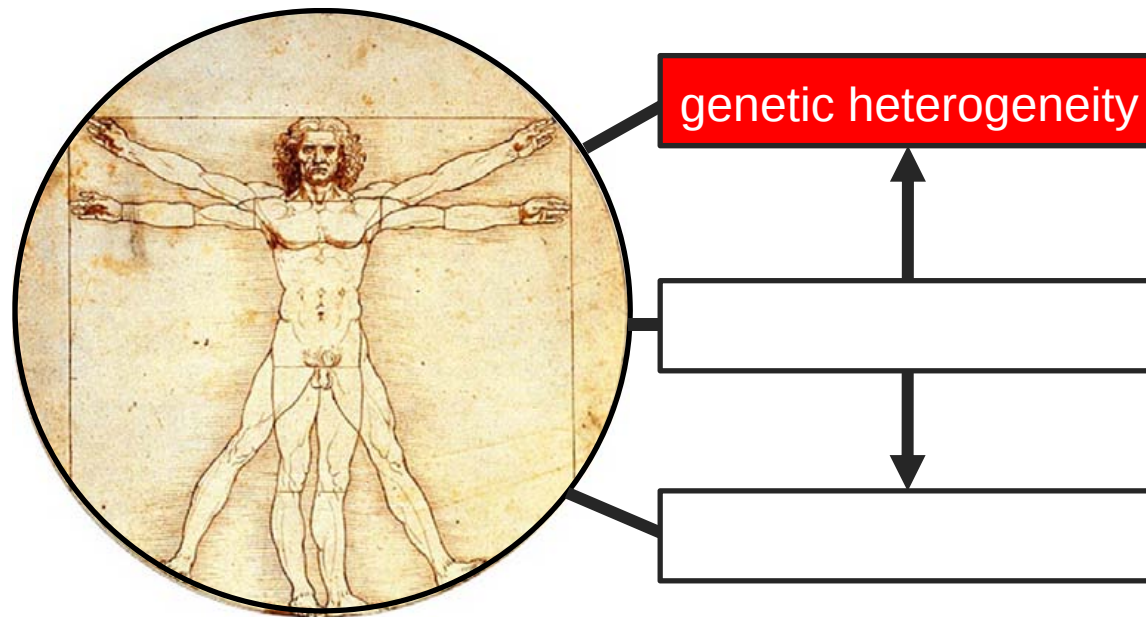


Profiling applications in industrial product development



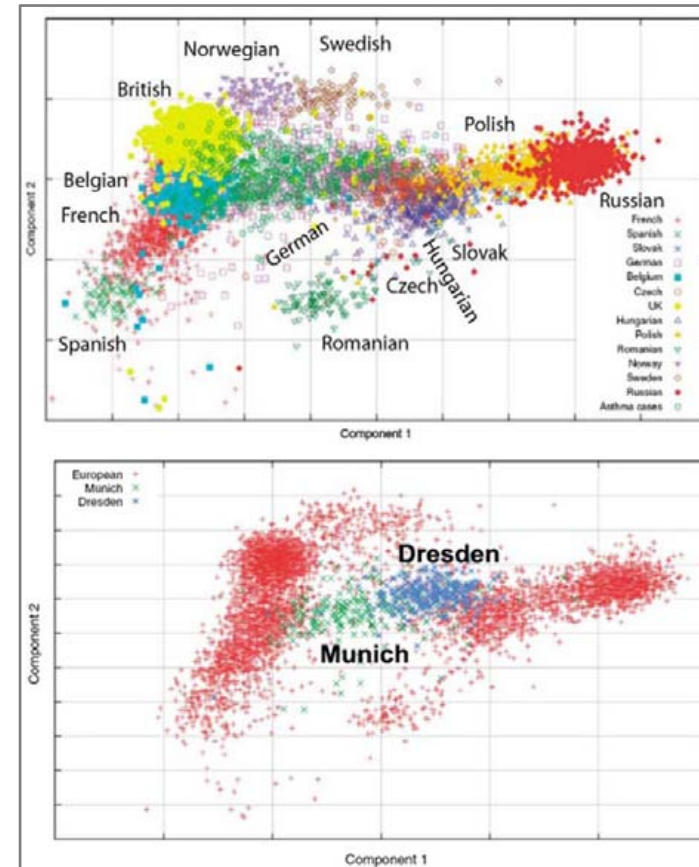
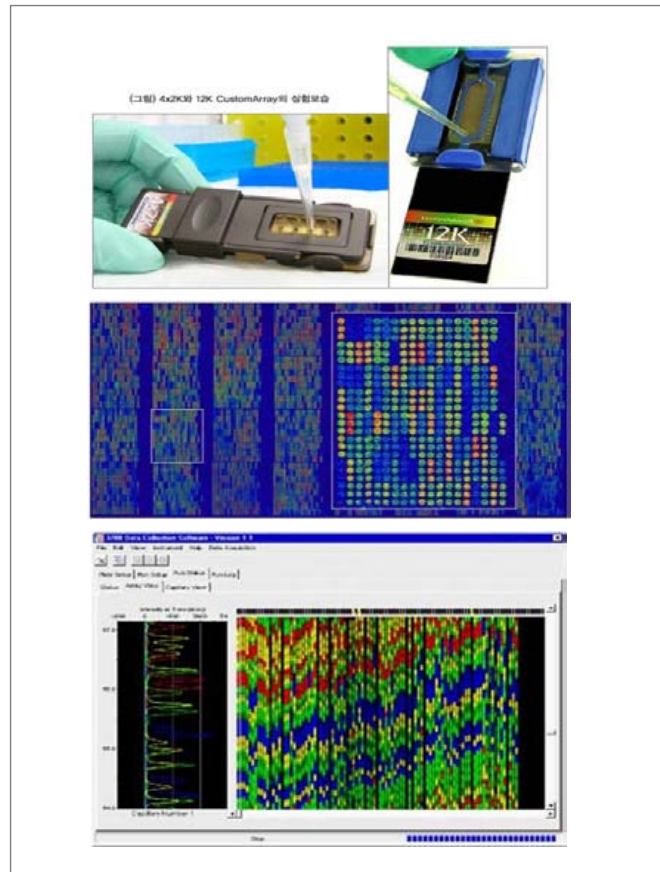


What defines the human metabolic phenotype ?





from the genome to the phenotype





The state of the art:

**identification of susceptibility genes
for diet-dependent processes**



identification of susceptibility genes for diet-dependent processes

- ✓ There is evidence for different requirements for micronutrients
example: **methylenetetrahydrofolatereductase SNP's & folic acid status**
- ✓ There is evidence for different capacities for metabolism and excretion of xenobiotics
examples: **N-Acetyl- and Glutathion-transferase SNP's**
- ✓ There is evidence for different beneficial effects of intake of MUFAS and PUFAS on lipid metabolism and cardiovascular disease risk
examples: **ApoA- and Apo-E polymorphisms**
- ✓ There is evidence for different susceptibilities for type 2 diabetes & metabolic syndrom
examples: **TCF7L2 and other gene SNPs**

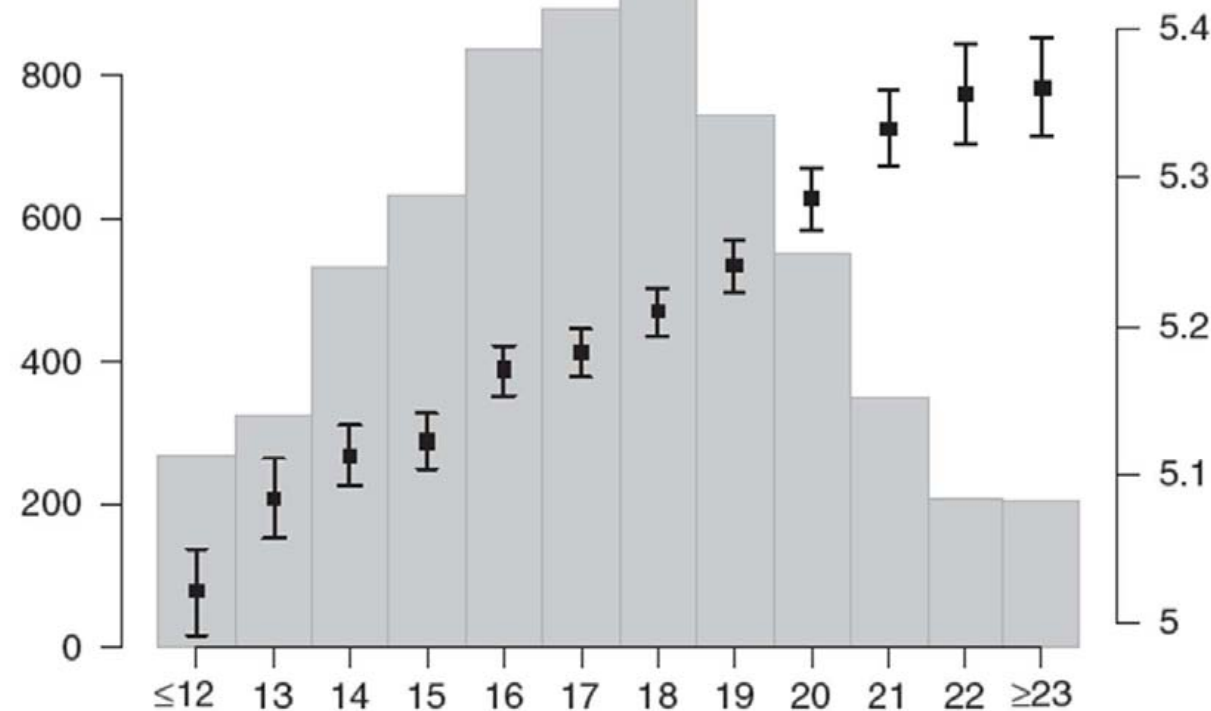


identified susceptibility genes in NIDDM

EXT2	Exostosin 2 pancreas development
WFS1	Wolfram syndrome 1/wolframin survival signal beta cells
CDKN2A/2B	Cyclin-dependent kinase inhibitor 2A/2B Tumorsuppressorgene
SCL30A8	Solute carrier family 30 [zinc transporter], member 30 insulin secretion
TCF2/HNF1B	HNF1 homoeobox B associated with T2D and (invers) prostata cancer
CDKAL1	Cyclin-dependent kinase 5 regulatory subunit associated protein 1-like 1:OR <u>1.39</u> per allele (p = 0.0004) Mechanism: Reduced insulin incretion
HHEX	Homoeobox, haematopoietically expressed: OR 0.81 per allele (p = 0.009) Mechanism: Reduced insulin incretion
IGF2BP2	Insulin-like growth factor 2 binding protein 2: OR 1.15 per allele (p = 0.049) Mechanism: pancreas development?, reduced insulin secretion
PPARG	<i>peroxisome proliferator-activated receptor</i> OR 0.76 per Allel (p = 0.010) Mechanism: fat regulation
FTO	Fat mass and obesity associated OR 1.15 per allele (p = 0.047) Mechanism: Appetite regulation?
DGKB	isotype of catalytic domain of DAG-kinase pancreas: DAG/PKC-dependent insulin secretion
ADCY5	adenylate cyclase 5 cAMP-dependent insulin secretion from beta cells
MADD	mitogen-activated protein kinase activating death domains control of β-cell mass
SCL39A13	Solute carrier family 30 [zinc transporter], member 30) TGF-β signalling
ADRA2A	α 2A adrenergic receptor in β-cell outward potassium channel – modifying insulin release
FADS1	Fatty acid desaturase synthesis of PUFA
CRY2	cryptochrome 2 circadian pacemaker
SLCA2	GLUT2-transporter mediates glucose uptake into β-cells, liver and other cells
IGF1	insulin-like growth factor 1



genetic loci for fasting plasma glucose levels



Variation in levels of fasting glucose depending on the number of risk alleles at newly identified loci, weighted by effect size in an aggregate genotype score for the Framingham Heart Study. The bar plots show the average and standard error of fasting glucose in mmol/l for each value of the genotype score based on the regression coefficient (right y axis), and the histogram denotes the number of individuals in each genotype score category (left y axis). Comparable results were obtained for the NFBC 1966 and ARIC cohorts. On average, the range spans ~0.4 mmol/l (~7.2 mg/dl) from low to high genotype score.



genotyping and nutrition and the commercial environment

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Nature Medicine | Commentary

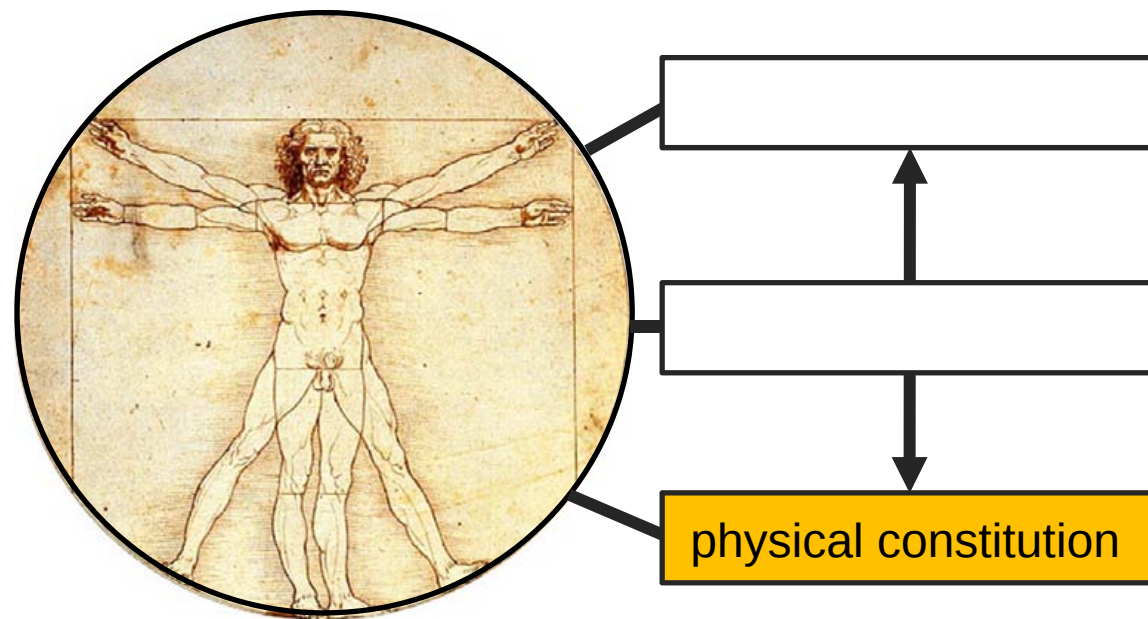
Christopher B Newgard & Alan D Attie

Getting biological about the genetics of diabetes

The first round of genome-wide association studies has not accounted for common human diseases to the extent that was expected. **New phenotyping approaches** and methods of data integration should bring these studies closer to their promised goals.



What defines the human metabolic phenotype ?





from genotype to phenotype



from genotype to phenotype

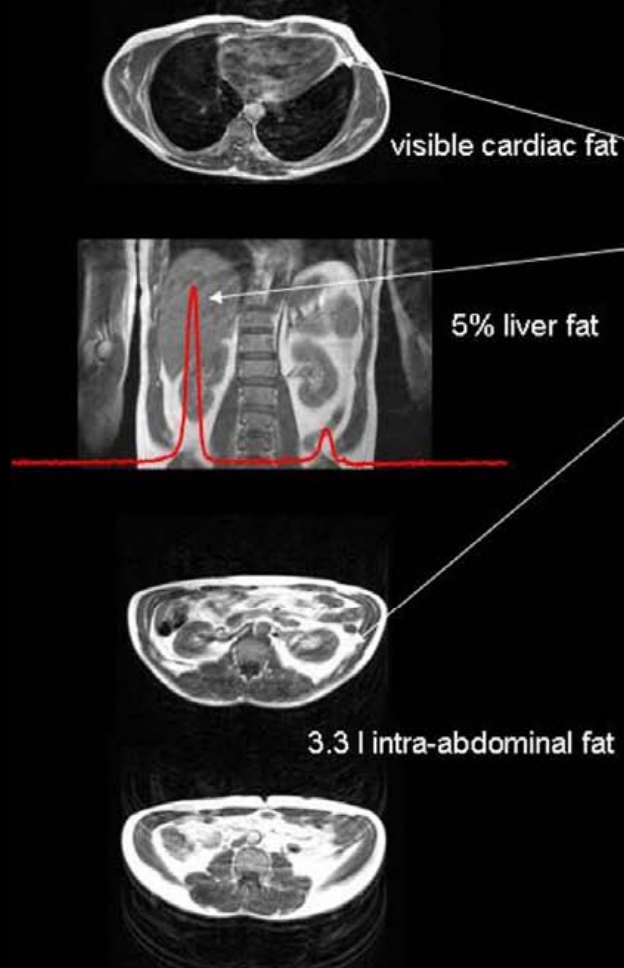


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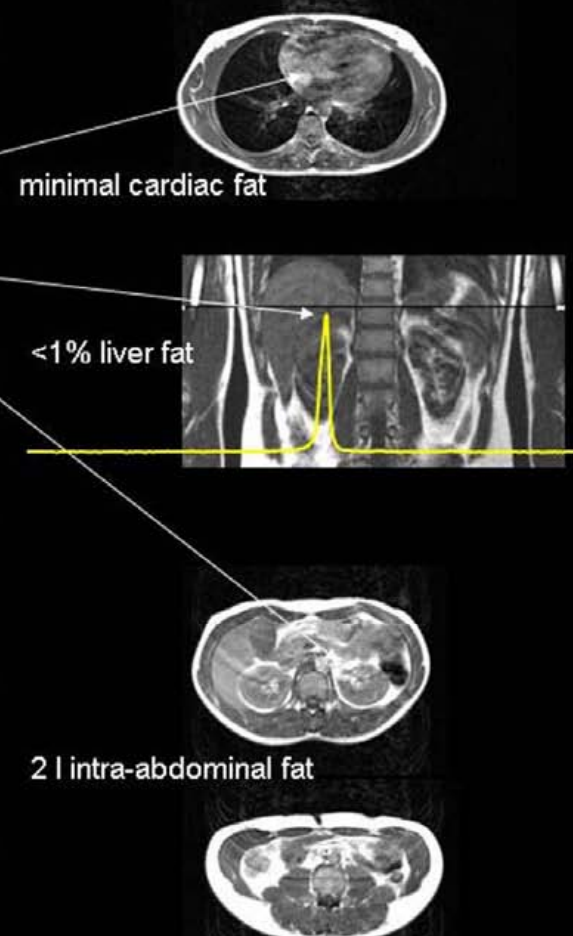


from genotype to phenotype

The TOFI Phenotype –
Thin Outside, Fat Inside



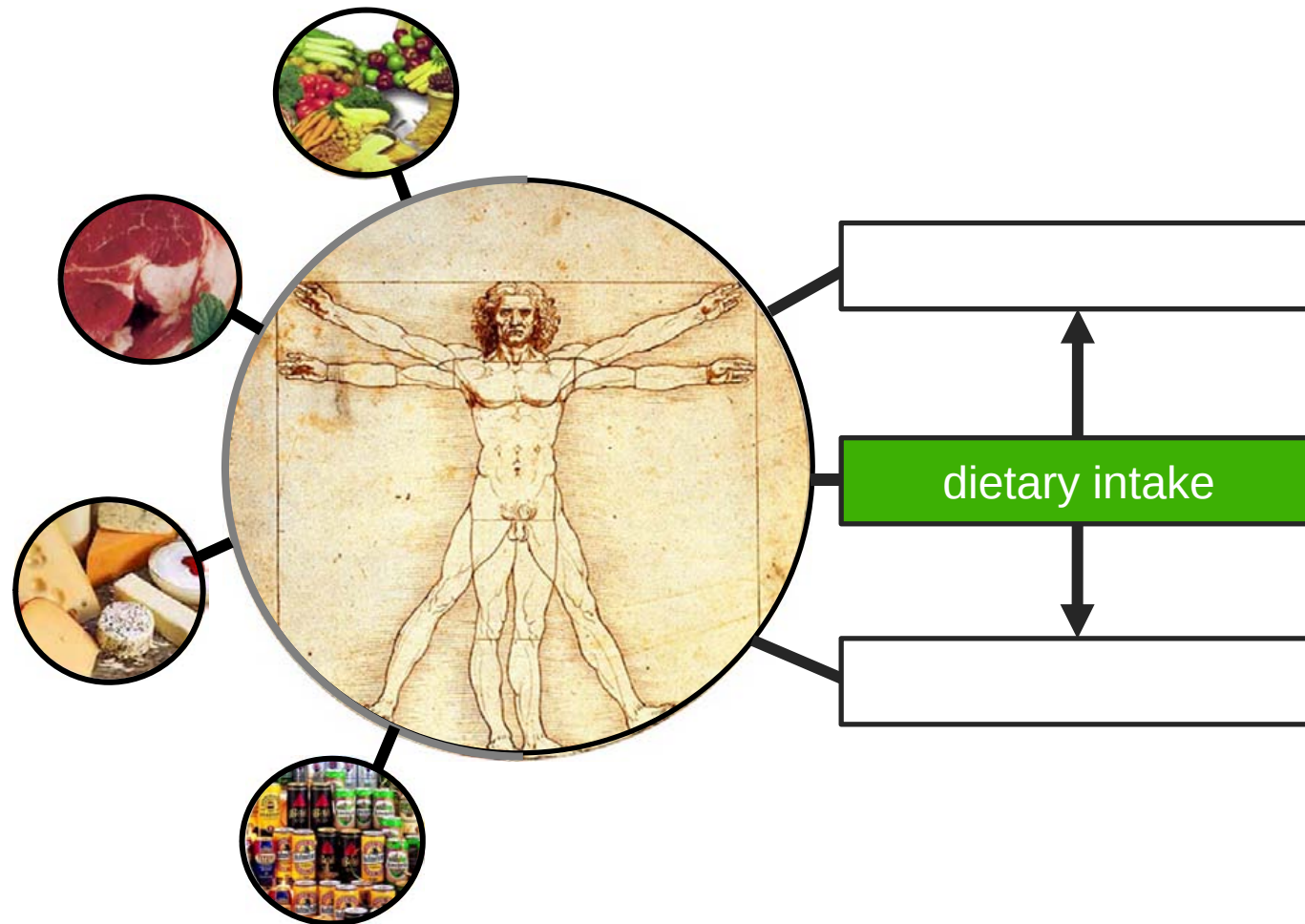
A FIPIO control subject –
Fat In Proportion Inside and Out



Jimmy Bell, Imperial College, London



What defines the human metabolic phenotype ?





Examples of profiling applications in human nutrition research.



Examples

The *FLAXSEED* intervention study

Biomarkers of a flaxseed intervention in human volunteers

The *ISOHEART* intervention trial

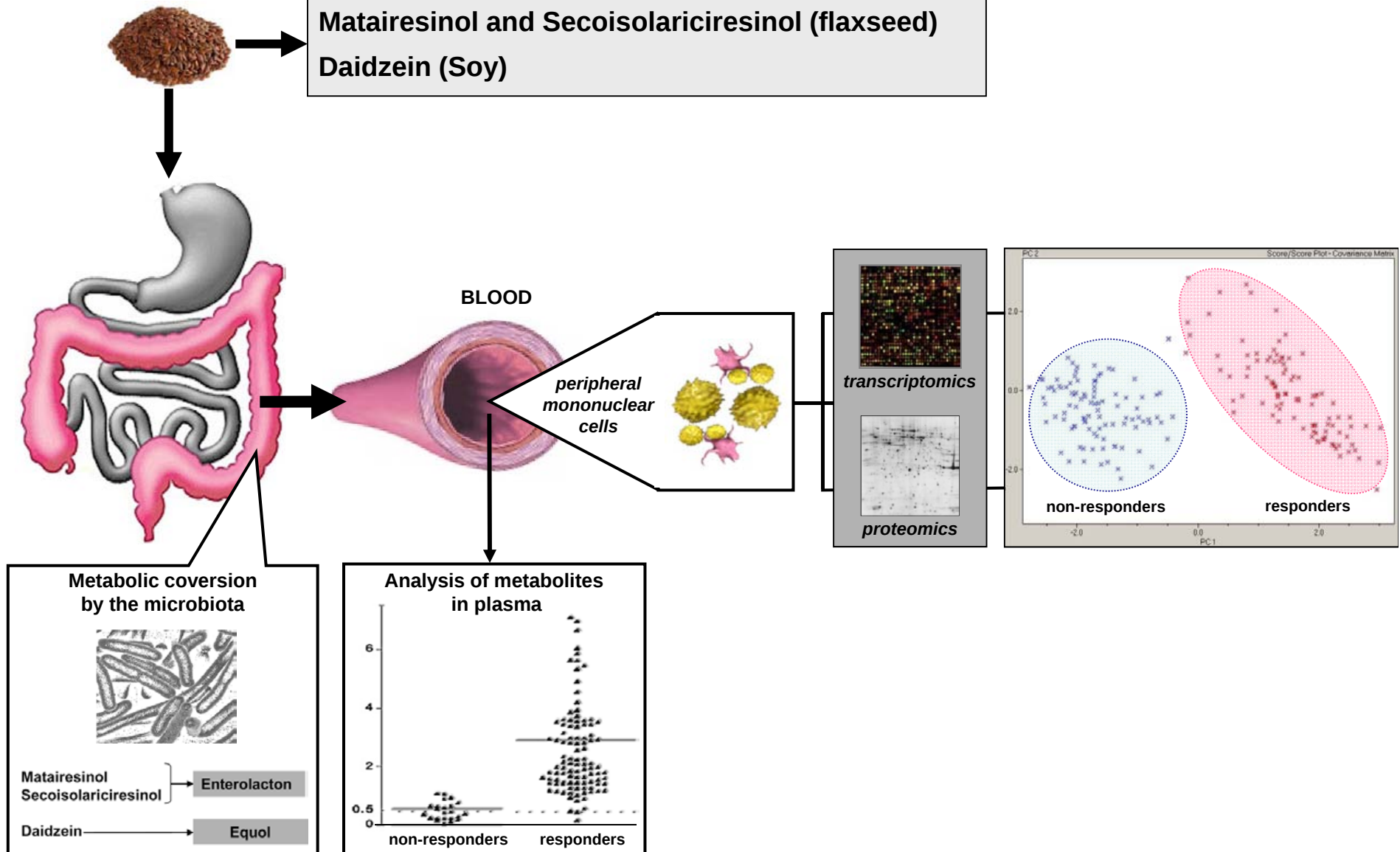
Transcriptomics and proteomic markers of a soy isoflavone intervention in postmenopausal women.



Functional ingredients

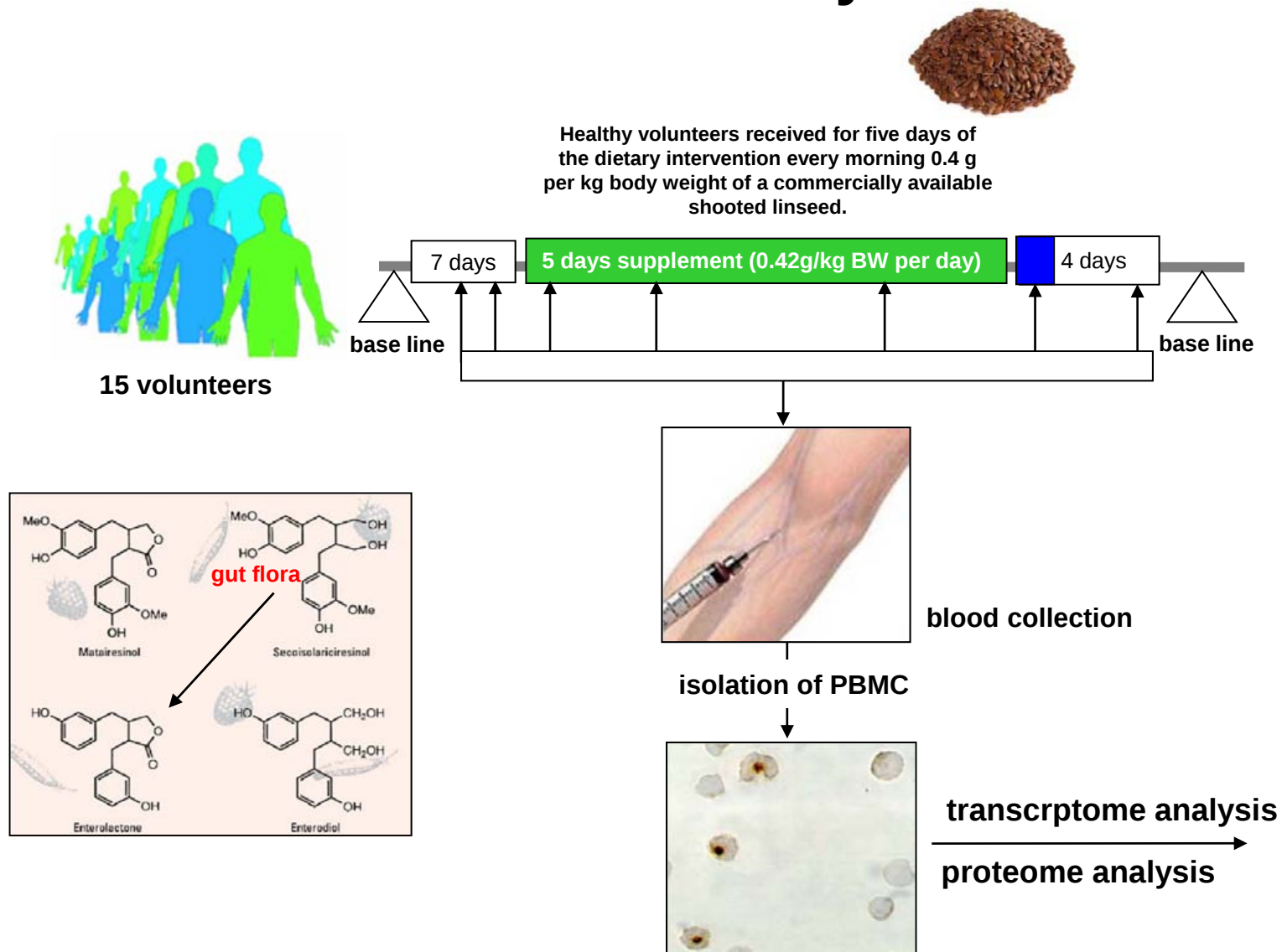
Matairesinol and Secoisolariciresinol (flaxseed)

Daidzein (Soy)





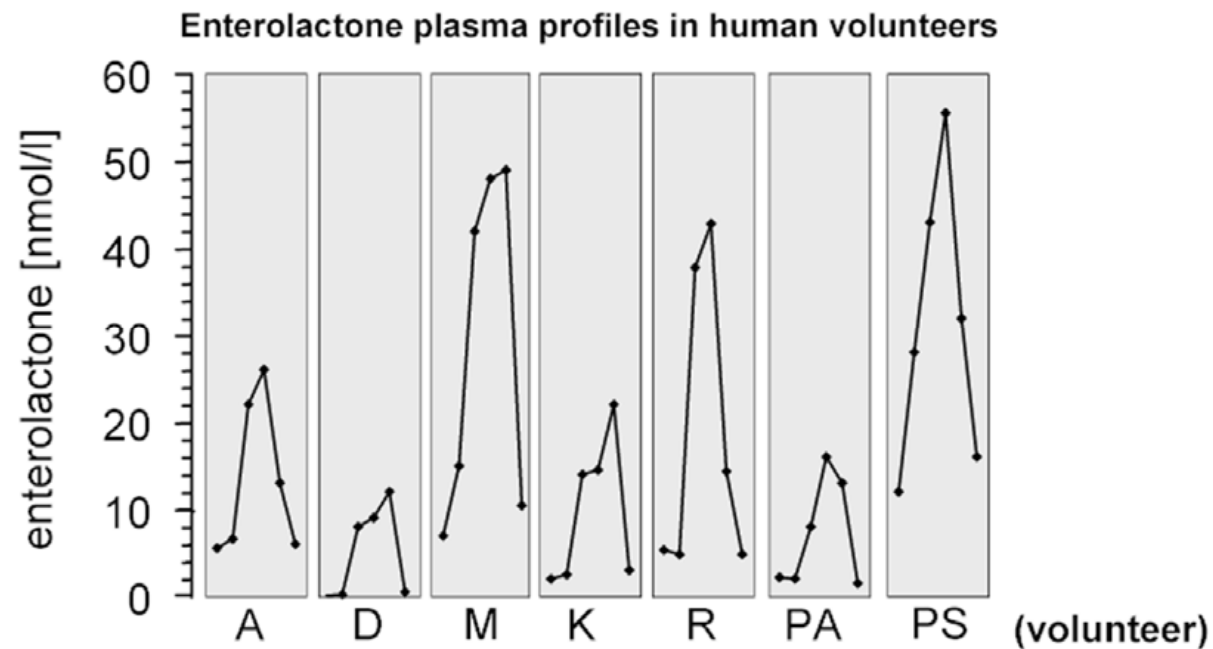
The *FLAXSEED* intervention study





Proteom-analysis for identification of markers of a flaxseed intervention

The human peripheral blood mononuclear cell proteome responds to a dietary flaxseed-intervention and proteins identified suggest a protective effect in atherosclerosis

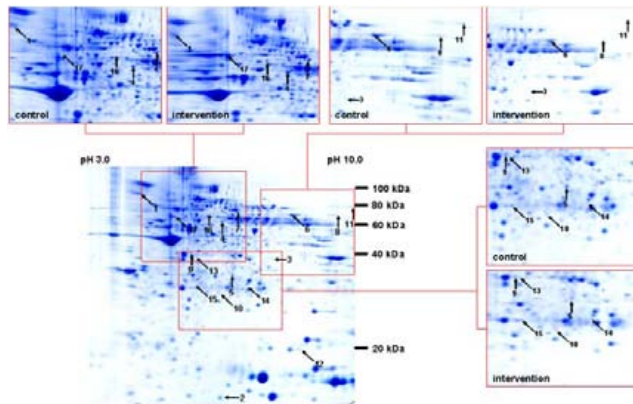


Phase	Day	Mean (nmol/L)	SEM	Median (nmol/L)	25. Percentile	75. Percentile
Pre-phase	-7	4.8	1.5	5.5	1.4	7.2
	0	8.4	3.9	5.4	1.4	14.2
Intervention-phase	2	24.9	5.9	21.8	8.1	42.4
	3	30.4	7.0	25.7	14.7	48.5
	7	22.0	5.2	14.8	12.1	32.0
Washout-phase	21	6.2	2.0	5.3	1.5	10.6



Proteom-analysis for identification of markers of a flaxseed intervention

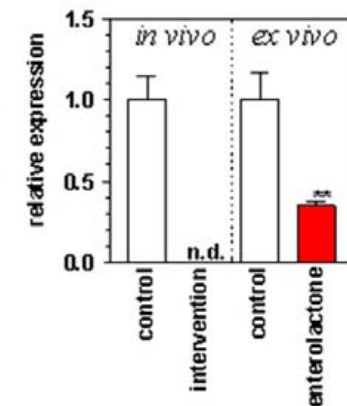
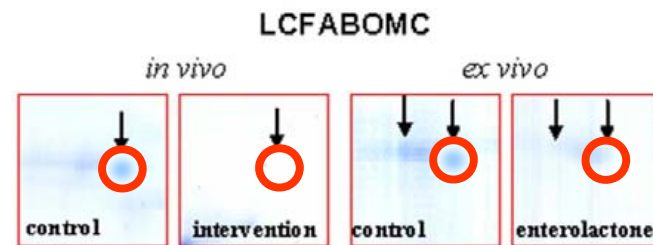
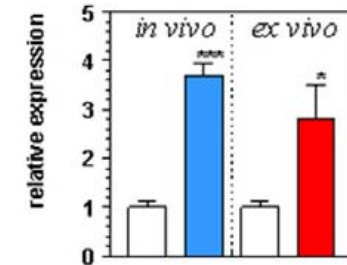
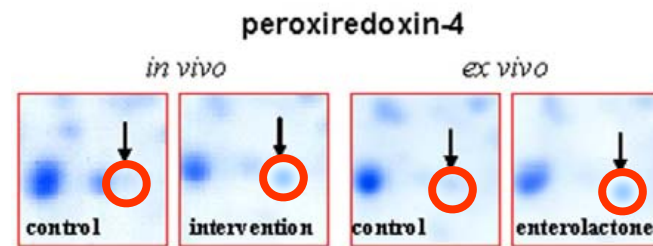
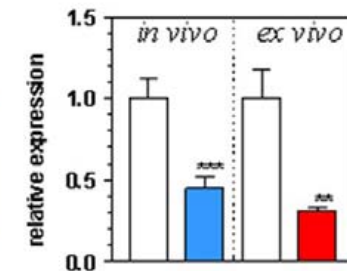
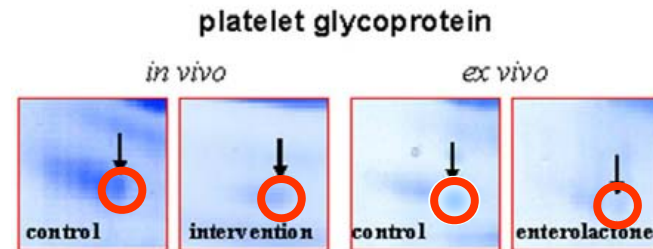
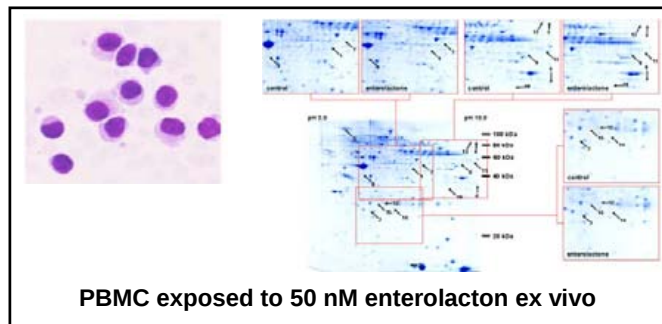
Identified protein-markers in PBMC



Spot No	Protein description	Theoretical M _r /pI	Measured M _r /pI	Protein amount		% Sequence-coverage	Accession No
				Intervention phase/pre-phase	Washout-phase/pre-phase		
	Chaperons						
1	Chaperonin-containing TCP-1 beta subunit homolog	96/6.4	58/6.0	2.05	0.86	36	AAC98906
2	Peroxiredoxin 4	38/5.3	31/5.9	3.70	1.05	64	Q13162
3	T-complex protein 1 subunit alpha (TCP-1-alpha)	121/5.8	61/5.8	2.01	0.95	30	P17987
4	60 kDa heat shock protein, mitochondrial precursor (hsp60)	210/4.6	61/5.7	only in intervention	n.d.	41	P10809
	Cytoskeletal proteins						
5	LIM protein	48/7.7	38/7.6	2.88	1.55	37	JC2324
6	Beta 5-tubulin	51/5.4	51/5.4	0.50	0.77	49	AAH20946
	Metabolism						
7	Pyruvate kinase isozymes M1/M2	134/8.7	58/8.0	2.04	1.84	51	P14618
8	Protein-L-isoaspartate (D-aspartate) O-methyltransferase(EC 2.1.1.77) splice form I	38/6.6	25/6.8	0.24	0.60	49	P22061
9	Long-chain-fatty-acid beta-oxidation multienzyme complex alpha chain precursor, mitochondrial	160/10.4	160/10.4	only in pre-phase	0.99	35	P40939
10	Cyclophilin A	21/8.7	18/7.7	only in pre-phase	0.76	50	P62937
11	TALDO 1 protein	54/5.4	37/5.8	only in intervention	only in intervention	26	AAH18847
12	Phosphoglycerate mutase 1 (Phosphoglycerate mutase isozyme B) (PGAM-B) (BPG-dependent PGAM 1)	39/6.9	29/6.8	2.86	1.65	56	P36871
	Gene regulation						
13	Purine-nucleoside phosphorylase (EC 2.4.2.1)	47/6.9	32/6.5	2.06	1.89	62	P00491
14	Stress-induced-phosphoprotein 1	154/6.8	63/6.4	2.01	only in pre-phase	44	P31948
	Other proteins						
15	Platelet glycoprotein IIIa/II Chain B, Crystal Structure Of Desoxy-Human Hemoglobin Beta6 Glu->trp	220/4.5	86/5.0	0.45	0.52	23	B36268
16		13/6.5	16/7.3	0.30	0.54	67	6HBWB
17	Gelsolin precursor	103/9.6	86/5.9	only in pre-phase	1.42	23	P06396

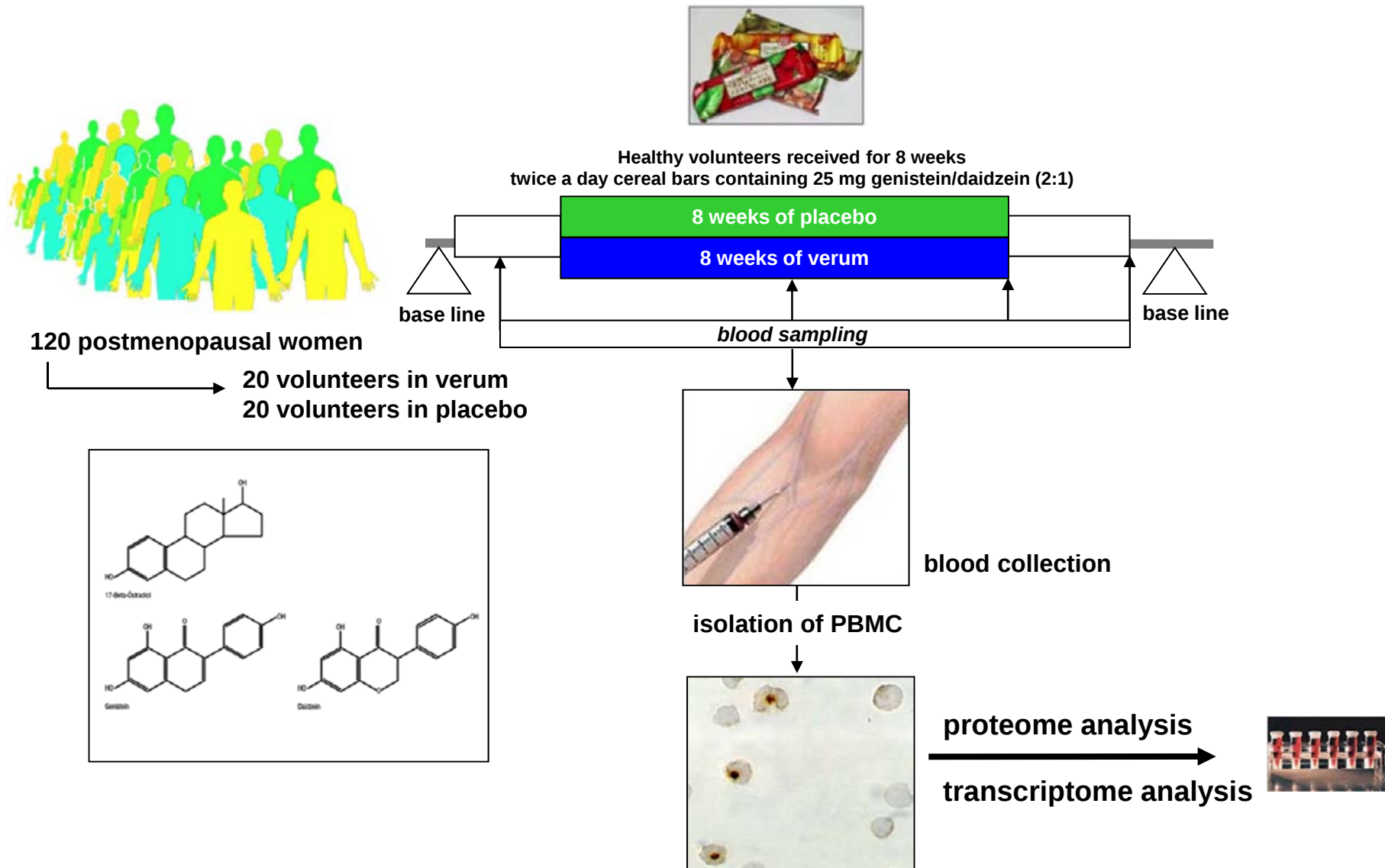


Proteom-analysis for identification of markers of a flaxseed intervention



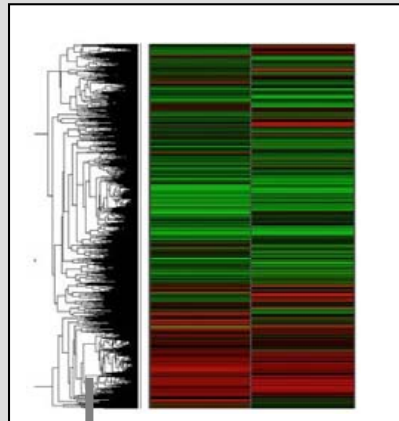


The *ISOHEART* intervention trial

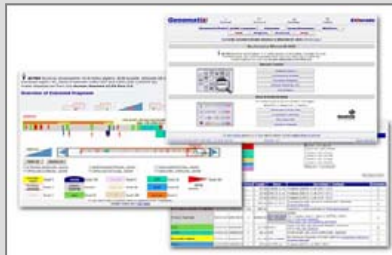




Profiling of PBMC of the volunteers undergoing isoflavone treatment

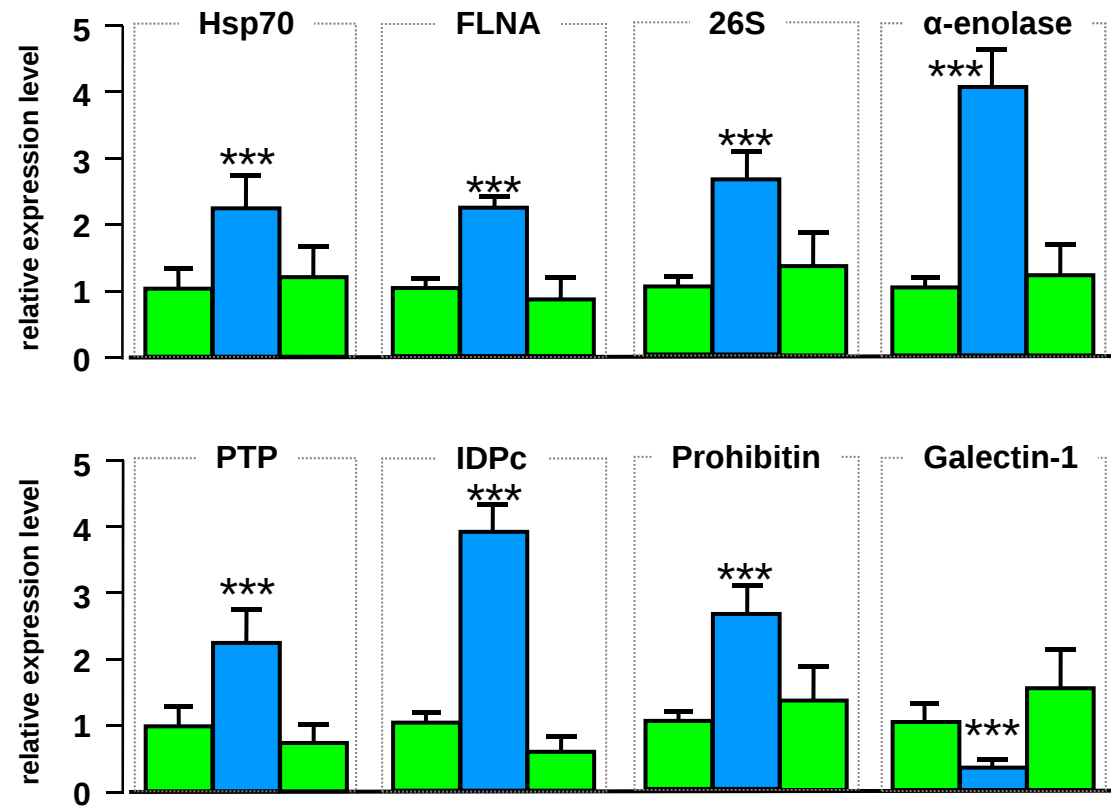


Promotor analysis and prediction
Pathway analysis tools



placebo

isoflavones





Profiling of PBMC of the volunteers undergoing isoflavone treatment

Clinical (inflammatory markers) of the volunteers undergoing isoflavone treatment

Plasma inflammatory factor concentrations at baseline (*t*0) and week 8 (*t*8) of the isoflavone and placebo intervention arms¹

	Isoflavones		Placebo		<i>P</i> ²
	<i>t</i> 0	<i>t</i> 8	<i>t</i> 0	<i>t</i> 8	
vWF (IU/dL)	104.96 ± 53.77 [116]	105.46 ± 53.07 [116]	103.27 ± 49.46 [115]	99.99 ± 39.92 [116]	0.883
sICAM-1 (ng/mL)	215.04 ± 51.60 [116]	220.40 ± 52.77 [117]	217.45 ± 52.21 [116]	217.78 ± 48.28 [115]	0.147
sVCAM-1 (ng/mL)	504.79 ± 134.39 [114]	503.48 ± 146.66 [113]	498.14 ± 129.00 [114]	499.76 ± 135.88 [111]	0.475
E-selectin (ng/mL)	42.14 ± 15.41 [117]	42.17 ± 15.82 [117]	40.67 ± 15.05 [117]	41.26 ± 15.17 [117]	0.307
MCP-1 (ng/mL)	259.36 ± 95.93 [117]	260.43 ± 101.23 [117]	262.40 ± 85.74 [117]	260.49 ± 106.17 [117]	0.928
Endothelin-1 (pg/mL)	1.15 ± 0.39 [107]	1.20 ± 0.43 [107]	1.15 ± 0.39 [106]	1.21 ± 0.40 [107]	0.800
hs-CRP (mg/L) ³	1.71 ± 1.89 [114]	1.70 ± 1.89 [113]	1.64 ± 1.73 [116]	1.76 ± 1.83 [113]	0.086

¹ All values are $\bar{x} \pm SD$, *n* in brackets. vWF, von Willebrand Factor; sICAM-1, soluble intracellular adhesion molecule 1; sVCAM-1, soluble vascular cell adhesion molecule 1; MCP-1, monocyte chemoattractant protein 1; hs-CRP, highly sensitive C-reactive protein.

Urinary isoflavone yields at baseline (*t*0) and week 8 (*t*8) of the isoflavone and placebo intervention arms¹

	Isoflavones		Placebo		<i>P</i> ²
	<i>t</i> 0	<i>t</i> 8	<i>t</i> 0	<i>t</i> 8	
Genistein (mg/d)	0.37 ± 0.40 [114]	7.27 ± 3.58 [117]	0.37 ± 0.40 [117]	0.42 ± 0.32 [117]	< 0.0001
Daidzein (mg/d)	0.16 ± 0.22 [114]	5.76 ± 2.70 [117]	0.22 ± 0.30 [117]	0.22 ± 0.32 [117]	< 0.0001
Equol (mg/d)	0.08 ± 0.06 [114]	0.85 ± 1.43 [117]	0.08 ± 0.05 [117]	0.11 ± 0.08 [117]	< 0.0001
Equol producers ³	0.10 ± 0.05 [31]	2.61 ± 1.73 [33]	0.09 ± 0.05 [33]	0.13 ± 0.09 [33]	< 0.0001
Equol nonproducers ⁴	0.07 ± 0.06 [82]	0.15 ± 0.08 [83]	0.08 ± 0.05 [84]	0.094 ± 0.08 [83]	< 0.0001

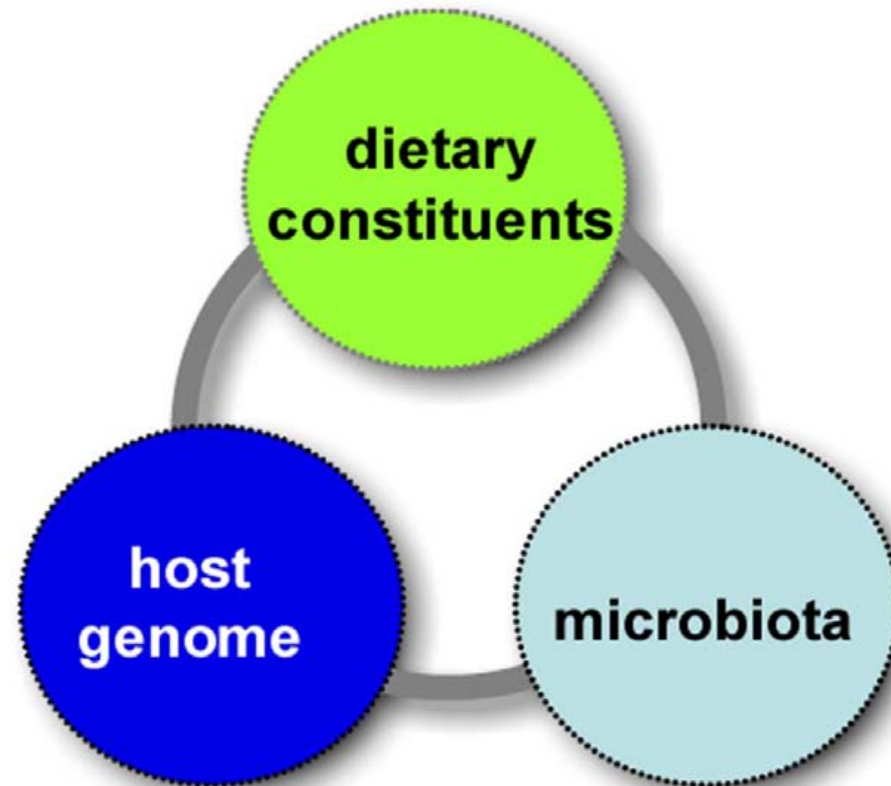
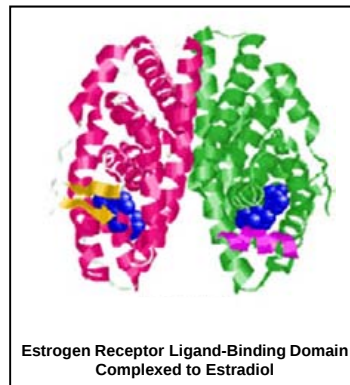
¹ All values are $\bar{x} \pm SD$, *n* in brackets.

The relative binding affinity of the R- and S-equol enantiomers for ERα were 0.47% and 2.0% with that of 17β-estradiol. However, S-equol is largely ERβ selective and has a relatively high affinity for this receptor subtype. S-equol binds ERβ with around 20% of the affinity of 17β-estradiol (equol: $K_i = 0.7$ nmol/L; 17β-estradiol: $K_d = 0.15$ nmol/L), whereas the R enantiomer only has 1% of the affinity.



Profiling of PBMC of the volunteers undergoing isoflavone treatment

Plasma vascular cell adhesion molecule 1 (VACM-1) concentrations in the volunteers according to estrogen receptor β AluI genotype and treatment

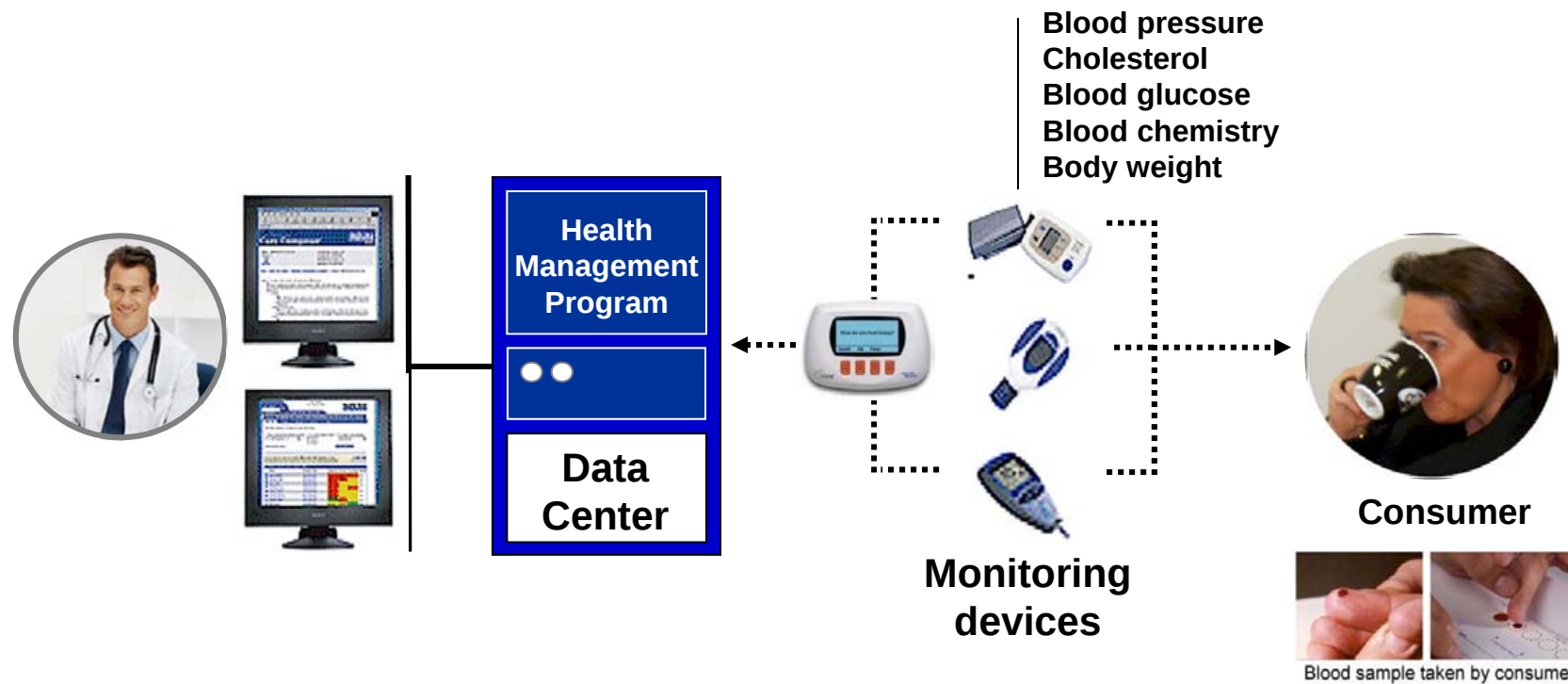




Nutrition, personalisation and e-health

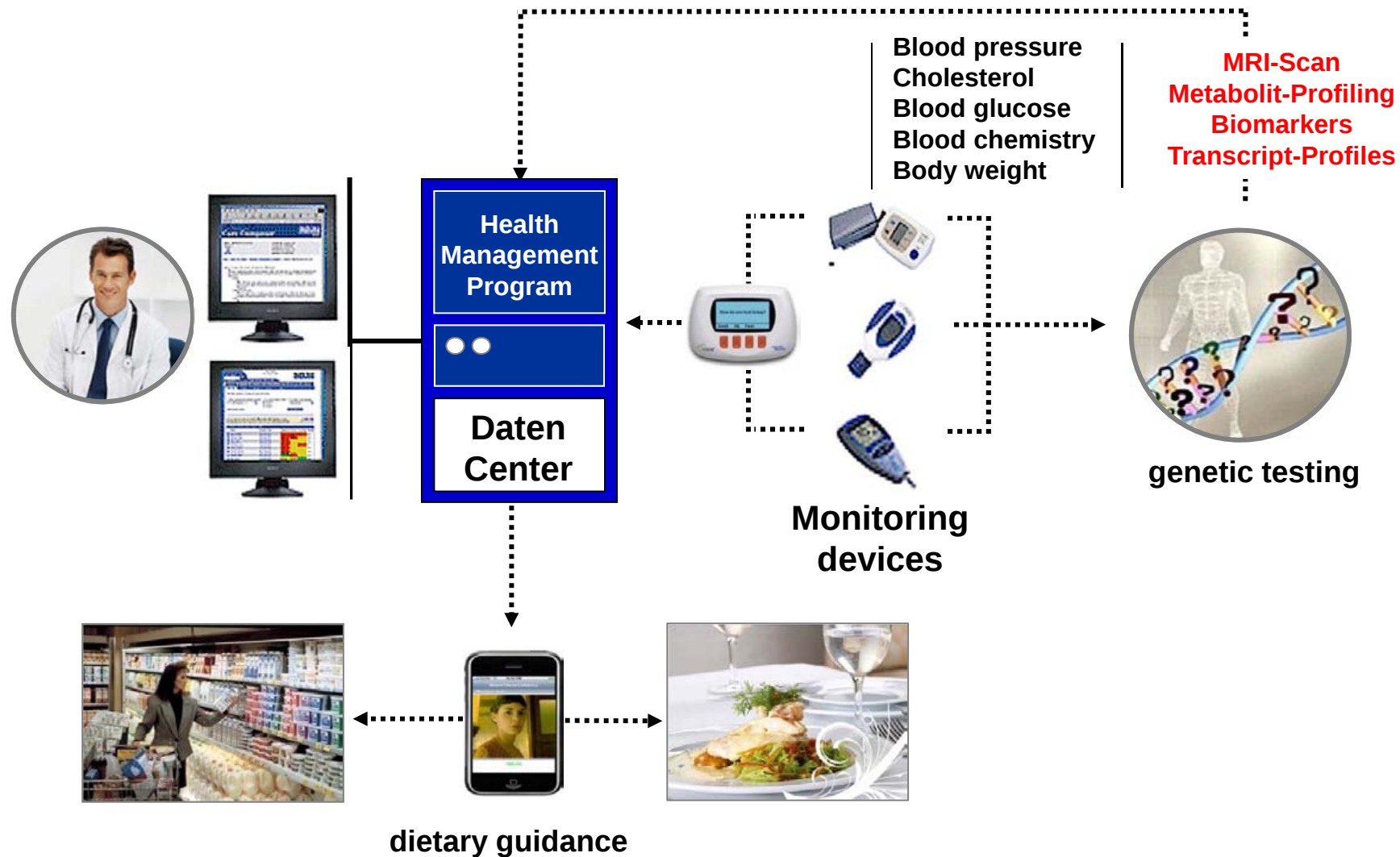


The diet and health service system





The diet and health service system





New health monitoring devices

Proteus Raisin System brings together networked pills, wearable physiologic monitoring, and mobile health for an integrated personalized medicine solution



Ingestible Technology

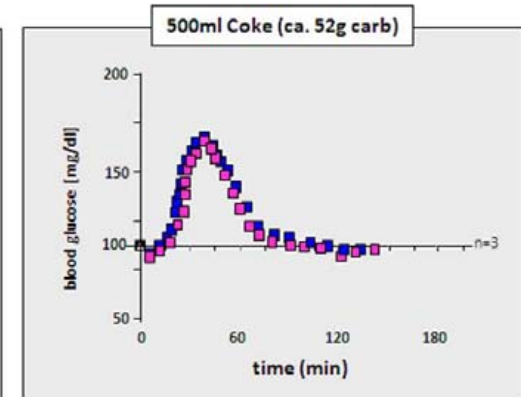
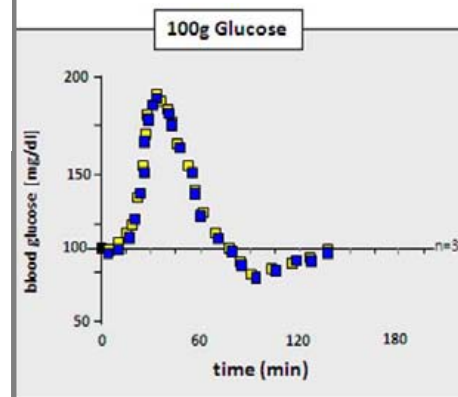


Proteus ingestible sensors

Proteus ingestible event markers (IEMs) are tiny, digestible sensors made from food ingredients, which are activated by stomach fluids after swallowing. Once activated, the IEM sends an ultra low-power, private, digital signal through the body to a microelectronic receiver that is either a small bandage style skin patch or a tiny device insert under the skin. The receiver date- and time-stamps, decodes, and records information such as the type of drug, the dose, and the place of manufacture, as well as measures and reports physiologic measures such as heart rate, activity, and respiratory rate.

The IEM is manufactured on silicon wafers, and is extremely economical to produce, costing a few cents per sensor in large quantities.

The IEM is the cornerstone of the company's Raisin™ System, which is currently in clinical development. The Raisin™ System measures the body's response to medications and is intended to improve the management of chronic diseases like heart failure, infectious disease and psychiatric disorders.



young at heart

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Company Technology Science Screenings Opportunities

get more *info*
about becoming a screening center.

see how it works

learn more

HELP THEM FIND OUT
Conduct a simple 90 second screening online

screening centers
Looking to grow revenue, drive traffic and promote wellness? Take advantage of our turn key cardiovascular screening center. We provide the technology, training and marketing to help you succeed.

in the news
Check out Young At Heart in the news.

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New health monitoring devices

(@) myFoodPhone Home About Us Product Sign Up News Testimonials

MyFoodPhone will help you to:

- Monitor what you eat
- Modify your habits
- Motivate yourself

And, best of all, it's Mobile!

VIEW TOUR
Animated presentation

Sign Up Today!
What are you waiting for? Empower yourself to lose weight. Right Now. **GO!**
Learn more about the product. **GO!**

Sign-In
Enter your username and password here.

Username: Password:

[Forgot your login?](#) **GO**

MyFoodPhone
Your Personal Nutrition Coach.

NEWS & EVENTS [New York, NY - May 12, 2005] For immediate release....
Your Cellphone Can Help You Lose Weight: myfoodphone.com

ABC (Channel 7 - New York)

VeriSign Secure Site Seal
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New health monitoring devices

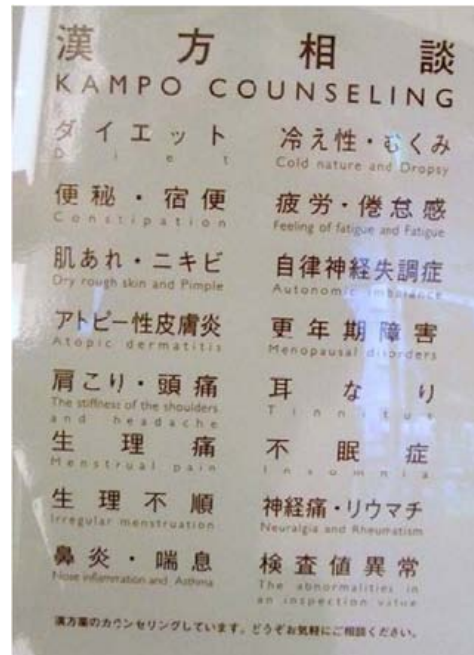


Philips Motiva is an interactive healthcare platform that connects patients with chronic conditions, e.g. (Chronic) Heart Failure, Diabetes Mellitus, and Chronic Obstructive Pulmonary Disease (COPD), to their healthcare providers – via the home television and a broadband internet connection.

<http://theonlinelearningcenter.com/shtml/motiva/motiva1/private/vnr.html>



The „new“ supermarket experience





Personalized investigation



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NATURE MEDICINE VOLUME 16 | NUMBER 9 | SEPTEMBER 2010

Despite continued doubts about the clinical utility of direct-to-consumer genetic tests, tens of thousands of people have sent away tubes full of their saliva to learn more about their genetic profiles. Armed with such DNA data, a number of early adopters are showing how empowering—and beneficial to science—personal genetic information can be. **Elie Dolgin** reports on one company's plans to make medical genetics more participatory.



Google Health

www.google.com/intl/en-US/health/about/index.html

Eurreca Google KLM Mendeley Spider Portal TNO webmail TNO-Spider TNO NuGO a NuGOWiki PubMed import Mendeley a WU bib nbx14 Andere bladwijzers

Google health

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Features

 **Manage your Health Information Online**
Is your health information in disarray? Google Health can help by offering a single place to organize and store your health information online. Track your wellness metrics, gather and organize your medical records, or import your health data directly into your account from connected doctors, hospitals and retail pharmacies. You can even create multiple profiles for family members or others you care for.

 **Set Personal Health Goals**
Do you want a better way to track your goals for weight, blood pressure, or other wellness metrics? Have you ever wanted to track your sleep patterns, record how much you walk during the day, or track your progress at the gym? Or are you trying to overcome a health problem or caring for someone who is? With Google Health you can set personalized goals online and monitor them regularly.

 **Track your Progress**
Create custom trackers for things you want to monitor like daily like sleep, how much coffee you drink a day, or how many times you exercise a week. You can also take notes or keep a diary on your how you are doing with a particular medical condition or a personal goal you set.

 **Share your Health Information**
Help better coordinate your care by sharing your health records with individuals in your care network - family members, friends and doctors. Share your wellness goals and progress notes with family and friends. You can stop sharing at any time and you will always be able to see who has access to your information. You can also print a wallet-sized version of your health profile to share with your doctor or family members in person.

 **Personalize your Health Needs with Content and Apps/Devices**
Access content about health topics from trusted sources and Google search results. You can also connect to apps that are integrated with Google Health that will help you better manage your health needs using our partner directory.

start suvimax 2 Windows ... Microsoft Pow... Herberg et al... Google Health... 18:17



← → ↺ 🏠 diygenomics.pbworks.com

📄 Eurreca 🌐 Google 📄 KLM 📄 Mendeley 📄 Spider Portal 📄 TNO webmail 📄 TNO-Spider 📄 TNO 📄 NuGO 📄 a NuGOwiki 📄 PubMed 📄 Import Mendeley 📄

Wiki Pages & Files

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FrontPage

last edited by 🧑 Melanie Swan 3 days ago 🕒 Page history

DIYgenomics - crowd-sourced clinical trials

Back to DIYgenomics.org
Contact [@DIYgenomics](https://twitter.com/DIYgenomics) or email "studies at DIYgenomics.org" for more information or to participate.

[OPEN-SOURCE SOFTWARE COMMUNITY](#) - [FUNDRAISING](#) - [HELP NEEDED](#) - [GENOMIC RESEARCH RESOURCES](#) - [PUBLISHING](#)

DIYgenomics citizen science experiments: Replicate, extend, and develop new studies linking genotype with phenotype/behavior/environment in peer cohorts. Studies may involve a combination of genomic data + phenotypic biomarker data + self-reported observational data + environmental data.

Disease Studies

1. [MTHFR Study: MTHFR mutation purportedly leading to Vitamin B deficiency and higher homocysteine levels](#) (in process, began 6/11/10)
2. [Aging Study](#): apply existing [GWAS](#) to peer cohort data for top ten areas of aging, measure corresponding phenotypic markers of aging, and attempt to ameliorate with interventions
 - Top ~ten biological mechanisms of aging in GWAS: neurodegenerative disease, osteoporosis, IGF-1/Insulin signaling, lipoprotein metabolism, inflammation, immune system function, DNA damage repair, telomere length, transcription (ex: [RNA editing](#)), catabolism, mitochondrial health, cell cycle/stem cell health, protein function, blood operations
 - Top ~ten phenotypic biomarkers of aging: blood pressure and hypertension, cholesterol (HDL/LDL/triglycerides; LDL particle size), BMI, Framingham Risk Score, VO2 max, erythrocyte glycosylation, telomere length, lymphocyte growth capability, granulocyte strength
 - Example: effect of CoQ10 deficiency ([paper](#))
3. Cholesterol management - link genes with physical biomarkers, test the efficacy of supplements ([niacin](#), etc.) and other remedies. References: [Ron Krauss](#) (DIYbio lab test: [Cholestech LDX](#))
4. Type 2 Diabetes/Obesity: apply novel loci/variants from Cristen Willer's recent work ([Nature Genetics paper](#))
5. Apply 160 variants implicated in cardiovascular disease to the peer cohort (Arking, Trends Genet 2009, [paper](#))
6. [Macular degeneration](#) study - examine variants, protective variants, and efficacy of supplementation products, participate in [MacuCLEAR](#) small molecule remedy clinical trials and explore other [new drugs](#)



Hercberg *et al. BMC Public Health* 2010, **10**:242
<http://www.biomedcentral.com/1471-2458/10/242>



STUDY PROTOCOL

Open Access

The Nutrinet-Santé Study: a web-based prospective study on the relationship between nutrition and health and determinants of dietary patterns and nutritional status

Serge Hercberg^{*1,2,3}, Katia Castetbon², Sébastien Czernichow^{1,3}, Aurélie Malon¹, Caroline Mejean¹, Emmanuelle Kesse¹, Mathilde Touvier¹ and Pilar Galan¹

Methods/design: Our web-based prospective cohort study is being conducted for a scheduled follow-up of 10 years. Using a dedicated web site, recruitment will be carried out for 5 years so as to register 500 000 volunteers aged ≥ 18 years among whom 60% are expected to be included (having complete baseline data) and followed-up for at least 5 years for 240 000 participants. Questionnaires administered via internet at baseline and each year thereafter will assess socio-demographic and lifestyle characteristics, anthropometry, health status, physical activity and diet. Surveillance of health events will be implemented via questionnaires on hospitalisation and use of medication, and linkage with a national database on vital statistics. Biochemical samples and clinical examination will be collected in a subsample of volunteers.



Pressemeldung: Oktober 2010

LAUSANNE. Konkret geht es um die Vorbeugung und Behandlung chronischer Krankheiten wie Diabetes, Fettleibigkeit und Alzheimer. Der Konzern gründet dazu die **NESTLÉ Health Science AG** und ein Forschungsinstitut. "Wir schaffen nicht nur ein neues Unternehmen, sondern eine ganz neue Industrie", sagte Verwaltungsratschef Peter Brabeck. Brabeck ist der geistige Vater der Strategie, die NESTLÉ vom reinen Nahrungsmittelanbieter zu einem **Gesundheits- und Wellnesskonzern** verwandeln soll. Bereits heute Erlösen die Schweizer mit gesunder Ernährung (Nutrition) mehr als zehn Mrd. Franken. Darin enthalten ist der Bereich NESTLÉ Health Care Nutrition mit einem Jahresumsatz von 1,6 Mrd. Franken, der jetzt die Basis für die neue Health Science AG bilden soll. Chef der neuen Tochter wird Luis Cantarell, der bislang das Amerika-Geschäft verantwortete. Die Vision eines Gesundheitskonzerns treibt Brabeck schon seit langem um. Er bezeichnet NESTLÉ bereits als "Life-Science-Company". Seine jüngste Initiative begründete Brabeck am Montag mit den sich verändernden wirtschaftlichen Bedingungen: Der starke Anstieg der Staatsschulden mache die Finanzierung der Alters- und Gesundheitsvorsorge immer schwieriger. Umso wichtiger sei es deshalb, chronischen Erkrankungen mit Gesundheitsprodukten vorzubeugen.



Was ist das Forschungsziel der nächsten 20 Jahre ?

Die umfassende Geno- und Phänotypisierung des Menschen im Kontext seiner Ernährung !

- *durch Einsatz der modernen Life Science Technologien*
- *durch Einsatz von nicht-invasiven bildgebenden Verfahren*
- *durch komplette Sequenzierung des jeweiligen Genoms*
- *durch gezielte Ernährungsstudien unter kontrollierten Bedingungen*
- *durch kybernetische (mathematische) Beschreibung des Stoffwechsels*