

Epigenetik und Mikrobiota, Marker in der personalisierten Ernährung

S. Lilja, B. Hippe, A Pointner, Haslberger, A.G.
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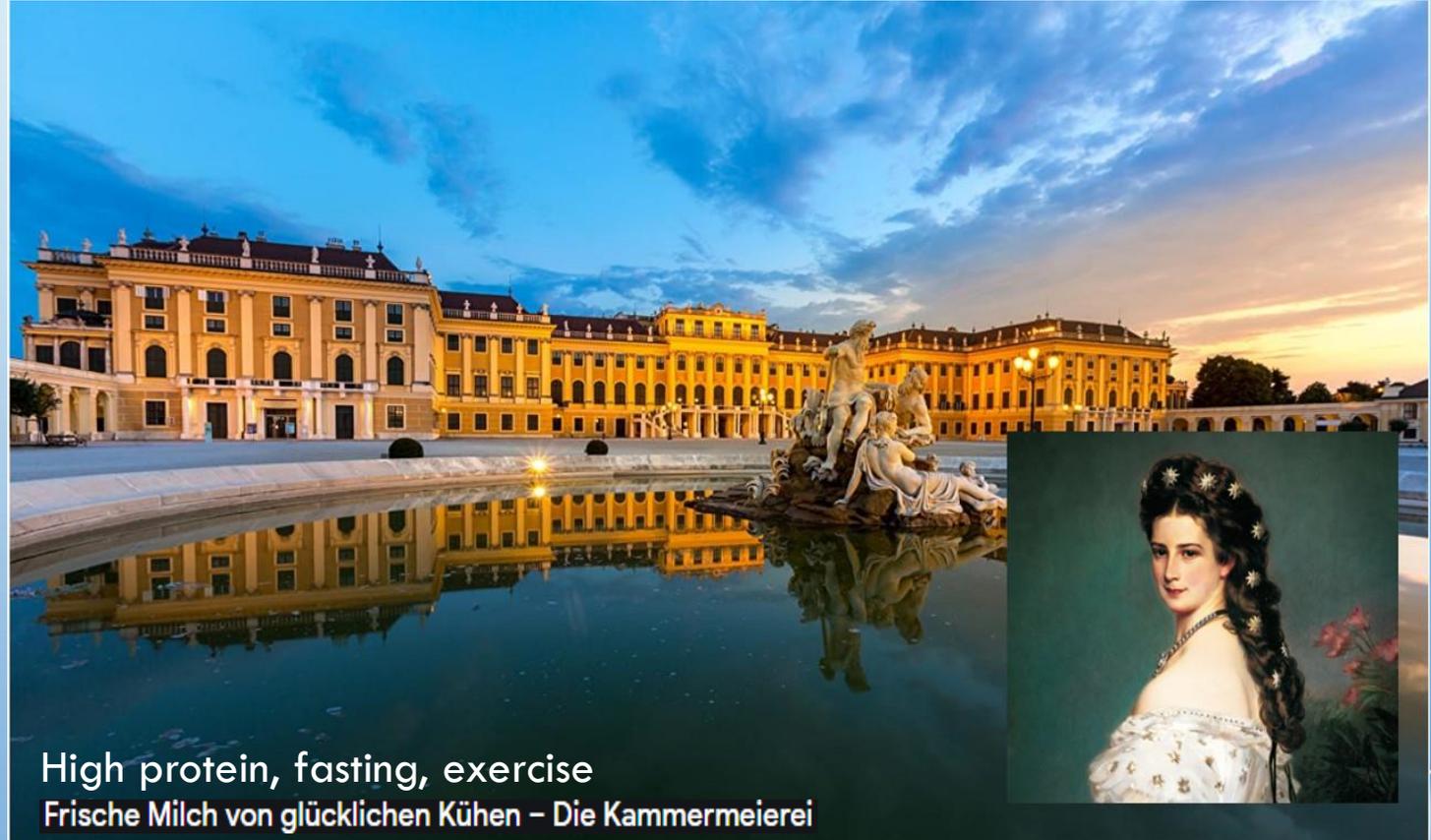


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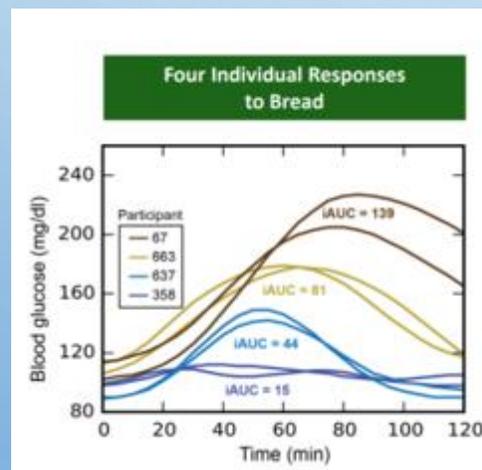
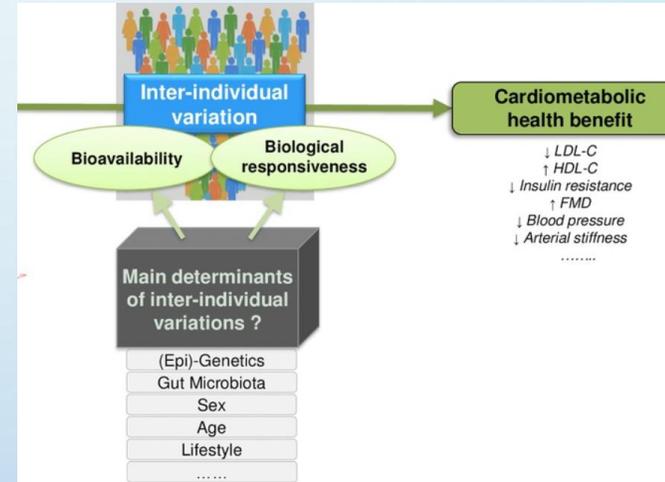
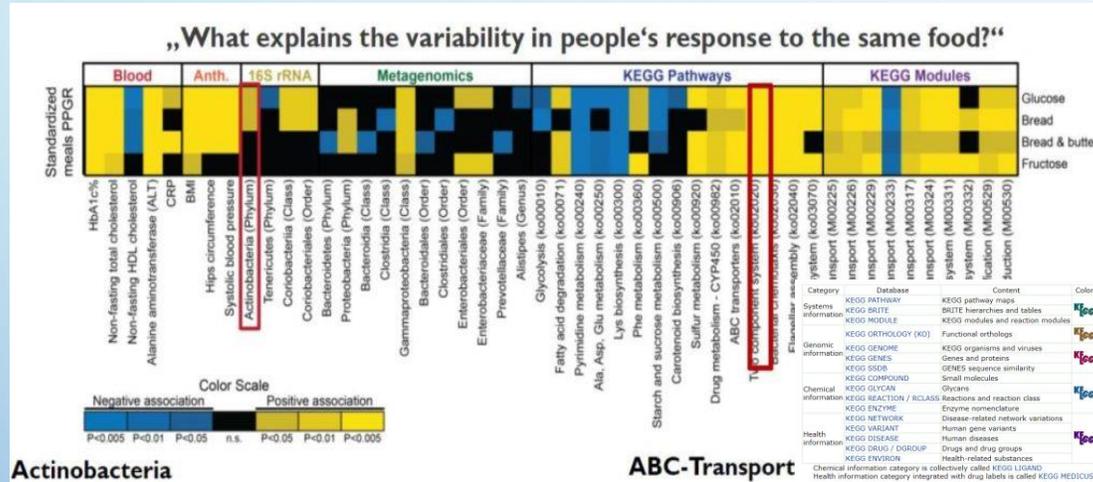
PERSONALISIERTE ERNÄHRUNG
Anwendungsreife auf dem Prüfstand

5. Oktober 2021, Bildungscampus Heilbronn



High protein, fasting, exercise
Frische Milch von glücklichen Kühen – Die Kammermeierei

SCIENCE: HIGHLY DIFFERENT PERSONAL RESPONSES TO DIETS, EG POST-PRANDIAL GLYCEMIC RESPONSES, EXPLANATIONS ?



Different people have different, opposite responses to standardized meal, bread, Zeevi et al., 2015, Cell

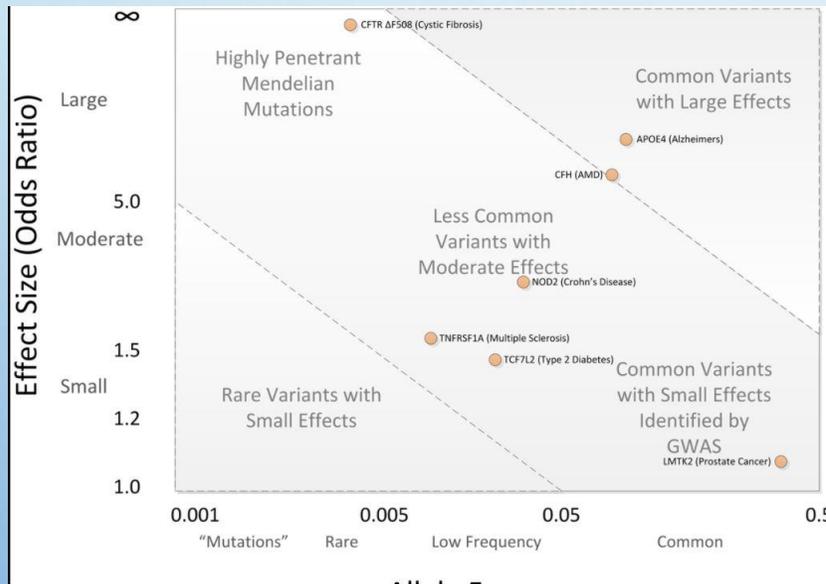
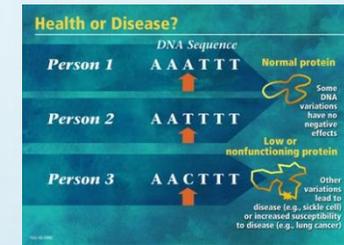
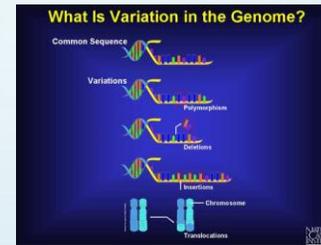
1600557 (1 of 16) DOI: 10.1002/rob.2048807 Med. Nutr. Food Res. 61, 6, 2015, 1600557

Review

Addressing the inter-individual variation in response to consumption of plant food bioactives: Towards a better understanding of their role in healthy aging and cardiometabolic risk reduction

Claudine Manach¹, Dragan Milenkovic¹, Tom Van de Wiele², Ana Rodriguez-Mateos³, Beáta de Rooij⁴, Maria Teresa Garcia-Conesa⁵, Rikard Landberg^{1,6}, Eileen R. Gibney⁷, Marina Hejblum⁸, Francisco Tomas-Barberan⁹ and Christine Morand¹

GWAS : SNPS, COMMON VARIANTS HAVE OFTEN ONLY MODERATE EFFECTS; IN DIFFERENT METABOLIC AREAS

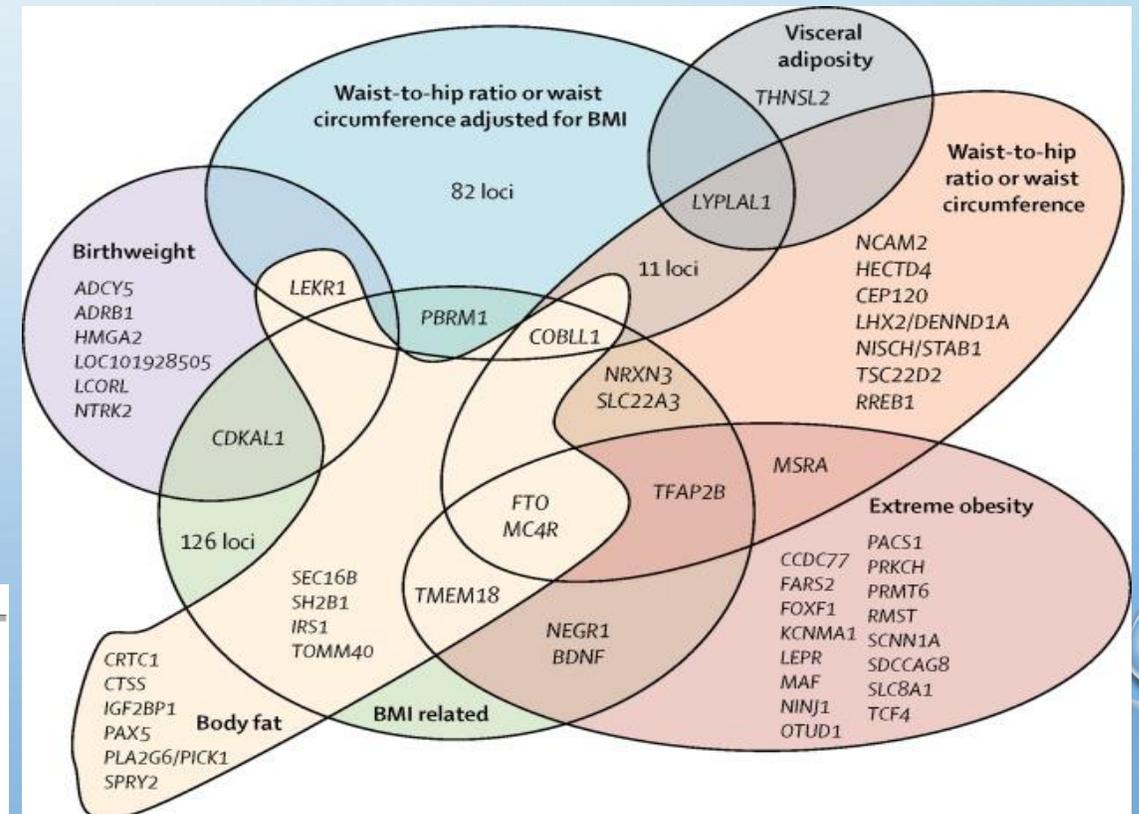


Methods

Prediction of individual genetic risk to disease from genome-wide association studies

Naomi R. Wray,^{1,4} Michael E. Goddard,^{2,3} and Peter M. Visscher¹

¹Genetic Epidemiology, Queensland Institute of Medical Research, Queensland 4029, Brisbane, Australia; ²Faculty of Land and Food Resources, University of Melbourne, Victoria 3010, Australia; ³Department of Primary Industries, Victoria 3049, Australia



MISSING HERITABILITY: WHAT IS MISSING TO UNDERSTAND A PHENOTYPE: GENE- ENVIRONMENT INTERACTIONS, EPIGENETICS, REVERSIBILITY

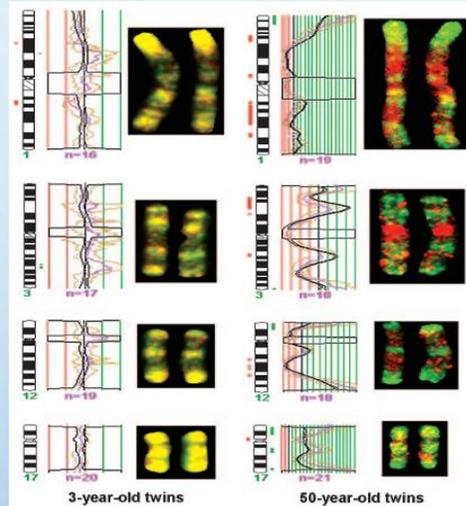
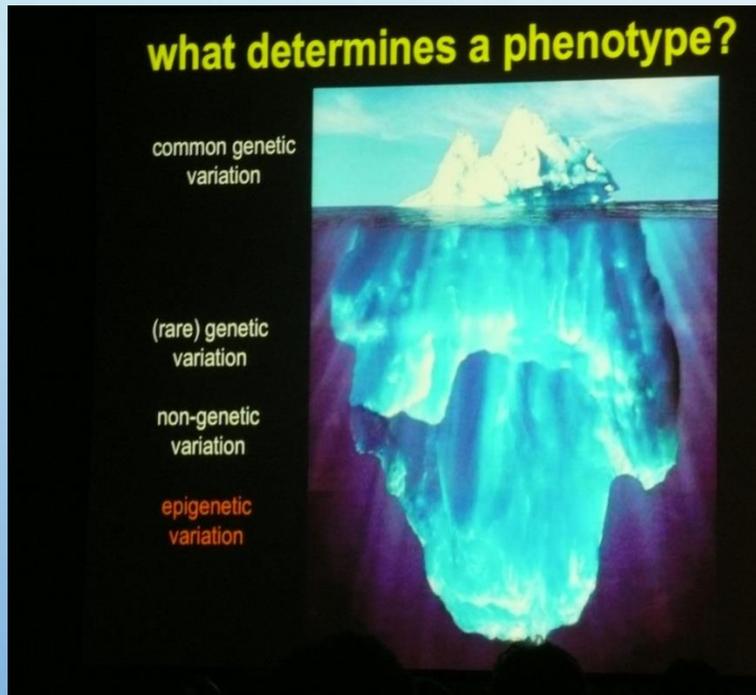


Fig. 3. Mapping chromosomal regions with differential DNA methylation in MZ twins by using comparative genomic hybridization for methylated DNA. Competitive hybridization onto normal metaphase chromosomes of the AIMS products generated from 3- and 50-year-old twin pairs. Examples of the hybridization of chromosomes 1, 3, 12, and 17 are displayed. The 50-year-old twin pair shows abundant changes in the pattern of DNA methylation observed by the presence of green and red signals that indicate hypermethylation and hypomethylation events, whereas the 3-year-old twins have a very similar distribution of DNA methylation indicated by the presence of the yellow color obtained by equal amounts of the green and red dyes. Significant DNA methylation changes are indicated as thick red and green blocks in the ideograms.

2003: Randy L Jirtle

Pregnant Agouti viable yellow (A^{vy}) mice were fed a diet high in methyl donors.

- folic acid
- choline
- vitamin B₁₂
- betaine

High Me Diet

Low Me Diet

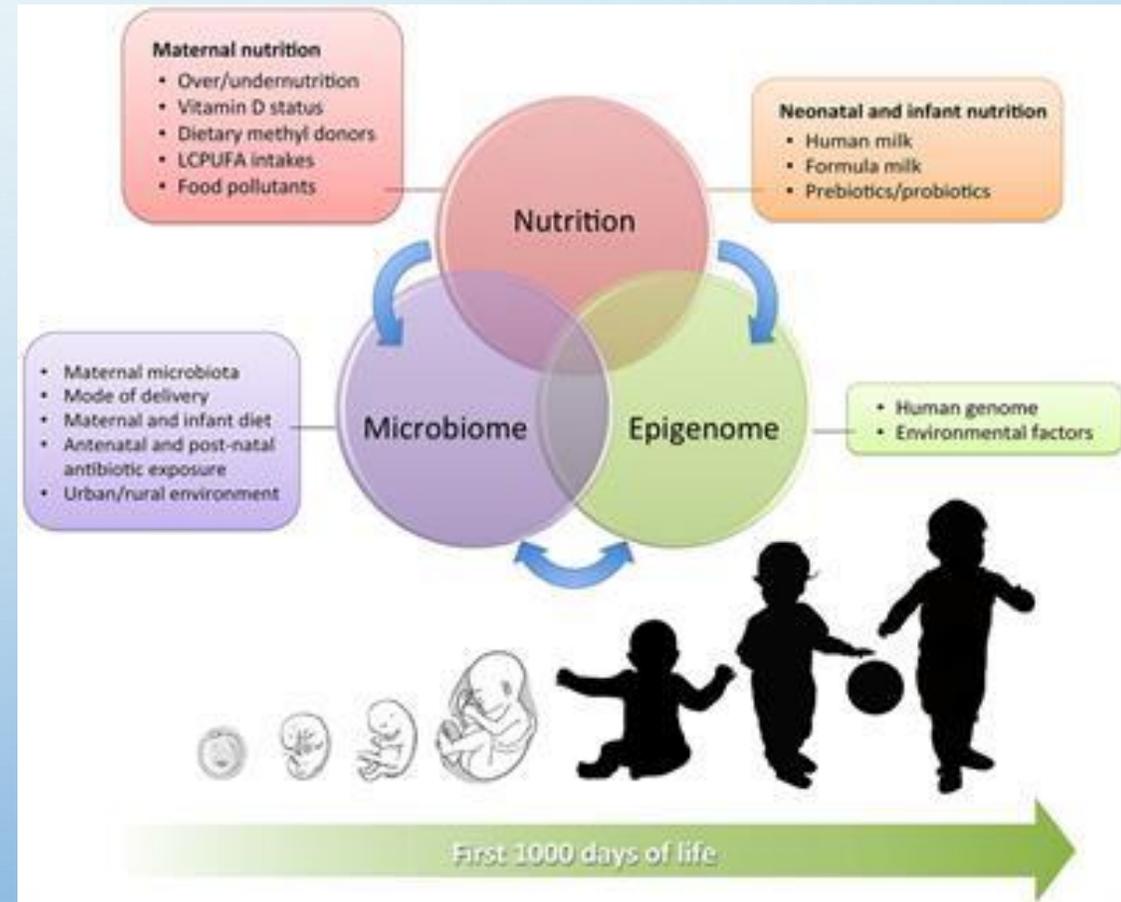
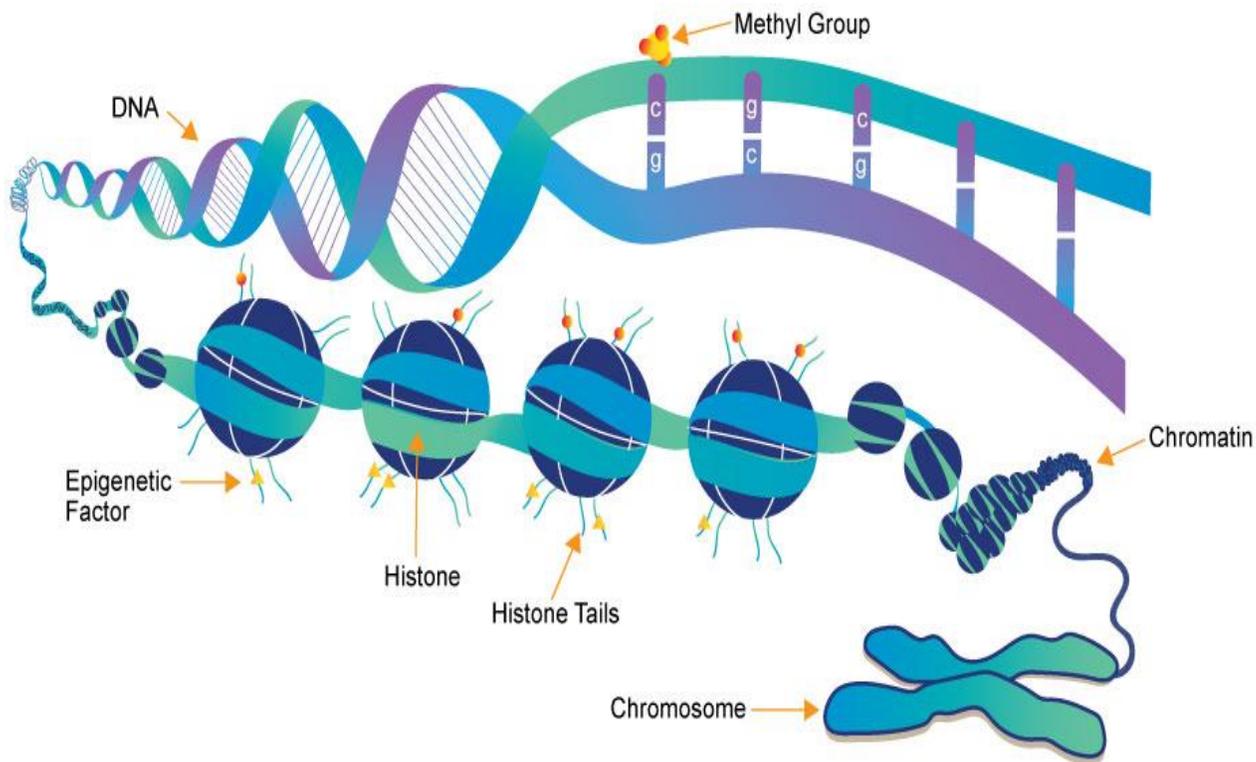
Source: randyjirtle.com, 2014

Epigenetic differences arise during the lifetime of monozygotic twins

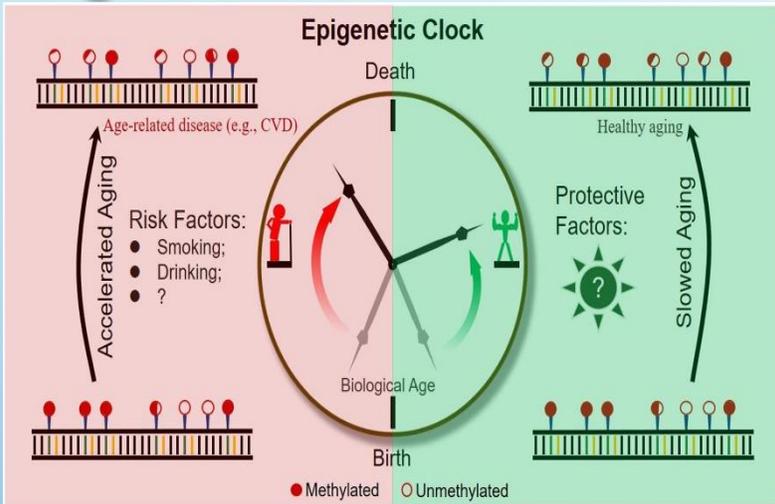
Mario F. Fraga*, Esteban Balestar*, Maria F. Paz*, Santiago Ropero*, Fernando Setien*, Maria L. Balestar*, Daniela Heine-Salfer*, Juan C. Cigudosa*, Miguel Urzeta*, Javier Benitez*, Manuel Estro-Chironet*, Abel Sanchez-Aguilera*, Charlotte Ling*, Emma Carlson*, Pernille Poulsen**, Allan Vaag**, Zarko Stefanac*, Tim D. Spector*, Yue-Zhong Wu**, Christoph Plau**, and Marel Esteller^{†‡§}



EPIGENETICS MECHANISMS, INTERACTIONS, EARLY IMPRINTING



CPG METHYLATION, EPIGENETIC CLOCK, REFLECT C.R., NUTRITION



Intrinsic age: Horvath
multiple tissues.
Extrinsic Hannum,
blood cell

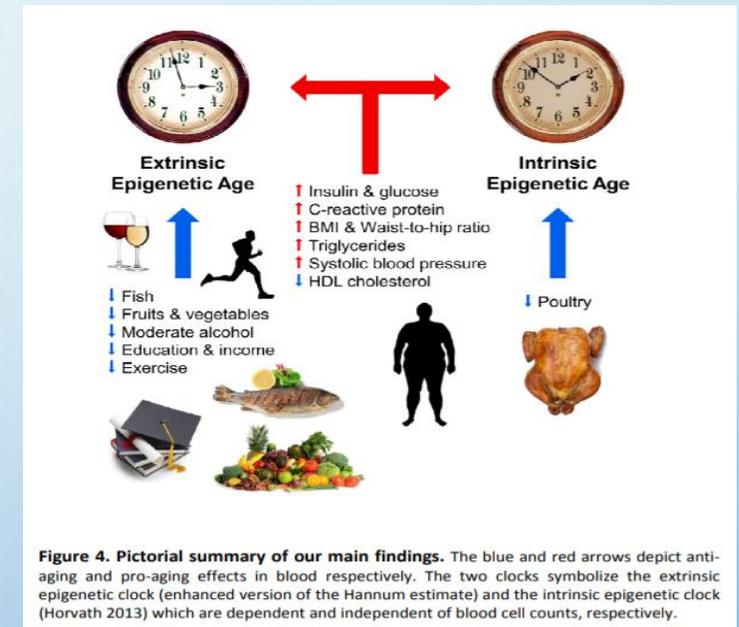
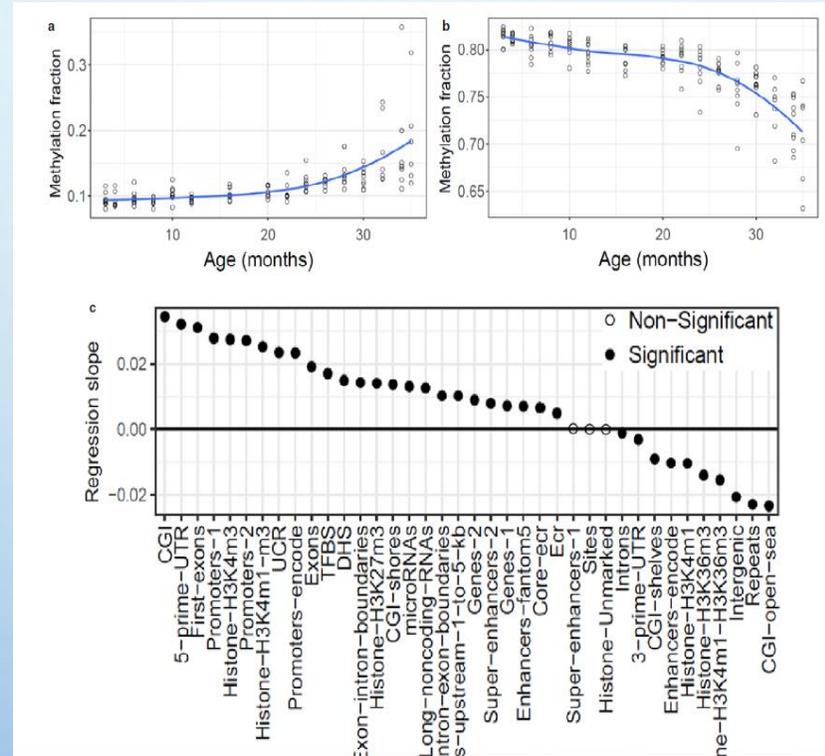


Figure 4. Pictorial summary of our main findings. The blue and red arrows depict anti-aging and pro-aging effects in blood respectively. The two clocks symbolize the extrinsic epigenetic clock (enhanced version of the Hannum estimate) and the intrinsic epigenetic clock (Horvath 2013) which are dependent and independent of blood cell counts, respectively.

www.aging-us.com

AGING 2017, Vol. 9, No. 2

Research Paper

Epigenetic clock analysis of diet, exercise, education, and lifestyle factors

Austin Quach^{1*}, Morgan E. Levine^{1*}, Toshiko Tanaka^{2*}, Ake T. Lu¹, Brian H. Chen², Luigi Ferrucci³, Beate Ritz^{4,5}, Stefania Bandinelli⁶, Marian L. Neuhouser⁷, Jeannette M. Beasley⁷, Linda Snetselaar⁸, Robert B. Wallace⁹, Philip S. Tsao^{10,11}, Devin Absher¹², Themistocles L. Assimes¹³, James D. Stewart¹⁴, Yun Li^{15,16}, Lifang Hou^{15,16}, Andrea A. Baccarelli¹⁷, Eric A. Whitset^{12,18}, Steve Horvath¹⁹

International Journal of
Molecular Sciences

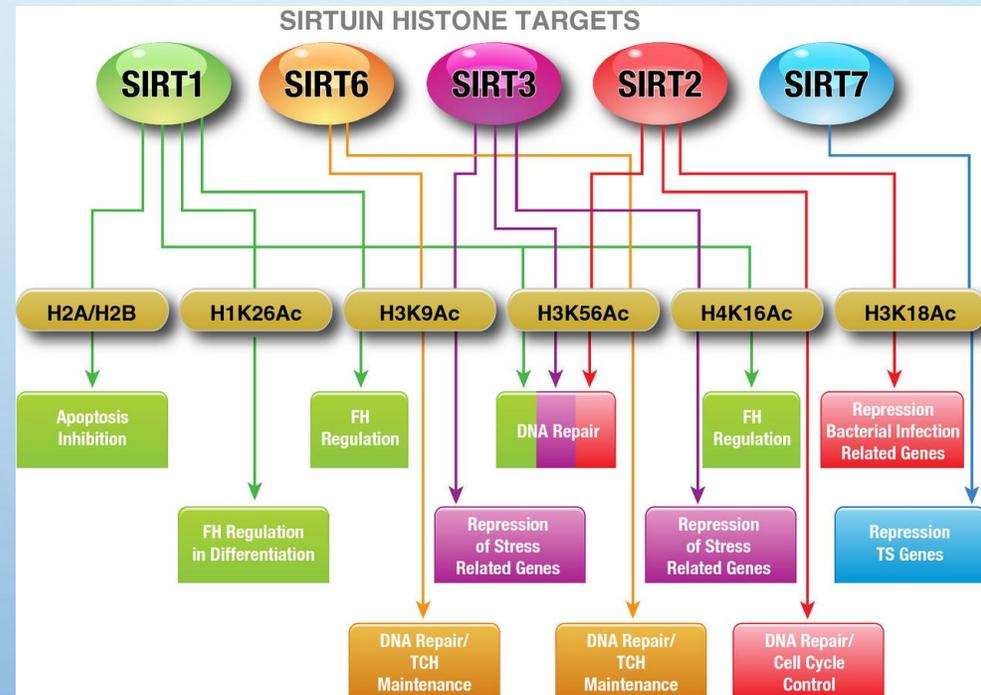
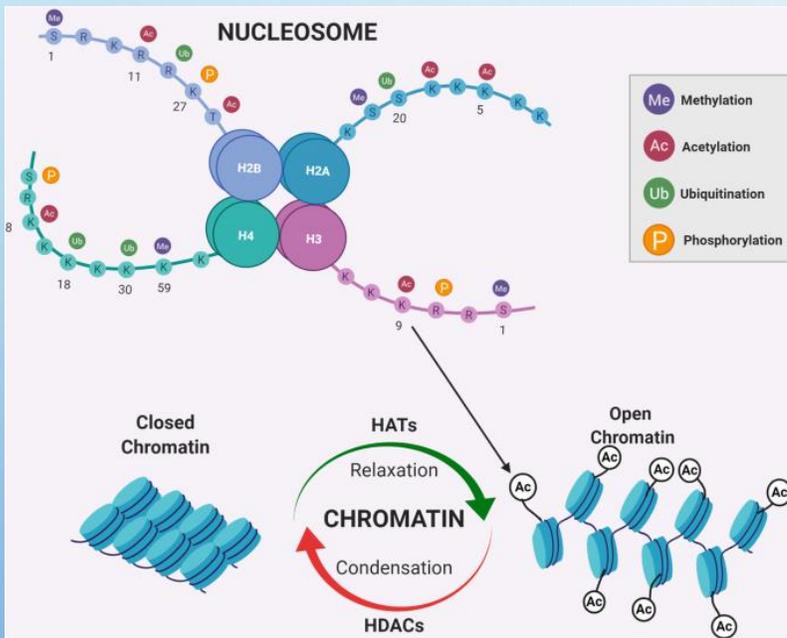
MDPI

Review

The Impact of Caloric Restriction on the Epigenetic Signatures of Aging

Noémie Gensous¹, Claudio Franceschi^{2,3}, Aurelia Santoro¹, Maddalena Milazzo¹, Paolo Garagnani^{1,4,5,6,7,*} and Maria Giulia Bacalini²

NUTRITION: CENTRAL IMPORTANCE EPIGENETIC HISTONE-MEDIATED REGULATION: E.G. C.R. REGULATE SIRTS, (HDACS; DO ALL BENEFIT FROM A SIRT DIET ?



EPIGENETIC MIRNAS: FOOD BORNE AND REGULATORS AND MARKERS OF METABOLIC MECHANISMS, PHENOTYPES, DISORDERS

PLOS ONE

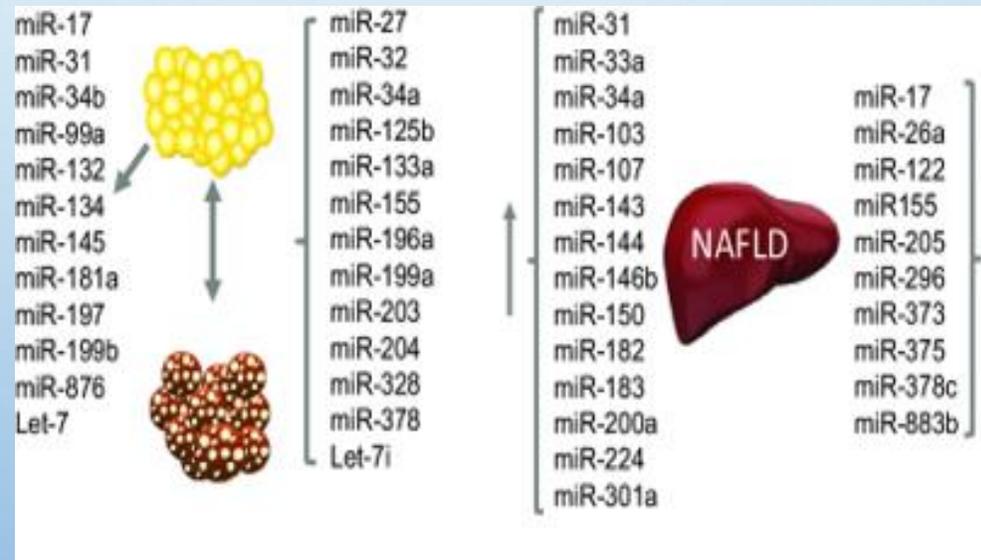
RESEARCH ARTICLE

Dietary MicroRNA Database (DMD): An Archive Database and Analytic Tool for Food-Borne microRNAs

Kevin Chiang¹, Jiang Shu¹, Janos Zemleni², Juan Cui^{1*}

¹ Department of Computer Science and Engineering, University of Nebraska-Lincoln, Lincoln, NE, United States of America, ² Department of Nutrition and Health Sciences, University of Nebraska-Lincoln, Lincoln, NE, United States of America

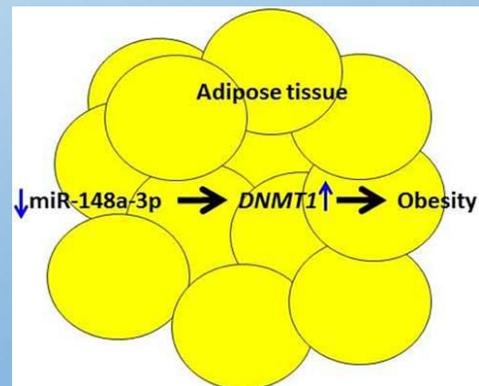
* jcui@unl.edu



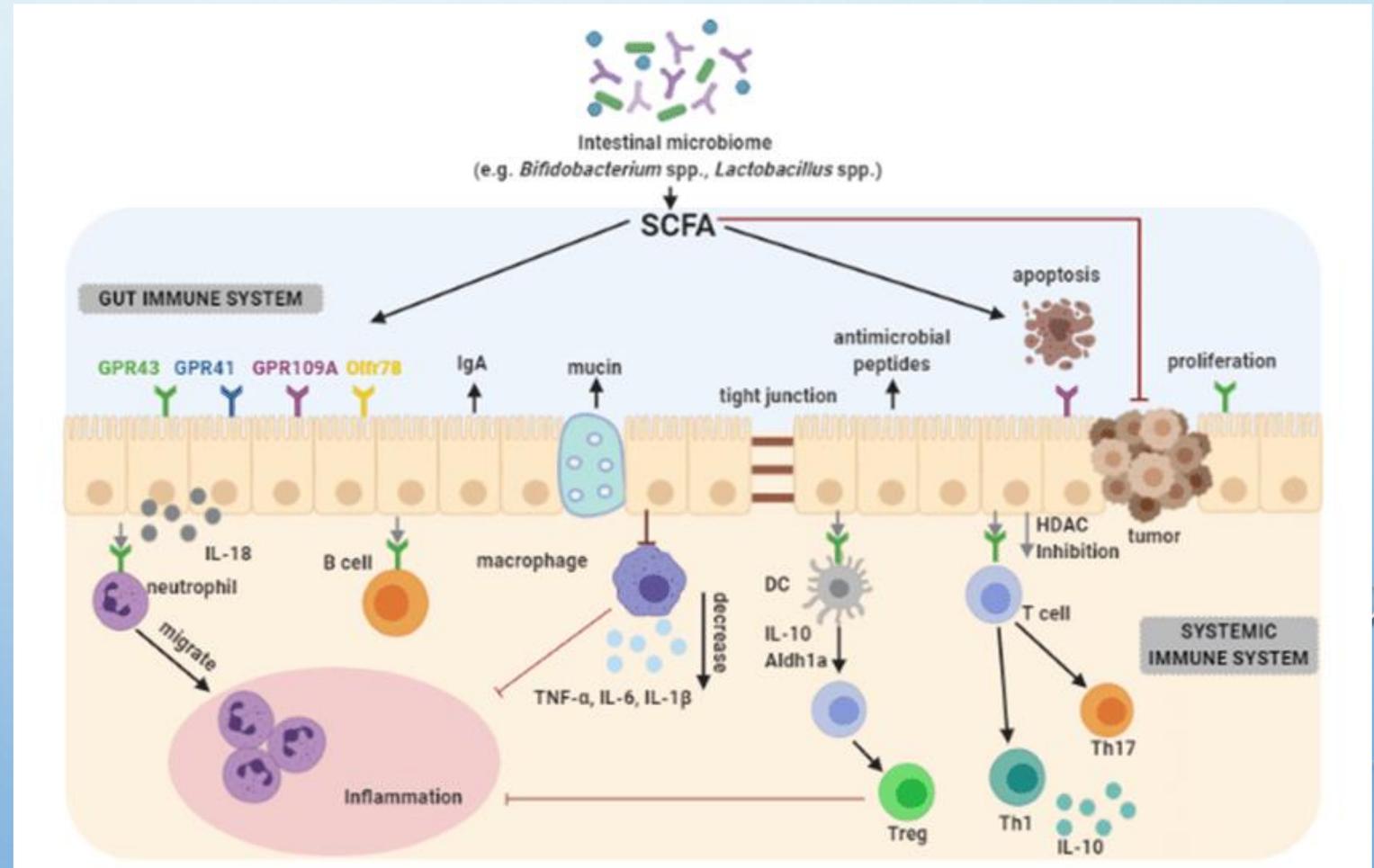
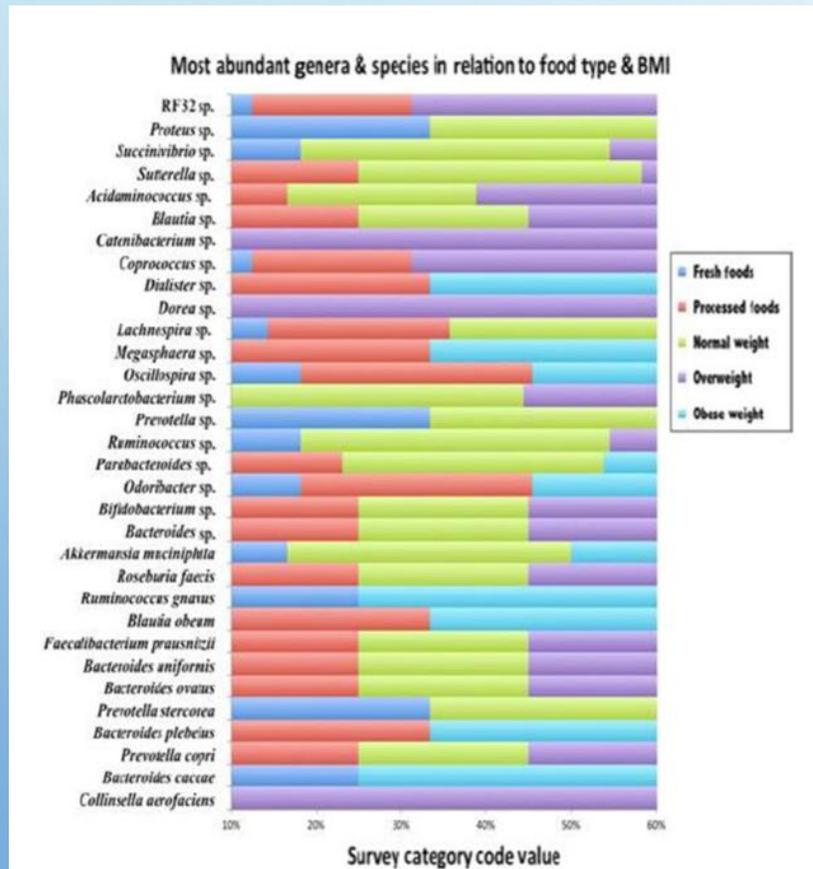
Mammalian Genome (2020) 31:134–145
<https://doi.org/10.1007/s00335-020-09835-3>

Epigenetic contribution to obesity

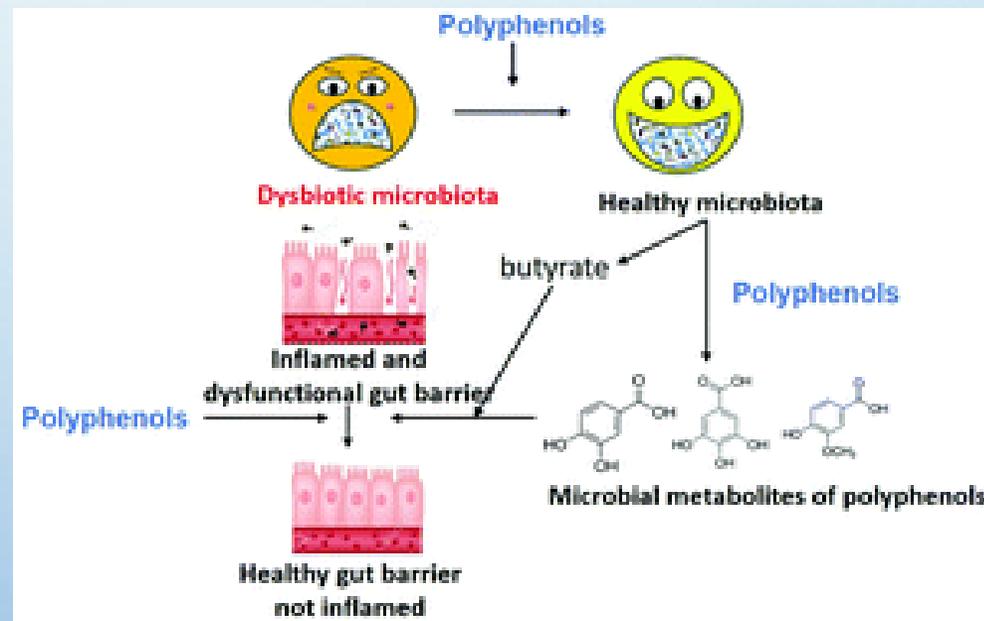
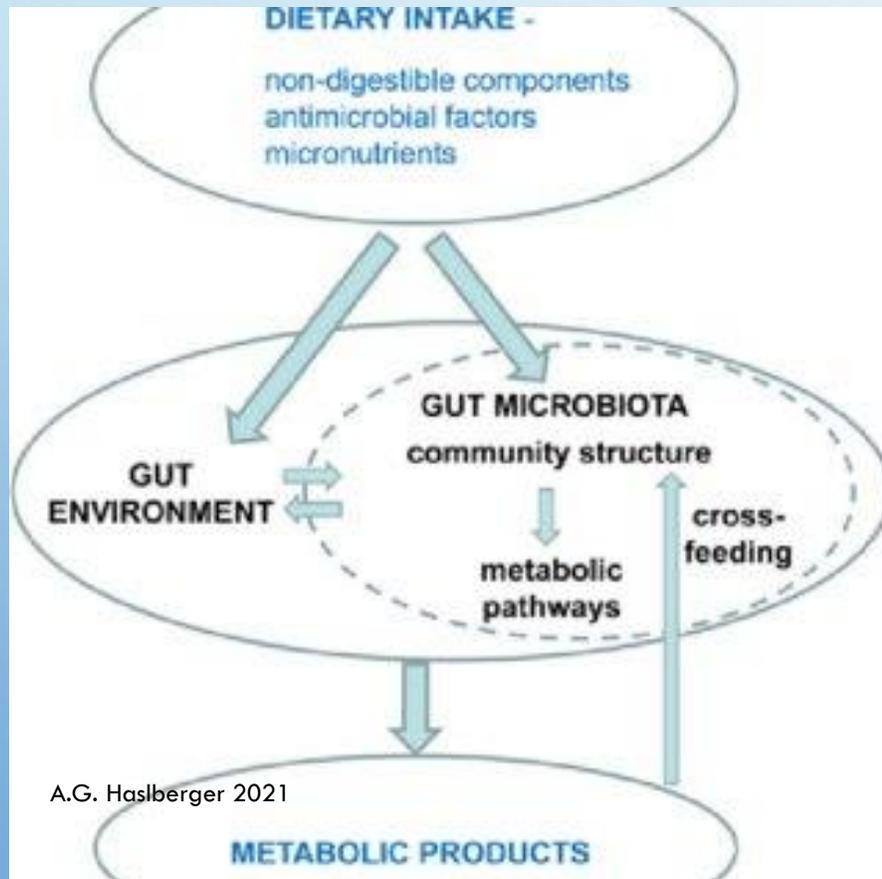
Meriem Ouni^{1,2} · Annette Schürmann^{1,2,3}



HIGH INDIVIDUAL DIVERSITY OF GUT MICROBIOTA REFLECTS NUTRITION AND LIFESTYLE , RESULTS IN DIFFERENT EXPRESSION OF METABOLITES ESP. SCFAS



HIGHLY PERSONAL DIFFERENT RESPONSES OF MICROBIOTA TO DIETS, (CROSSFEEDING) AND METABOLISATION OF FOODS



Article | Open Access | Published: 12 March 2018

Understanding the prebiotic potential of different dietary fibers using an *in vitro* continuous adult fermentation model (PolyFermS)

Sophie A. Poeker, Annelies Geirnaert, Laura Berchtold, Anna Greppi, Lukasz Krych, Robert E. Steinert, Tomas de Wouters & Christophe Lacroix

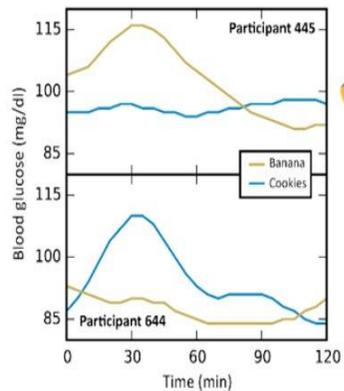
Scientific Reports 8, Article number: 4318 (2018) | Cite this article

H. Flint

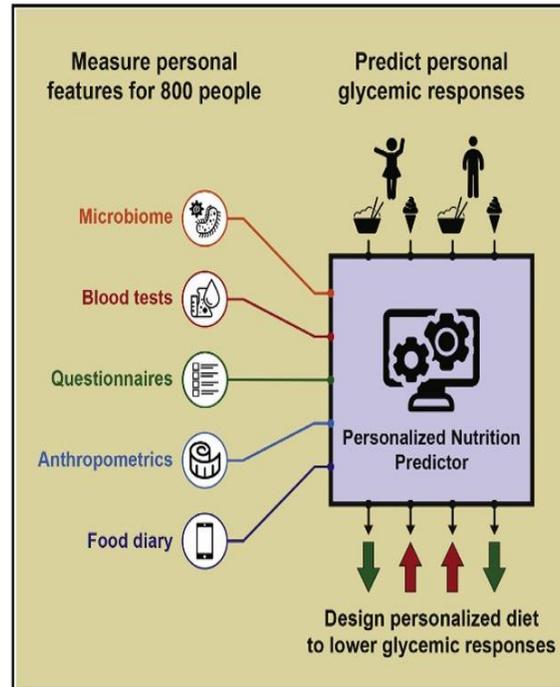
CORRELATION VON MICROBIOTA STRUCTURE WITH GLYCEMIC RESPONSES USED FOR ALGORITHMS FOR DIETARY ADVICE

Beispiel - Personalized Nutrition by Prediction of Glycemic Responses David Zeevi, 2016

- 800 Personen – jeder hat andere „post meal Glucose response“

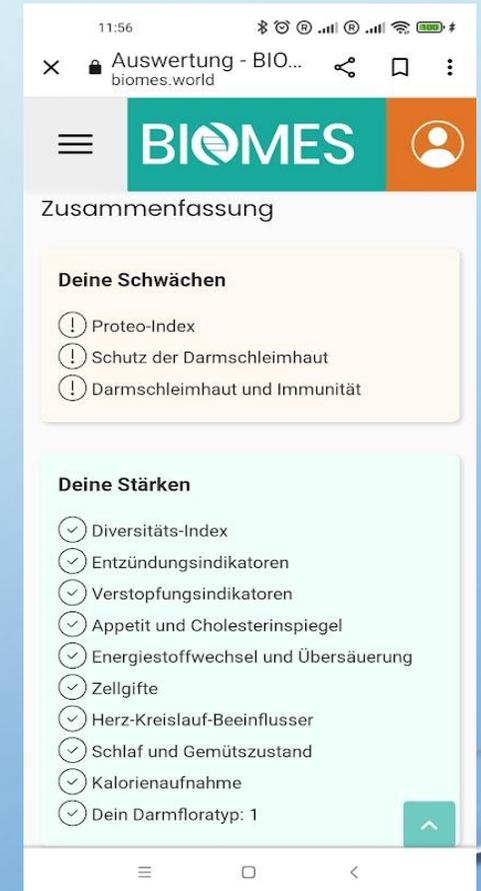


Mikrobiota Zusammensetzung beeinflusst Blutglucoselevel



Eran Elinav and Eran Segal, Weizmann Institute of monitoring the blood sugar, diets, and other traits of 800 people, they built an algorithm that can accurately predict how a person's blood-sugar levels will spike after eating any given meal.

They also used these personalized predictions to develop tailored dietary plans for keeping blood sugar in check.



SO, GENETIC AND MICROBIOTA ANALYSIS FOR PERSONAL DIETARY PLANS, BUT OF CENTRAL IMPORTANCE ARE INTERACTIONS MICROBIOTA WITH EPIGENETIC SYSTEM; HOST GUT INTERACTIONS E.G. IN C.R., FASTING (FASTING MIMETICS)

Cell Metabolism

Functional Gut Microbiota Remodeling Contributes to the Caloric Restriction-Induced Metabolic Improvements

Graphical Abstract



Authors

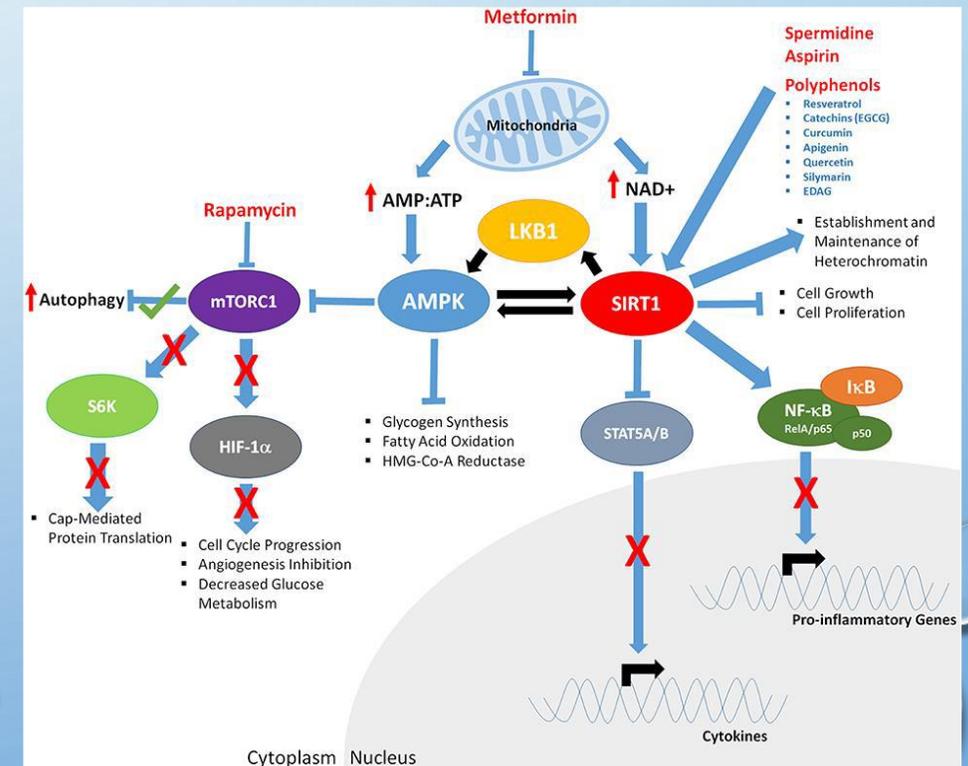
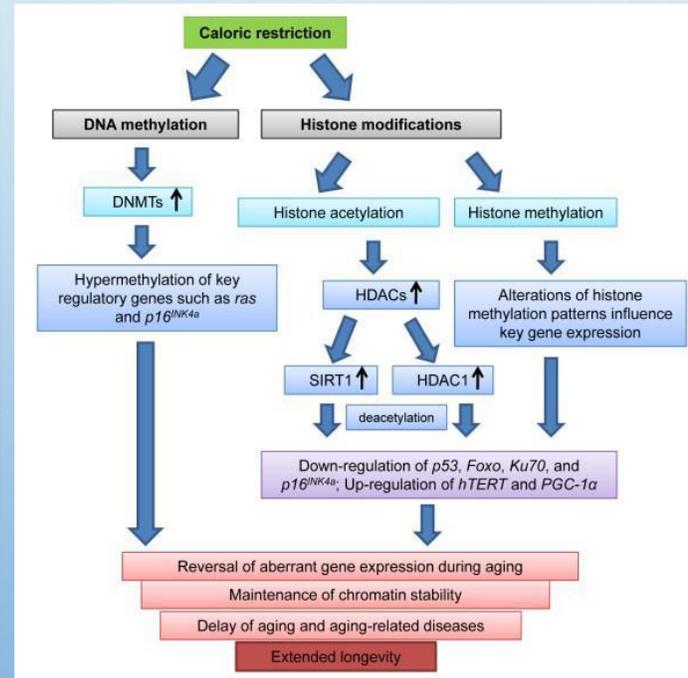
Salvatore Fabbiano, Nicolas Suárez-Zamorano, Claire Chevalier, ..., Andrew Macpherson, Jacques Schrenzel, Mirko Trajkovski

Correspondence

mirko.trajkovski@unige.ch

In Brief

Fabbiano et al. show that gut microbiota remodeling is important for the metabolic improvements associated with caloric restriction, including fat browning and improved glycemic control. They link the systemic beneficial metabolic effects to reduced endotoxin production, leading to increased type 2 immune response in the adipose tissue.



CASE STUDY: COMPARING FASTING AND A FASTING MIMETIC SIRT-FOOD SHOT: MICROBIOTA, EPIGENETICS



Buchinger Fasting < 120 kcal/day
n: 22 in Pernegg Monastery

Feces , blood spots, before and
After the end, first solid feces



STOFF	WIRKSTOFF	MENGE / 25ML	Wirkstoff
Blueberry Extract	Anthocyanins/ Anthocyanidin..	40 mg	14mg 10mg
Broccoli Extract	Sulpharapane, Glucoraphin..	30 mg	
Apfel extract	Phlorentin, Quercetin..	50 mg	
Citrus extract	Naringin..	40 mg	
Nikotinamid	Nikotinamid ribosid	24 mg	
Zinkgluconat	Zink	7.5 mg	

Wasser, Stevia, Erythrit

Active (N. 131) Placebo (n: 30)
Intervention 3 months

Feces, Blood spots before, after 1,3 month

BUCHINGER FASTING RESULTED IN A RISE IN THE DISTRIBUTION OF PROTEOBACTERIA, INCREASED MICROBIOTA DIVERSITY AND A SIGNIFICANT INCREASE IN CHRISTENSENELLA

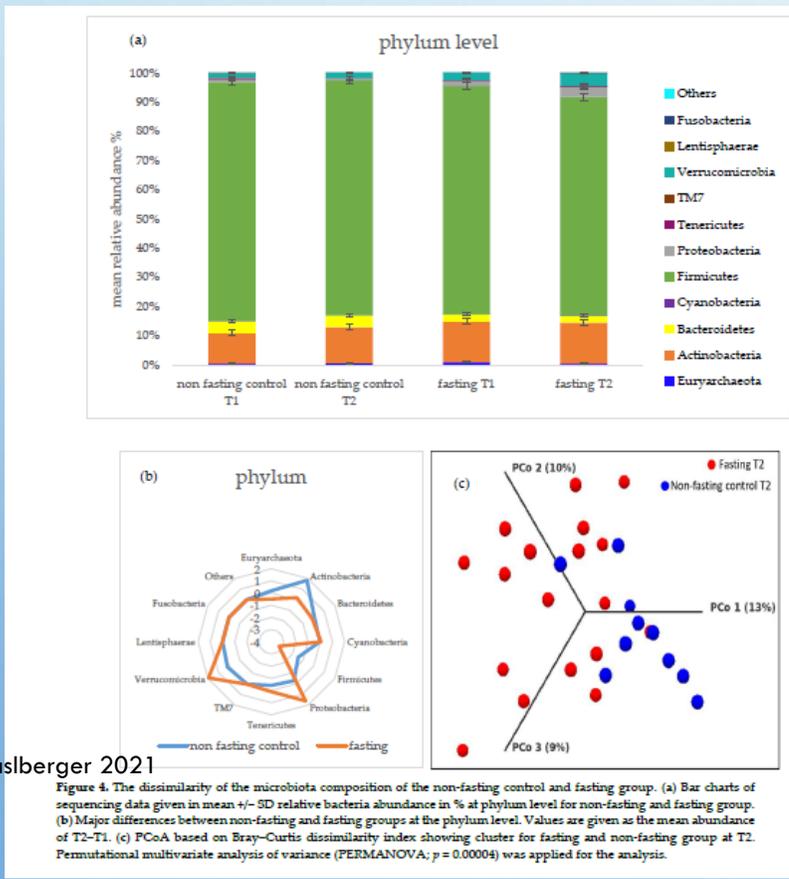


Figure 4. The dissimilarity of the microbiota composition of the non-fasting control and fasting group. (a) Bar charts of sequencing data given in mean \pm SD relative bacteria abundance in % at phylum level for non-fasting and fasting group. (b) Major differences between non-fasting and fasting groups at the phylum level. Values are given as the mean abundance of T2-T1. (c) PCoA based on Bray-Curtis dissimilarity index showing cluster for fasting and non-fasting group at T2. Permutational multivariate analysis of variance (PERMANOVA, $p = 0.00004$) was applied for the analysis.

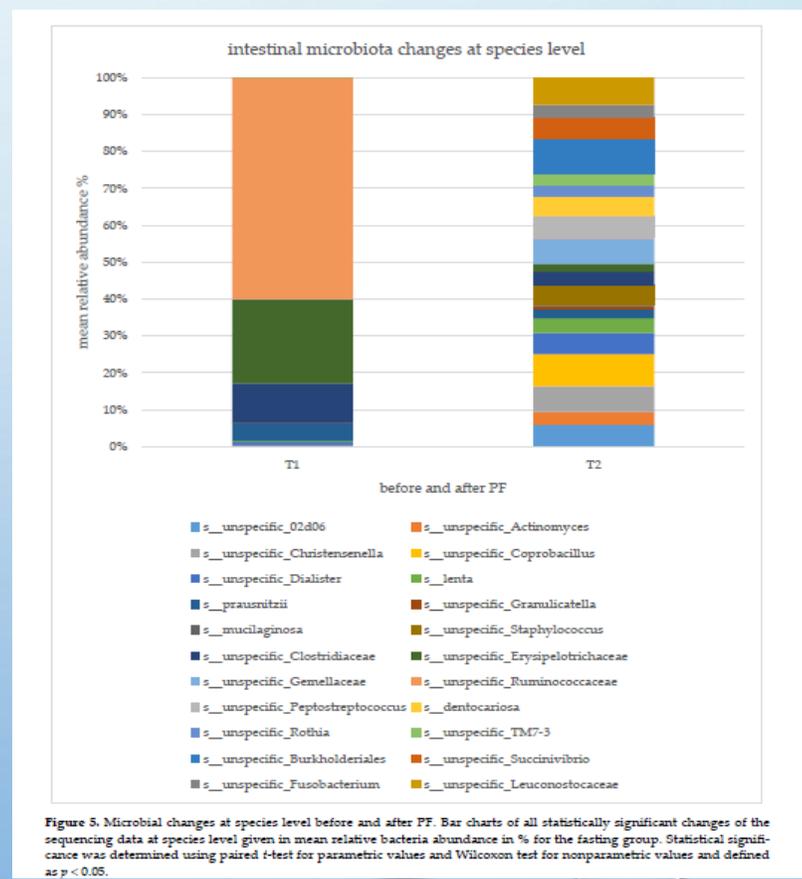


Figure 5. Microbial changes at species level before and after PF. Bar charts of all statistically significant changes of the sequencing data at species level given in mean relative bacteria abundance in % for the fasting group. Statistical significance was determined using paired t-test for parametric values and Wilcoxon test for nonparametric values and defined as $p < 0.05$.

3M SIRT INDUCING DRINK INCREASED ACTINOBACTERIA. FIRMICUTES/BACTEROIDETES RATIO DECREASED AND CORRELATED WITH BMI. ONLY FASTING INCREASED BUTYRATE SIGNIFICANTLY

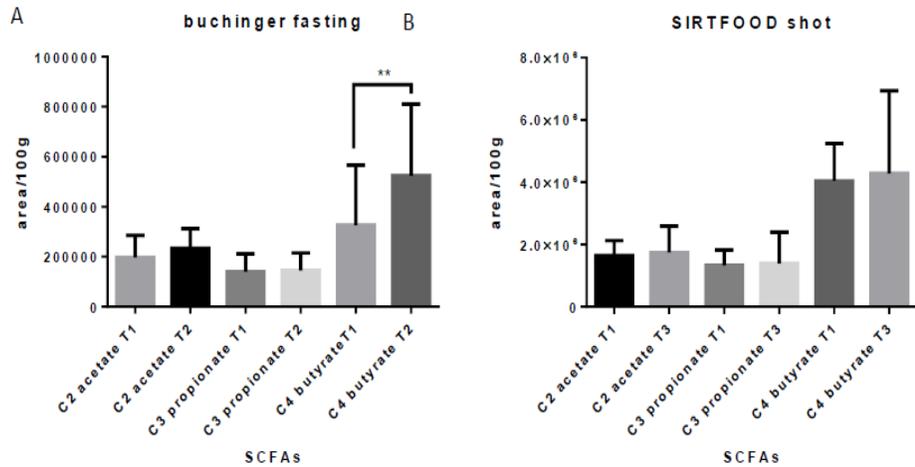


Figure 7: Amount of SCFAs produced given as area/100g stool for buchinger fasting (A) and SIRTFOOD shot (B) interventions. Statistical significance between timepoint 1 (T1) and end (T2 or T3) of the intervention was determined using paired t-test for parametric values and Wilcoxon test for nonparametric values.

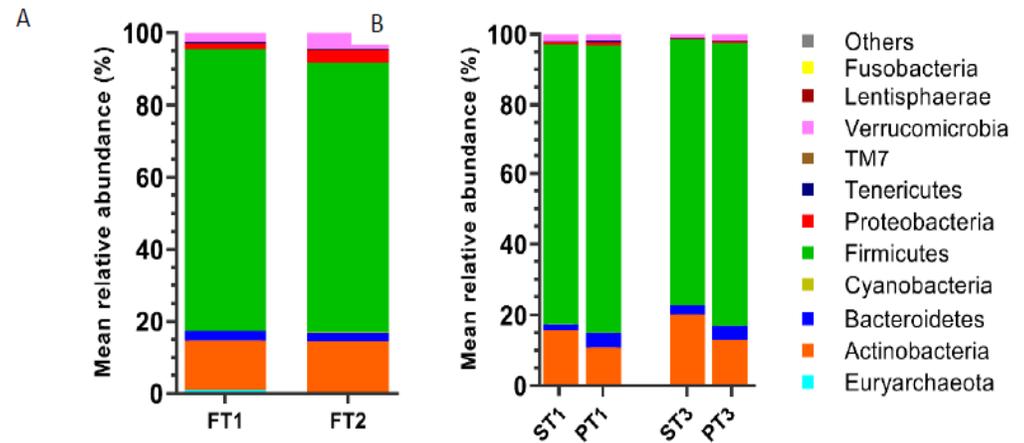


Figure 6: Abundance microbiota by phyla for fasting group (A), SIRTFOOD shot (ST1 vs ST3) (B) and placebo group (PT1 vs PT3) (B). Results are expressed in percentage of the mean of relative abundance for the different phyla. Statistical significance between timepoint 1 (T1) and end (T2 or T3) of the intervention was determined using paired t-test for parametric values and Wilcoxon test for nonparametric values.

POSITIVE CORRELATION OF THE ABUNDANCE OF BUTYRATE-PRODUCING BACTEROIDETES WITH MIR125, SIRT-1 EXPRESSION, TELOMERE LENGTH

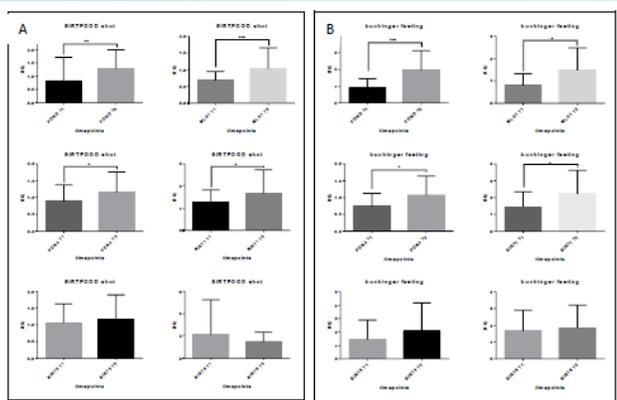


Figure 4: RQ selected mRNA gene expression (*FoxO1*, *MLH1*, *PDK4*, *SIRT1*, *SIRT3*, *SIRT6*) SIRTFOOD shot and buching fasting. The results are expressed as mean +/- SD. Statistical significance between timepoint 1 (T1) and end (T2 or T3) of the intervention was determined using paired t-test for parametric values and Wilcoxon test for nonparametric values.

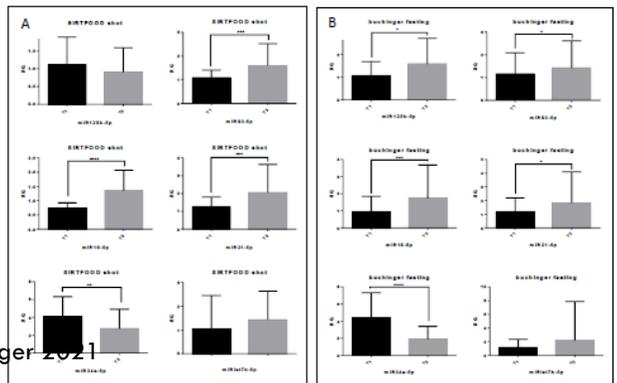


Figure 5: RQ selected mRNA gene expression (*miR125b-5p*, *miR93-5p*, *miR16-5p*, *miR21-5p*, *miR34a-5p*, *miRlet7b-3p*) SIRTFOOD shot and buching fasting. The results are expressed as mean +/- SD. Statistical significance between timepoint 1 (T1) and end (T2 or T3) of the intervention was determined using paired t-test for parametric values and Wilcoxon test for nonparametric values.

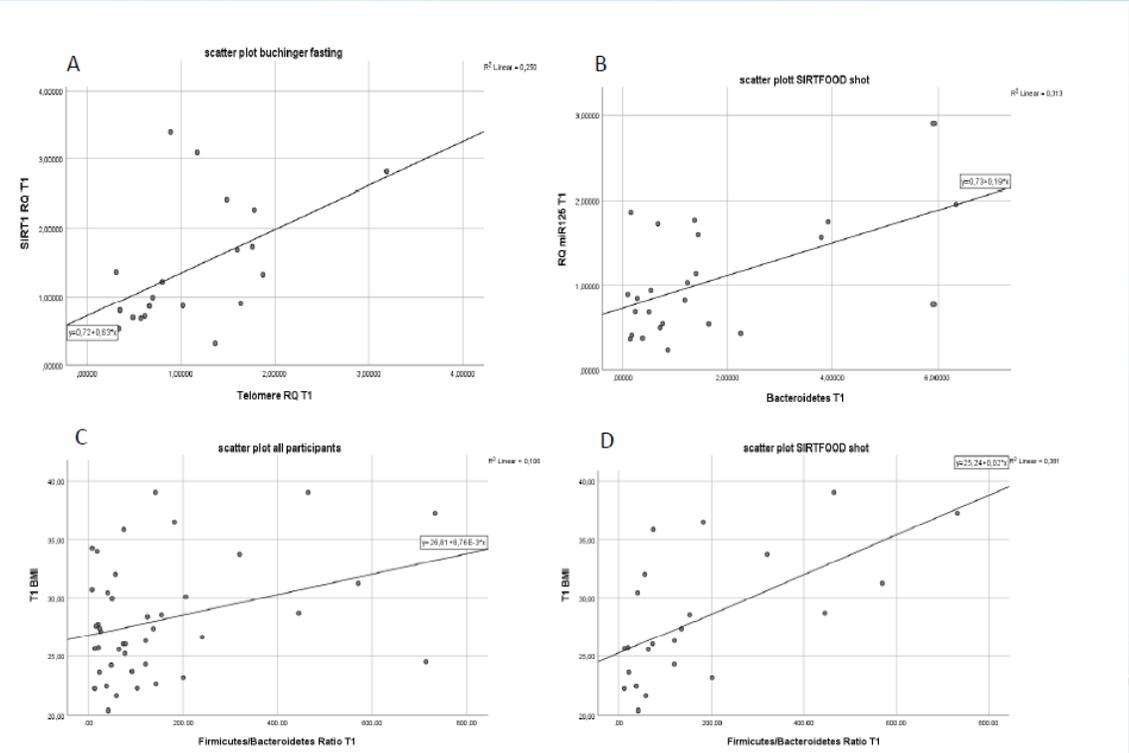


Figure 8: spss output scatter plots. (A) shows a positive correlation between telomere length and SIRT1 expression for buching fasting at baseline. Bacteroidetes and miR125b-5p positively correlated in the SIRTFOOD shot intervention at baseline (B). For all participants the ratio of Firmicutes/Bacteroidetes increased with higher BMI (C), which was also seen for the SIRTFOOD shot intervention Discussion (D). Statistical significance was defined as $p < 0.05$.

CONCLUSIONS

In conclusion fasting and to some extend fasting mimetics result in beneficial modulation of microbiota (e.g diversity, SCFA, BHP) and metabolism (e.g SIRT3, mtDNA, telomer length)

Microbiota structure seems to interfere with the expression of Sirtuins and metabolism relevant miRNAs

Hindawi
Oxidative Medicine and Cellular Longevity
Volume 2020, Article ID 4793125, 13 pages
<https://doi.org/10.1155/2020/4793125>



Research Article

Epigallocatechin Gallate Effectively Affects Senescence and Anti-SASP via *SIRT3* in 3T3-L1 Preadipocytes in Comparison with Other Bioactive Substances

Stephanie Lilja,¹ Julia Oldenburg,¹ Angelika Pointner,¹ Laura Dewald,¹ Mariam Lerch,¹ Berit Hippe,² Olivier Switzeny,² and Alexander Haslberger¹ 



International Journal of
Molecular Sciences



Article

Five Days Periodic Fasting Elevates Levels of Longevity Related *Christensenella* and Sirtuin Expression in Humans

Stephanie Lilja ¹, Carina Stoll ¹, Ulrike Krammer ¹, Berit Hippe ¹, Kalina Duszka ¹, Tewodros Debebe ², Ingrid Höfner ³, Jürgen König ¹, Angelika Pointner ¹ and Alexander Haslberger ^{1,*}

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Functional Foods in Health and Disease [Home](#) [Editorial Team](#) [Iss](#)

[Home](#) > [Vol 10, No 10 \(2020\)](#) > [Lilja](#)

Fasting and fasting mimetic supplementation address sirtuin expression, miRNA and microbiota composition

Stephanie Lilja, Hanna Bäck, Kalina Duszka, Berit Hippe, Lucia Suarez, Ingrid Höfner, Tewodros Debebe, Jürgen König, Alexander Haslberger

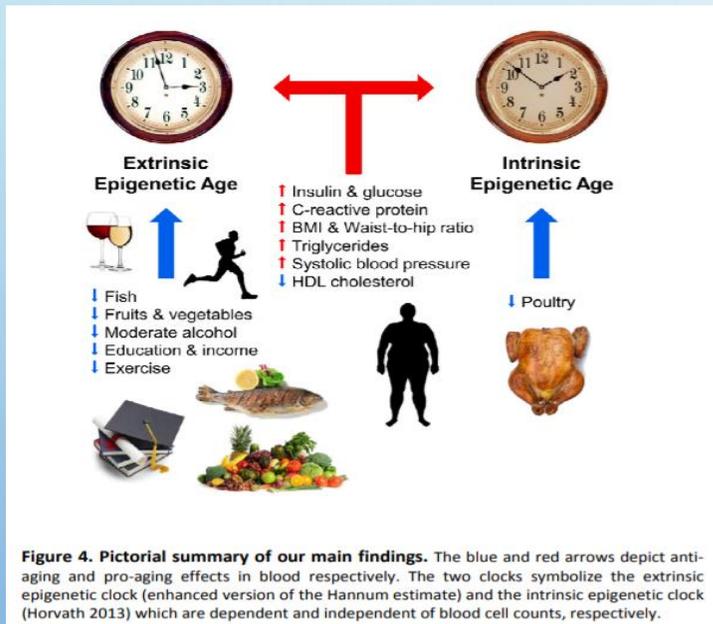
Bioactive Compounds in Health and Disease 2021; 4(4): 45-62 [BCHD](#) [Page 45 of 62](#)
[Research Article](#) [Open Access](#)



Increased Sirtuin expression, senescence regulating miRNAs, mtDNA, and bifidobacteria correlate with wellbeing and skin appearance after Sirtuin-activating drink

Stephanie Lilja, Hanna Bäck, Carinna Stoll, Anna Mayer, Angelika Pointner, Berit Hippe, Ulrike Krammer, Alexander G. Haslberger*

PERSONAL DIFFERENT RESPONSES TO NUTRITION AFFECT AGING, E.G. CLOCK AND OTHER HALLMARKS OF AGING. THIS RESULTS IN PERSONAL TYPES OF AGING, AGEOTYPES ?

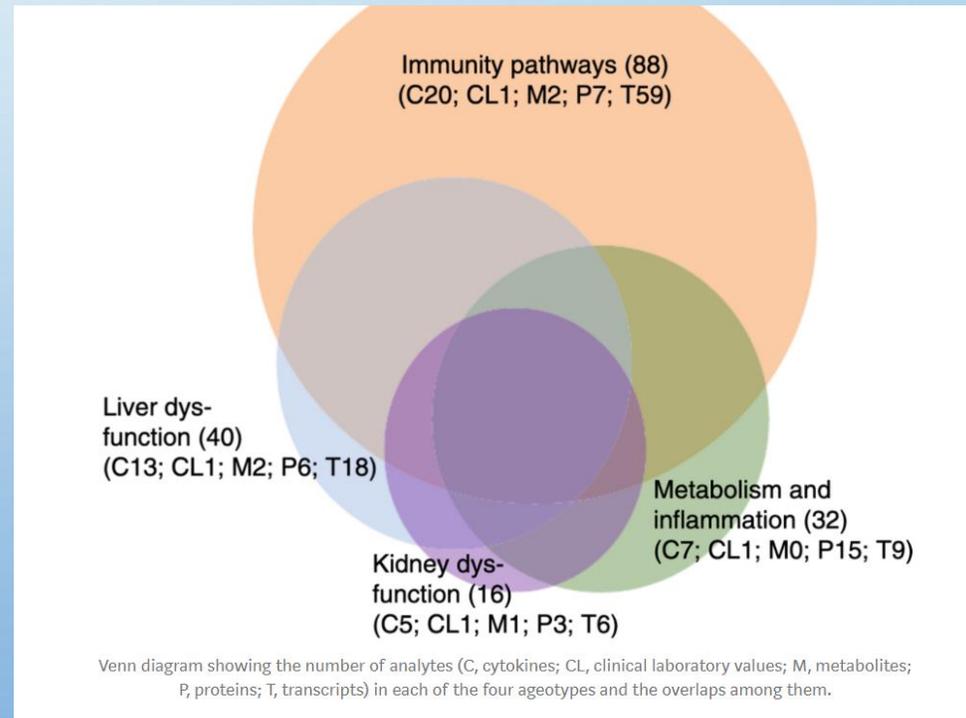


nature **medicine** FOCUS | LETTERS
<https://doi.org/10.1038/s41591-019-0798-5>

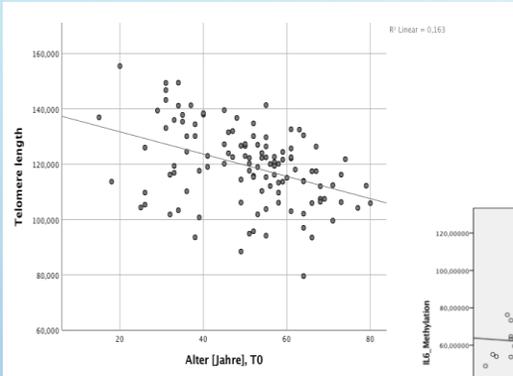
Personal aging markers and ageotypes revealed by deep longitudinal profiling

Sara Ahadi^{1,2}, Wenyu Zhou^{1,2}, Sophia Miryam Schüssler-Florenza Rose^{1,2}, M. Reza Sallani¹, Kévin Contrepols^{1,2}, Monika Avina¹, Melanie Ashland¹, Anne Brunet^{1,2} and Michael Snyder^{1,2*}

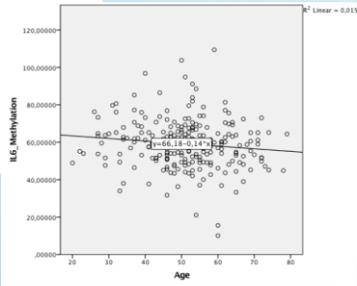
AG Haslberger



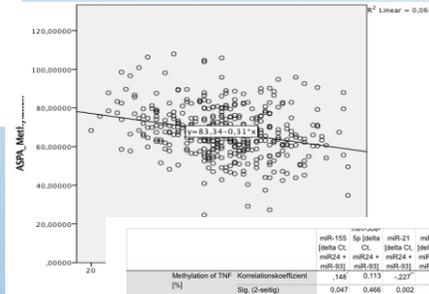
FACES OF PERSONAL AGING: CORRELATIONS OF AGE WITH TELOMERS, CPG-METHYLATION, INFLAMMATION, MIRNAS(N>500)



Correlation age with telomere-shortening



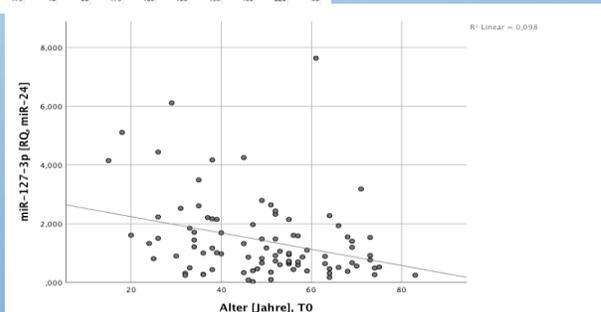
Correlation age with CPG methylation ASPA



Correlation age with CPG methylation IL6

	miR-155	miR-21	miR-19	miR-20b	miR-106	miR-30c	miR-127	miR-21	miR-15a	miR-328	3p	Methylation	Methylation	on of	n	
	[delta	Cl	[delta	Cl	[delta	Cl	[delta	Cl	[delta	Cl	[delta	Cl	Cl	Cl	Cl	
Methylation of TNF	-148	0.113	-227	-217	0.026	-0.066	199	0.148	-378	-171	-377	1.000	-0.165	-260	-252	
Sig. (2-seitig)	0.047	0.466	0.002	0.030	0.795	0.516	0.007	0.339	0.005	0.021	0.050	0.000	0.072	0.000	0.016	
N	182	44	182	100	99	100	182	44	54	182	106	106	203	120	199	91
Methylation of IL6	0.020	-0.439	232	0.035	258	368	0.068	-0.443	0.146	232	-0.034	-286	-0.165	1.000	211	0.100
Sig. (2-seitig)	0.839	0.025	0.015	0.791	0.044	0.003	0.483	0.023	0.435	0.015	0.788	0.020	0.072	0.013	0.481	
N	109	26	109	61	61	61	109	26	31	109	65	65	120	138	136	56
Methylation of Line	-0.034	-0.183	-0.082	-0.175	0.031	0.101	-0.040	-0.127	-0.047	-0.078	-1.96	-367	-265	211	1.000	0.087
Sig. (2-seitig)	0.648	0.229	0.278	0.079	0.755	0.313	0.599	0.405	0.731	0.299	0.042	0.000	0.000	0.013	0.409	
N	179	45	179	102	101	102	179	45	55	179	108	108	199	136	220	93

Correlation age with miRNA-127

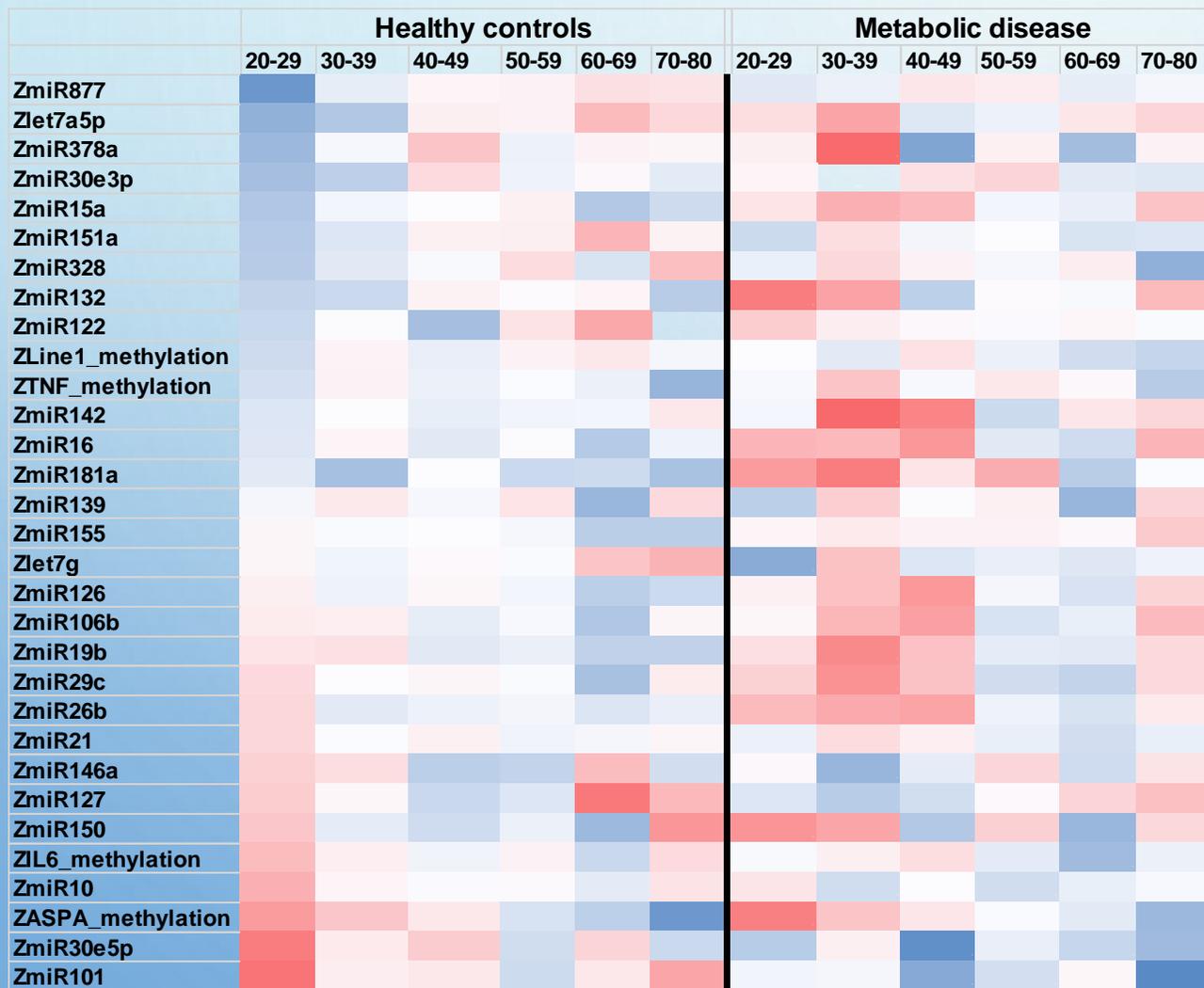


AGE DEPENDENT EPIGENETIC MARKERS: IN THE METABOLIC DISEASE GROUP (MD) CORRELATIONS ARE DISRUPTED, N>300

Marker	correlation analysis			age group comparison			direction
	All	HC	MD	all	HC	MD	
ASPA							--
IL6							--
TNF							--
miR-19b							--
miR-let-7a-5p							++
miR-877							++
miR-151a							++
miR-127							-+
miR-30e-5p							--
miR-150							
miR-21							
miR-101							

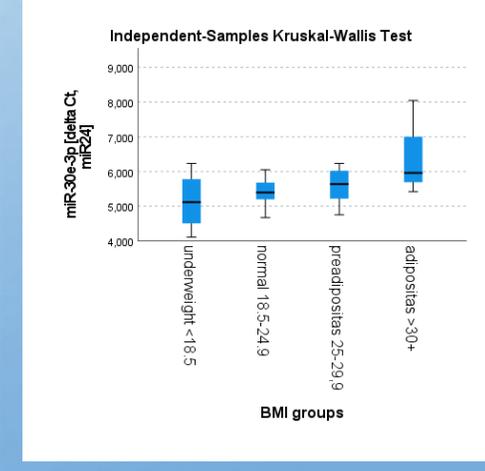
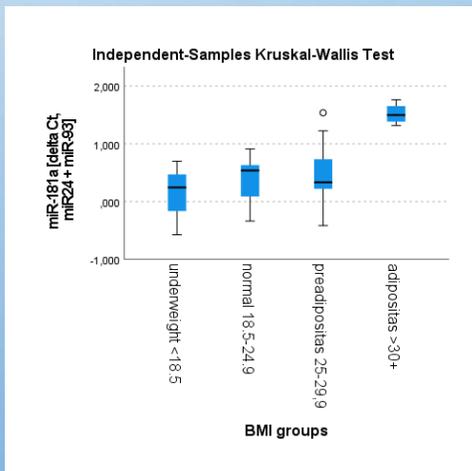
	correlation			Age Group			
	All	Healthy controls	Metabolic disorders	All	Healthy controls	Metabolic disorders	
ASPA	<0,001	<0,001	<0,001	p=0,000, korr. R2=0,185	0,001, korrR2 =0,207, überall 9<0,001, außer zwischen 40:59 zu 60-79:0,013	korrR2 = 0,140, 20-39:40-59: p = 0,041; 20-39:60-79: p=0,002	ANOVA Univariat
IL6	Trend (pearson: -0,127, p=0,079)	Pearson -0,73, p=0,412	Pearson -0,201, p=0,108	Sign. (20-39:60-79, p=0,029) korr R2=0,026	Trend means	Trend means	ANOVA Univariat
TNF	Trend (spearman -0,054, p=0,384)	spearman -0,053, p=0,491	pearson -0,105, p=318	Trend means	Trend means	Trend means	Kruskal Wallis
miR-19b	Linear regression: p=0,018; (spearman -0,298*, p=0,005)	Linear regression: p=0,027 (spearman -0,352** p=0,008)	spearman -0,174, p=0,341	Sign. 20-39:40-59 p=0,047	Trend p=0,06		Kruskal Wallis
miR-let-7a-5p	Linear regression: p=0,028 (pearson 0,236*, p=0,028)	Linear regression: p=0,001 (pearson 0,445** p=0,001)	pearson -0,085, p=0,613	Trend means	sign. (20-39:40-59: p=0,023); sign. (20-39:60-79: p=0,028) korrR2 = 0,162	sign. (20-39:40-59: p=0,027) korrR2 = 0,145	ANOVA Univariat
miR-877	Trend (spearman 0,207, p=0,058)	Trend Linear regression: 0,054 (spearman 0,288*, p=0,047)	spearman 0,105, p=0,544	X	Trend means	X	Kruskal Wallis
miR-151a	Trend (spearman 0,151, p=0,166)	(spearman 0,295* p=0,039)	spearman 0,059, p=0,727	X	Trend means	X	Kruskal Wallis
miR-127	Trend (pearson 0,288, p=0,055)	pearson 0,196, p=0,336	Trend pearson 0,444, p=0,057	Sign. (40-59:60-79, p=0,016) korr R2=0,133	Sign. (40-59:60-79 p=0,046) korrR2= 0,167	X	ANOVA Univariat
miR-30e-5p	Trend (spearman -0,246, p=0,163)	Trend spearman-0,436, p= 0,055	spearman 0,048 p =0,869	Trend means	Trend means	Trend means	Kruskal Wallis
miR-150	Trend (pearson -0,114, p=0,522)	pearson 0,082, p=0,731	pearson -0,416, p=0,139				
miR-21	Trend (pearson, -0,091, p=0,153)	pearson -0,094, p=0,233	pearson -0,098, p=377				
miR-101	Trend (pearson: -0,228, p=0,195)	Trend: pearson -0,317, p=0,173	pearson -0,074, p=0,803				

DIFFERENT AGING PATTERNS (AGE RELATED MIRNAS) IN METABOLIC DISEASE GROUP

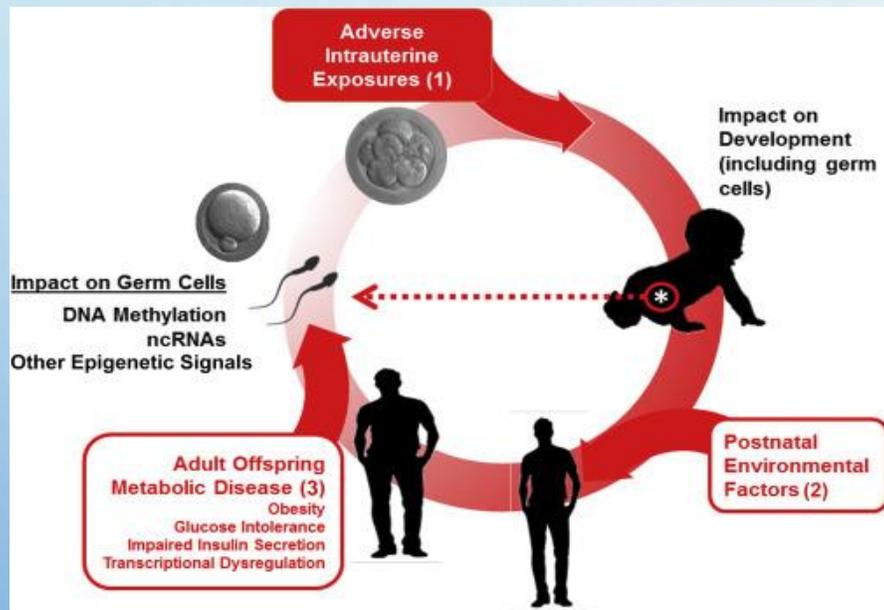


Marker	All	healthy controls	metabolic disorders
mi-181a	0,454* (Pearson)	x	0,777**
mi-378a	0,396* (Pearson), Linear regression (p=0,28)	x	0,864**
mi30e-5p	-0,339	-0,429	0,357
mi30e-3p	0,361* (spearman), Linear regression (p=0,042)	x	0,573
mi122	x	-0,359	x
mi101	x	-0,353	x
let7g	x	-0,360*	x
mi139*	p=0,007	p=0,004	

*Kruskal Wallis test between BMI groups



CONCLUSION: COMPLEX DISEASES (AGING) CAN ARISE FROM (A MIXTURE OF) PERSONAL DIVERSE CAUSES, AN ARGUMENT IN FAVOR OF PERSONALLY SPECIFIC INTERVENTIONS (E.G. METABOLIC DISEASE)



Cell Metabolism
Perspective
Epigenetic Mechanisms of Transmission of Metabolic Disease across Generations
 Vicencia Michelle Sales,¹ Anna C. Ferguson-Smith,² and Mary-Elizabeth Patti^{1,2}

		Metabolic disorder
Hereditary SNPs Somatic mutations		Symptomatic treatment
Epigenetic (hereditary) or acquired mismethylations, Histone modifications or ncRNA structure		Causative treatment ? Epigenetic active additives? mTOR – Inhibitors ? Nutrition, Lifestyle
Delivery or accessed microbiota dysbiosis		Causative treatment ? pro-, pre, postbiotics? Nutrition, Lifestyle
Psycho- neuro- immune endocrine axis		

CONSEQUENCES FOR INTERVENTION: FLAGSHIP EU-FOOD4ME STUDY RESULTS PROVE „PERSONAL NUTRITION DOES BETTER THAN ON SIZE FITS ALL“, J. MATHERS

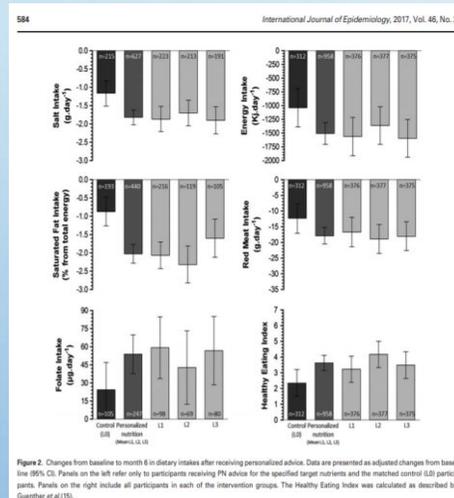
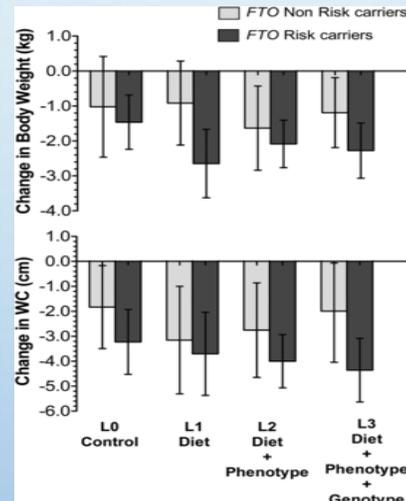


Figure 2. Changes from baseline to month 6 in dietary intakes after receiving personalized advice. Data are presented as adjusted changes from baseline (95% CI). Panels on the left refer only to participants receiving PN advice for the specified target nutrients and the matched control (L0) participants. Panels on the right include all participants in each of the intervention groups. The Healthy Eating Index was calculated as described by Guenther et al.¹⁵.

Changes of dietary intake after personalised advice
Healthy eating index



Changes in adiposity markers were greater in participants who were informed that they carried the *FTO* risk allele (level 3 AT/AA carriers) than in the nonpersonalized group




Does personalised nutrition work?

Professor John Mathers, Newcastle University, UK

John Mathers leads work on the design, delivery and evaluation of outcomes from the Food4Me project's Proof-of-Principle study. He is professor of human nutrition and director of the Human Nutrition Research Centre, Newcastle University, UK.





International Journal of Epidemiology, 2017, 46, 578-588
doi: 10.1093/ije/dyx188
Advance Access Publication Date: 12 August 2019
Original article



Interventions

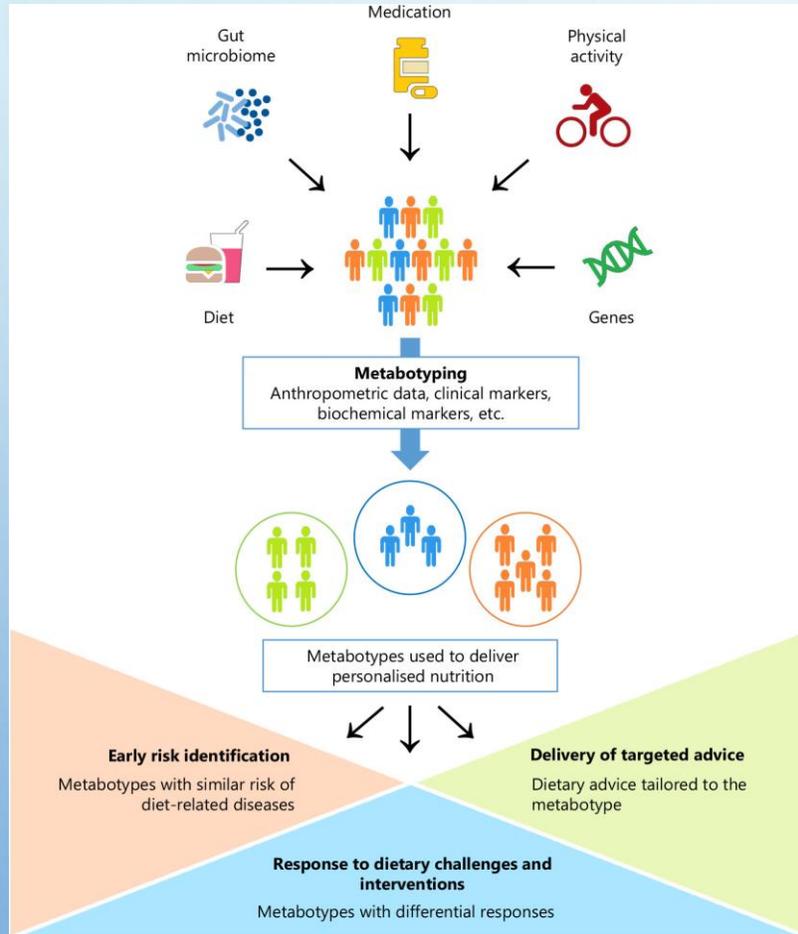
Effect of personalized nutrition on health-related behaviour change: evidence from the Food4Me European randomized controlled trial

Carlos Celis-Morales,^{1,2} Katherine M Livingstone,^{1,11} Cyril FM Marsaux,² Anna L Macready,² Rosalind Fallaize,³ Clare B O'Donovan,⁴ Clara Woolhead,⁵ Hannah Forster,⁶ Marianne C Walsh,⁴ Santiago Navas-Carretero,⁷ Rodrigo San-Cristobal,⁸ Lydia Tairigoti,⁹ Christina P Lambrinou,⁶ Christina Mavrogianni,⁶ George Moschonis,⁶ Silvia Kolossa,⁷ Jacqueline Hallmann,⁷ Magdalena Godlewska,⁸ Agnieszka Surwitto,⁸ Iwona Traczyk,⁷ Christian A Drevon,⁷ Jildau Bouwman,¹⁰ Ben van Ommen,¹⁰ Keith Grimaldi,¹¹ Laurence D Parrell,¹² John NS Matthews,¹³ Yannis Manios,⁶ Hannelore Daniel,⁷ J Alfredo Martinez,⁷ Julie A Lovegrove,⁹ Eileen R Gibney,⁴ Lorraine Brennan,⁴ Wim HM Saris,² Mike Gibney,⁴ and John C Mathers,^{1,2,13,14} on behalf of the Food4Me Study

Can genetic-based advice help you lose weight? Findings from the Food4Me European randomized controlled trial¹⁻³

Carlos Celis-Morales,^{4,5,16,18} Cyril FM Marsaux,^{6,16,18} Katherine M Livingstone,^{4,16,18} Santiago Navas-Carretero,⁷ Silvia Kolossa,¹⁶ Rosalind Fallaize,⁷ Anna L Macready,⁸ Clare O'Donovan,⁹ Clara Woolhead,⁹ Hannah Forster,⁹ Mike J Gibney,⁹ Agnieszka Surwitto,¹² Iwona Traczyk,¹² Christian A Drevon,¹¹ Keith Grimaldi,¹⁴ Jildau Bouwman,¹⁵ Mike J Gibney,⁹ Marianne C Walsh,⁹ Eileen R Gibney,⁹ Lorraine Brennan,⁹ Julie A Lovegrove,⁹ J Alfredo Martinez,⁹ Wim HM Saris,^{6,17,18} and John C Mathers^{1,17,18}

DEFINITION OF METABOTYPES FROM GENETIC-, MICROBIOTA- METABOLOMICS BASED INFORMATION, METABOTYPING



AG Haslberger

Molecular Nutrition
Food Research

Research Article | Open Access | CC BY NC ND

Evaluation of the Metabotype Concept Identified in an Irish Population in the German KORA Cohort Study

Anna Riedl, Elaine Hillesheim, Nina Wawro, Christa Meisinger, Annette Peters, Michael Roden, Florian Kronenberg, Christian Herder, Wolfgang Rathmann, Henry Völzke, Martin Reincke ... See all authors

First published: 11 February 2020 | <https://doi.org/10.1002/mnfr.201900918> | Citations: 1

Hillesheim et al. *Nutr Metab (Lond)* (2020) 17:82
<https://doi.org/10.1186/s12986-020-00499-z> Nutrition & Metabolism

RESEARCH Open Access

Optimisation of a metabolotype approach to deliver targeted dietary advice

Elaine Hillesheim^{1,2}, Miriam F. Ryan¹, Eileen Gibney¹, Helen M. Roche^{2,3} and Lorraine Brennan^{1,2*}

2021

25

CONSEQUENCES OF METABOTYPES, DIETS NEXT STEP TRACKERS

Spectrum of Possibilities for Human Metabolism

Carbo Types

Increasing need for Carbohydrates
Decreasing need for Proteins,
Fats & Purines



Mixed Types

Relatively balanced need for
Carbohydrates, Proteins, Fats & Purines



Protein Types

Increasing need for Proteins,
Fats & Purines
Decreasing need for Carbohydrates



Carbo Type Characteristics:

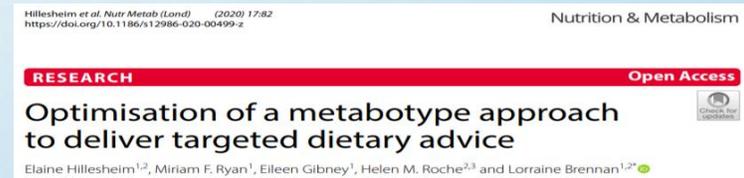
- Casual relationship with food
- Skipping a meal is usually not a big deal
- Needs high quality Vegetable and/or Fruit nutrition at their

Mixed Types:

Can identify with some characteristics of both Carbo Types & Protein Types - but, typically

Protein Type Characteristics:

- Intense relationship with food - loves to eat & tends to eat fast
- Skipping a meal IS a big deal
- Needs some high quality animal Protein & Fat at every meal to



Personalisation of additives for Prevention

Monitoring basic hallmarks of health/aging. Use of mixes of supplements, functional foods which address specific mechanisms „Achilles Fersen Concept“



Safety of Food Additives

Food additives are thoroughly studied, including extensive toxicological testing, before they are approved for use in food.

U.S. FDA "Guidance for Industry and Other Stakeholders: Toxicological Principles for the Safety Assessment of Food Ingredients" (Redbook).

Food additive identity, purity and quality is provided through adherence to specifications, which are developed prior to use in food.

Food additives have been used safely for decades.



Precision Probiotics + Prebiotics with Viome's Gut Intelligence™ Test

For gut health

AND WHAT HAPPENS TO OUR PYRAMIDE? BUT ALREADY THE DIETARY REFERENCE VALUES 1992 US USDA-PYRAMIDE, USED AN INDIVIDUALISED APPROACH, AGE, LIFESTYLE (WORK)

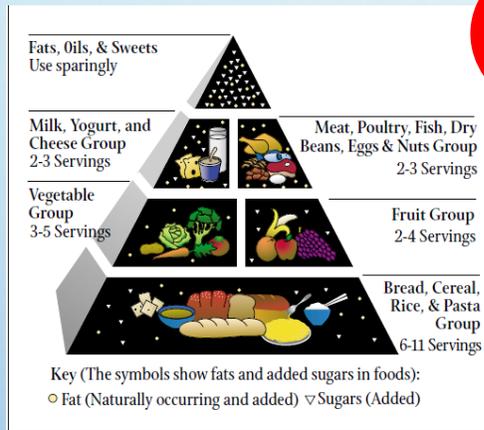


FIGURE 2
1992 FOOD PYRAMID

TABLE 1
MY PYRAMID FOOD PLAN FOR A 38-YEAR-OLD MALE WHO EXERCISES AN AVERAGE OF 30-60 MINUTES PER DAY.

Food group	Recommendation (per day)
Grains	9 oz. (half should be whole grains)
Vegetables	3.5 cups
Fruits	2 cups
Milk	3 cups
Meat and beans	6.5 oz.
Oil	8 tsp.
Discretionary	410 calories (extra fats and sugars)
Total daily calories	2,600

TABLE 2
SAMPLE MENU THAT MEETS THE MY PYRAMID RECOMMENDATIONS FOR HEALTHY EATING FOR A 38-YEAR-OLD MALE WHO EXERCISES AN AVERAGE OF 30-60 MINUTES PER DAY.*

Breakfast	Bagel with fruit other than raisins, 1 large (3.5" to 3.75" diameter) Orange juice, 1.5 cup
Snack	Black beans, cooked, no fat added, 1 cup Rice, brown, medium-grain, cooked, 5 cup Rend, multigrain, toasted, 2 large slices
Lunch	McDonald's garden salad French fries, from frozen, deep-fried, 1 small fast-food order Milk, 1%, 1 cup

Anatomy of MyPyramid

One size doesn't fit all

USDA's new MyPyramid symbolizes a personalized approach to healthy eating and physical activity. The symbol has been designed to be simple. It has been developed to remind consumers to make healthy food choices and to be active every day. The different parts of the symbol are described below.

Activity

Activity is represented by the steps and the person climbing them, as a reminder of the importance of daily physical activity.

Moderation

Moderation is represented by the narrowing of each food group from bottom to top. The wider base stands for foods with little or no solid fats or added sugars. These should be selected more often. The narrower top area stands for foods containing more added sugars and solid fats. The more active you are, the more of these foods can fit into your diet.

Personalization

Personalization is shown by the person on the steps, the slogan, and the URL. Find the kinds of amounts of food to eat each day at www.MyPyramid.gov.

Proportionality

Proportionality is shown by the different widths of the food group bands. The widths suggest how much food a person should choose from each group. The widths are just a general guide, not exact proportions. Check the website for how much is right for you.

Variety

Variety is symbolized by the 6 color bands representing the 5 food groups of the Pyramid and oils. This illustrates that foods from all groups are needed each day for good health.

Gradual Improvement

Gradual improvement is encouraged by the slogan. It suggests that individuals can benefit from taking small steps to improve their diet and lifestyle each day.



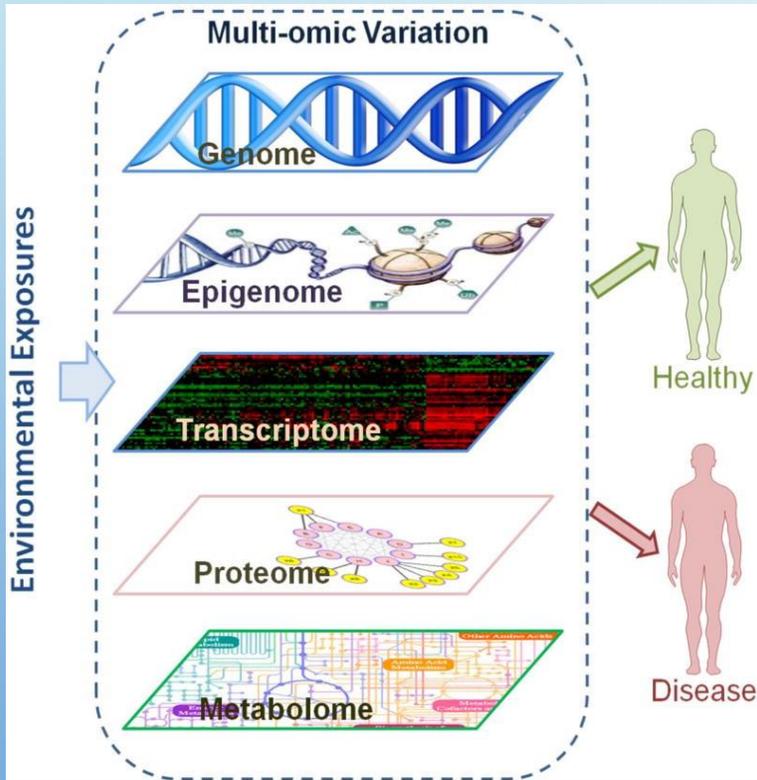
MyPyramid.gov
STEPS TO A HEALTHIER YOU



FIGURE 1
THE NEW FOOD PYRAMID ATTEMPTS TO IMPROVE HEALTH THROUGH A MORE INDIVIDUALIZED APPROACH TO DIET

John Neustadt
Integrative Medicine 2005

IMPORTANCE OF GOOD MARKERS, NUTRITION: FOLLOWING THE WAY OF PERSONALISED, PREZISION MEDICINE, CFDNA) ?



Epigenetic markers, quite stable, eg condens events over longer time spans

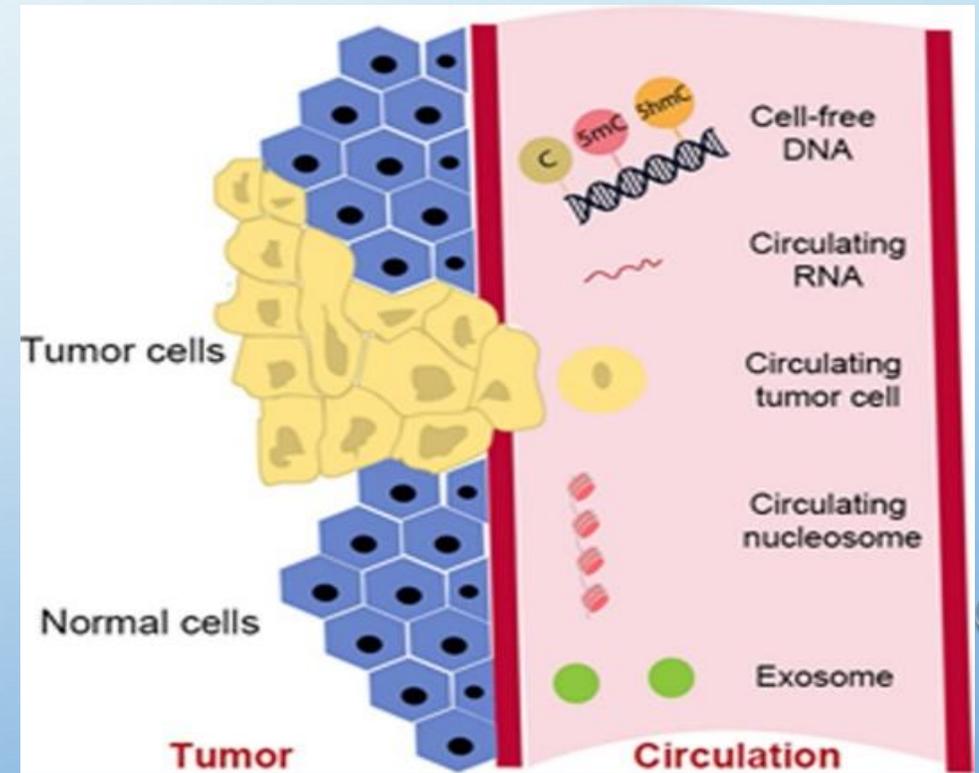
Metabolomic marker reflect more immediate events

ORIGINAL ARTICLES
Epidemiology Biostatistics and Public Health - 2016, Volume 13, Number 2

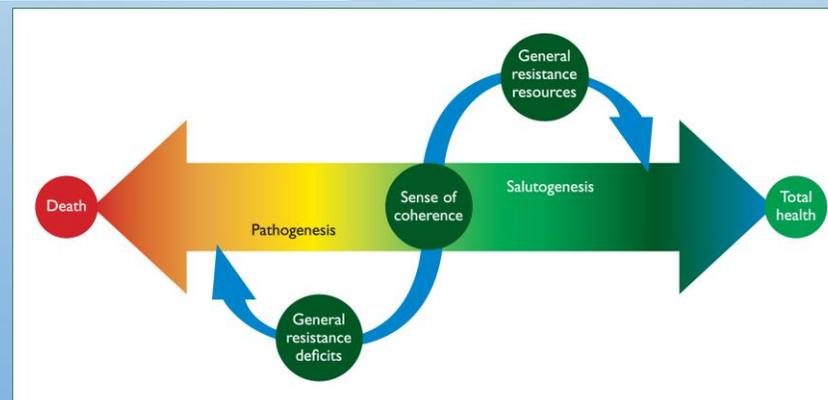
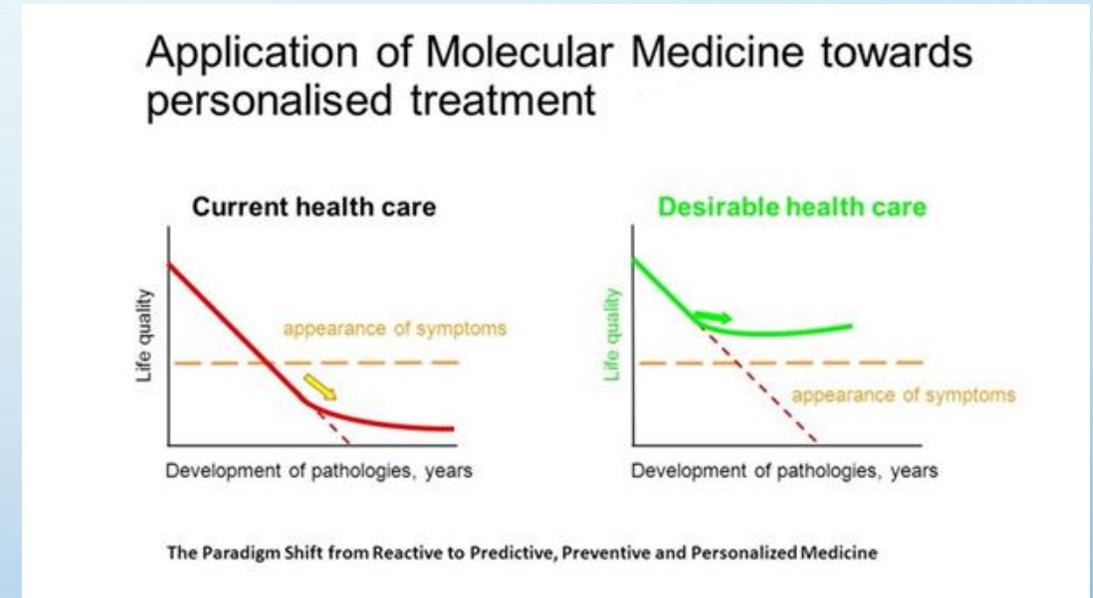
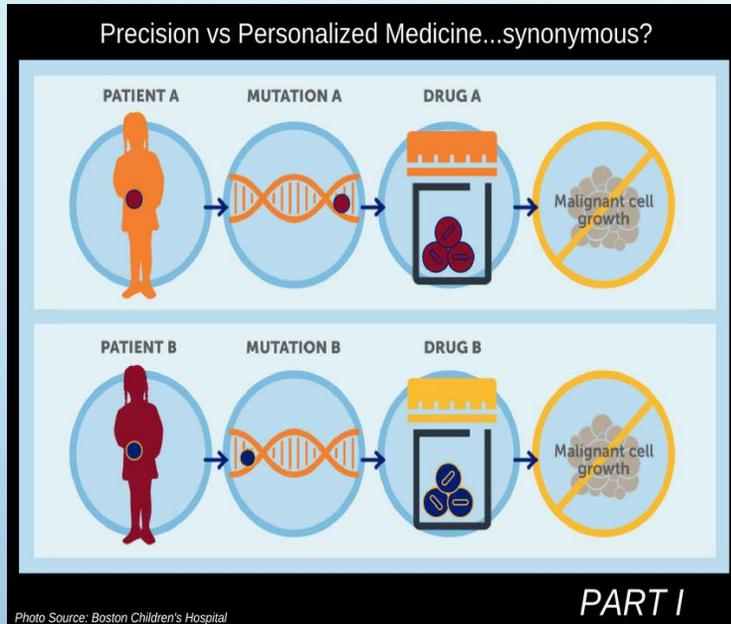
The Relevance of Epigenetic Biomarkers for Breast Cancer and Obesity for Personalised Treatment in Public Healthcare: A Systematic Review

Andrea Goettler^[1], Alexander Haslberger^[2], Elena Ambrosino^[3]

[1] Faculty of Health, Medicine & Life Sciences, University of Mozaarich, 6229 ER Mozaarich, The Netherlands
[2] Dep. for Nutritional Research, University of Vienna, Althanstrasse 14, 1090 Vienna, Austria
[3] Elena Ambrosino Institute of Public Health Genomics, Department of Genetics and Cell Biology, Research Institute CROW, Faculty of Health, Medicine & Life Sciences, University of Mozaarich



Discussion: Prevention, intervention, Salutogenesis personal or precision medicine, synonyme? personal or precision nutrition, synonyme?



PRECISION, PERSONALISED NUTRITION, WHERE WE ARE, WHERE TO GO

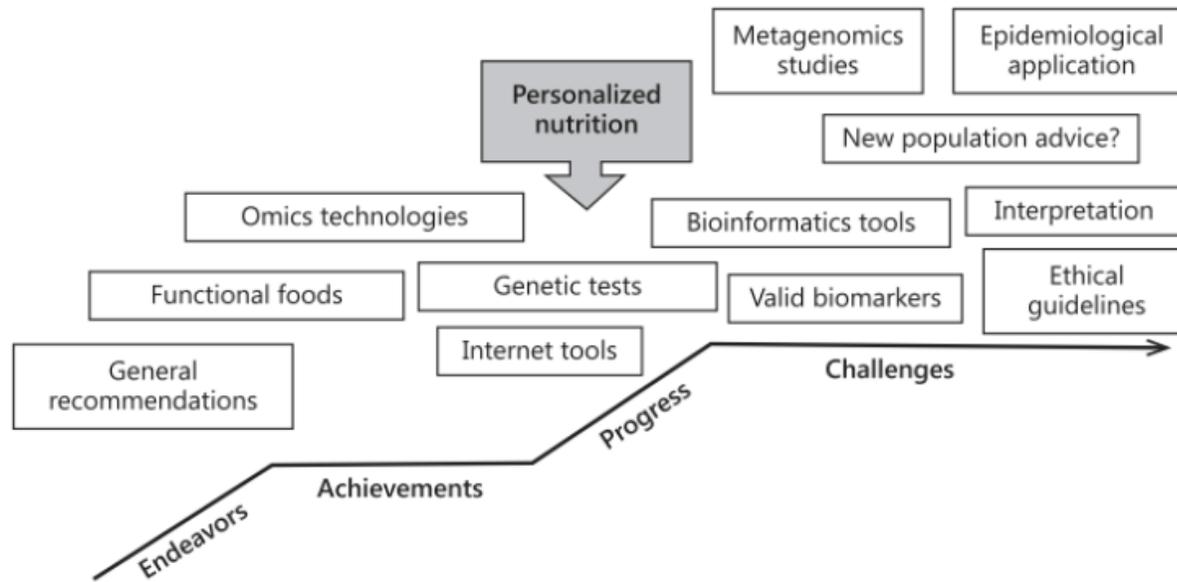


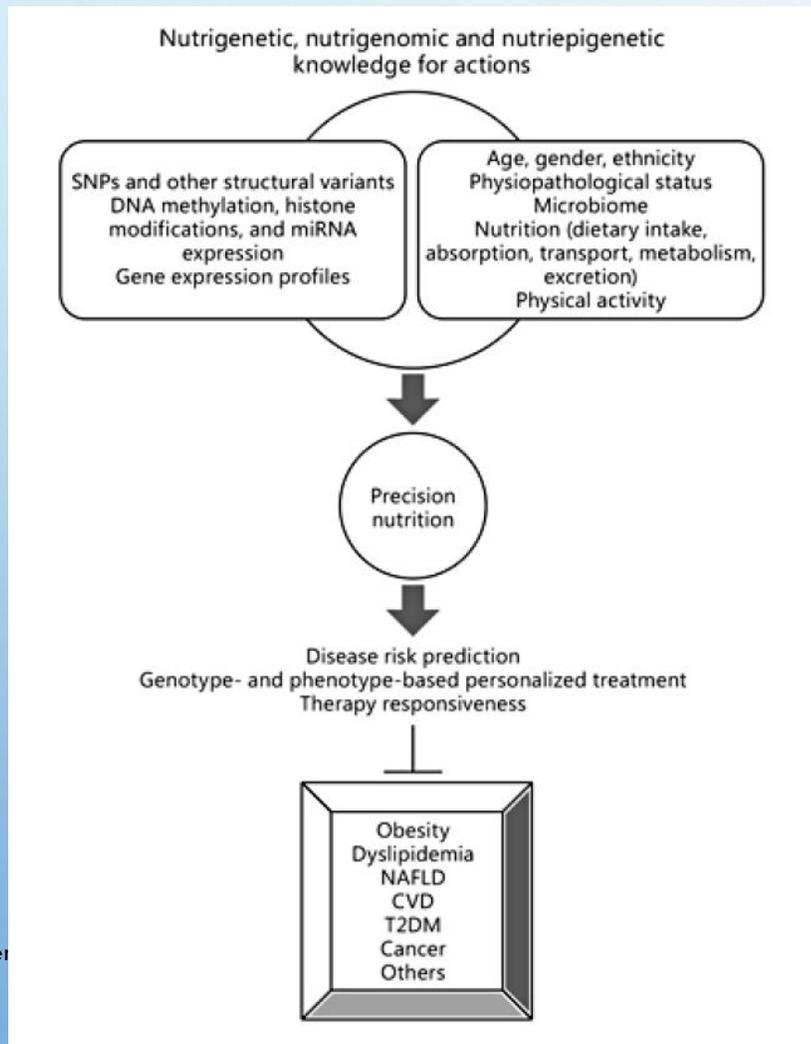
Fig. Achievements already made and challenges faced by personalised nutrition (Prasad et al., 2016)

Personalisierte Ernährung und Einteilung/ Klassifizierung von metabolischen Typen basierend auf genetischen, epigenetischen und mikrobiologischen Analysen

Personalized nutrition and classification of metabolic types based on genetics, epigenetics and gut microbiota

Stephanie Lilja, Diana Gessner, Christina Schnitzler, Nicola Stephanou-Rieser, Claudia Nichterl, Angelika Pointner, Elena Tomeva, Marlene Remely, Alexander Haslberger

PRECISION-, PERSONALISED NUTRITION, THE WAY WE MAY GO



Mobile apps and wearable devices facilitate real-time assessment of dietary intake and provide feedback which can improve glycaemic control and diabetes management.

By integrating these technologies with big data analytics, precision nutrition has the potential to provide personalised nutrition guidance for more effective prevention and management of complex metabolic diseases

(D. D. Wang & Hu, 2018).

