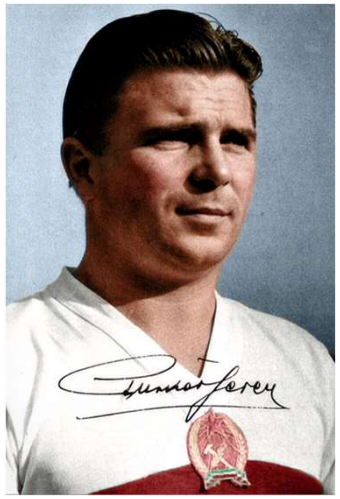
Microbiome &
teljesítmény avagy
NUTRITION DOPING

Dr Richard E. Schwab
BioMteam & MiND Brain-Gut Center
Budapest Hungary

1. **Introduction**
2. Microbiome
3. Barrier and inflammation
4. Clinical practice




METABOLIZMUS • 2021. • XIX. ÉVFOLYAM • 1. SZÁM • 38–42.

METABOLIZMUS

A cikk online változata megtalálható a www.metabolizmusonline.hu weboldalon

Schwab Richárd dr.[1], Bán Orsolya[1], Stella Péter dr.[1], Peták István dr.[2], Kempler Péter dr.[3], Kopper Bence dr.[4]

[1]MIND Brain-Gut Center Budapest, [2]Semmelweis Egyetem, Farmakológiai Intézet, [3]Semmelweis Egyetem, Belgyógyászati és Onkológiai Klinika, [4]Testnevelési Egyetem

„TÁPLÁLKOZÁSDOPPING" – AVAGY A MIKROBIOM-SÉRÜLÉS SZEREPE A VISSZAVONULT ÉLSPORTOLÓK BETEGSÉGKOCKÁZATAIBAN

A mikrobiom-kutatás új perspektívákat nyithat a sportolók táplálkozásában is. A sportolók teljesítményorientált táplálkozása olyan maradandó sérüléseket okozhat a bélflórát alkotó baktériumok biodiverzitásában, amelyek tartós gyulladásra hajlamosító tényezők lehetnek. Ez egyrészt rövid és középtávon a vázizomrendszer sérüléseinek kockázatát növelheti, ugyanakkor olyan hosszú távú kardiovaszkuláris és daganatos kockázatokat jelenthet, amelyek az elhízás és a klasszikus klinikai tünetek nélkül „némák" maradhatnak klinikai értelemben. A kumulált kockázatok és következményeik ugyanakkor a visszavonulás után egyszerre manifesztálódhatnak, ami összességében magyarázatul szolgálhat az élsportolóknak az átlag népességhez képest elmaradó, vagy annak megfelelő morbiditási és mortalitási mutatóinak vonatkozásában.

Az új generációs szekvenáláson alapuló mikrobiom-vizsgálatokkal – amelyek a biodiverzitás matematikai mérőszámát is képesek meghatározni, illetve tetszőleges, korábban ismeretlen dysbioticus fajok kimutatására is alkalmasak – objektivizálni lehet a mikrobiom-sérülést. Ez lehetővé teszi a visszavonult, mind a ma ismert szövődményekkel rendelkező sportolók tercier, mind a tünetmentes sportolók szekunder prevencióját (rehabilitációját), illetve az utánpótlás és az aktív sportolók primer prevencióját is. A mikrobiom-kutatás jelenlegi eredményei alapján mindennek a teljesítményt tekintve nem kell kompromisszumokat jelentenie, ugyanakkor jelentős életminőség javulást és a betegségkockázatok csökkenését eredményezheti sportolóink számára.

Kulcsszavak: sportolók, táplálkozás, probiotikum, elhízás, diabétesz, mikrobiom

East Germany's doping program casts long shadow over victims

The sports doping program carried out by the former East Germany has been history for decades. But for many athletes of the era, the scars — both physical and mental — have not gone away.

Many GDR athletes were given performance-enhancing drugs

Berlin resident Birgit Boese can get to work and back home every day, but that's about all she can manage. The 48-year-old former athlete depends on crutches to get around, and she and her husband have to forego visits together to the theater, movies or local swimming pool.

"Because of the pain, we can't really take part in public life," she said.

Once groomed to compete in the shot put and compete against the world's top athletes, today she says she suffers from an irregular heartbeat, high-blood pressure, diabetes, nerve damage, kidney problems, and a list of other ailments that have made her all but an invalid.

She says she knows what is to blame for her poor health - little blue pills she was given by her coaches at a special sports school in the German Democratic Republic starting at age 11.

She was told they were vitamins; now she knows they were anabolic steroids, meant to increase her muscle mass, strengthen her endurance and put her on in the center of the winners' podium at the Olympics, gold around her neck.

Boese wants the government to make it easier for doping victims to get state aid

She and 183 other former athletes who were victims of communist East Germany's notorious doping program, many of whom still suffer from the long-term effects of the drugs, were awarded compensation in 2006 from the German Olympic

The term doping is widely used for procedures to enhance performance and is **unethical, and therefore prohibited**, by most international sports organizations, including the International Olympic Committee.

Furthermore, athletes taking explicit measures to evade detection exacerbate the ethical violation with overt deception and cheating.

WHY IS IT BANNED?
Mostly due to long term health effects

MIND CLINIC

Mitől fájhat a hasad futás közben?

2020.09.19. | Frissítve: 2020.09.22. |

IBS Diagnostic Criteria (Rome IV)

Recurrent abdominal pain on average at least 1 day per week during the previous 3 months that is associated with two or more of the following:

- *Related to defecation (may be increased or unchanged by defecation)*
- *Associated with a change in stool frequency*
- *Associated with a change in stool form or appearance*

The Rome IV criteria (May 2016) only require abdominal pain in defining this condition; "discomfort" is no longer a requirement owing to its nonspecificity
Supporting symptoms include the following:

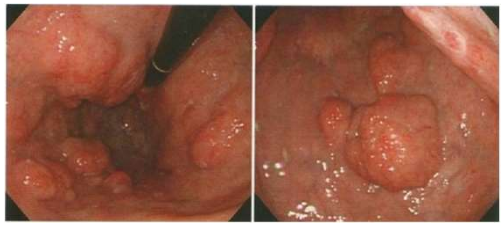
- *Altered stool frequency / form / passage (straining and/or urgency)*
- *Mucorrhea*
- *Abdominal bloating or subjective distention*

https://www.medscape.com/answers/180389-10034/ (2018 Feb)

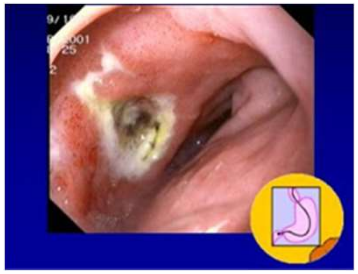
History tells us that barrier dysfunction caused by specific dysbiosis may results in inflammation - cancer disease spectrum

- we need to identify the cause of disease. Symptom-based approach leave us with a heterogeneity of causes and frustration in terms of using even potentially successful therapies

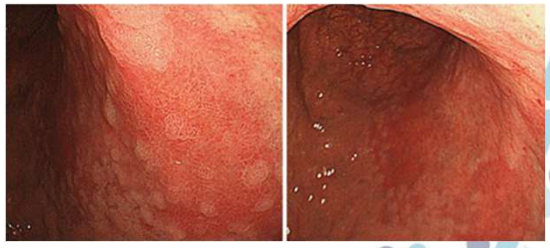
H. pylori



MALT lymphoma



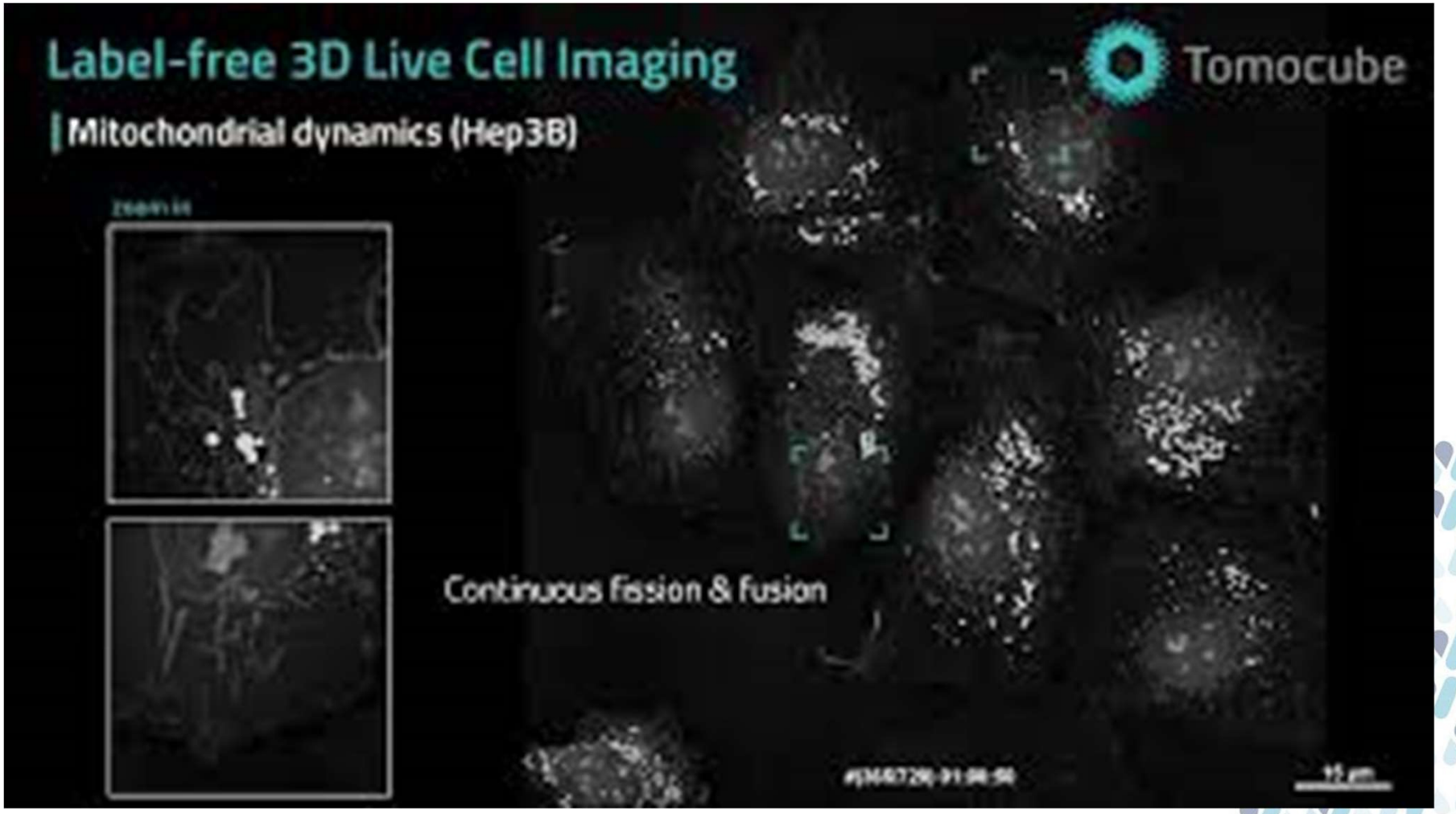
duodenal ulcer



gastritis

MIND CLINIC

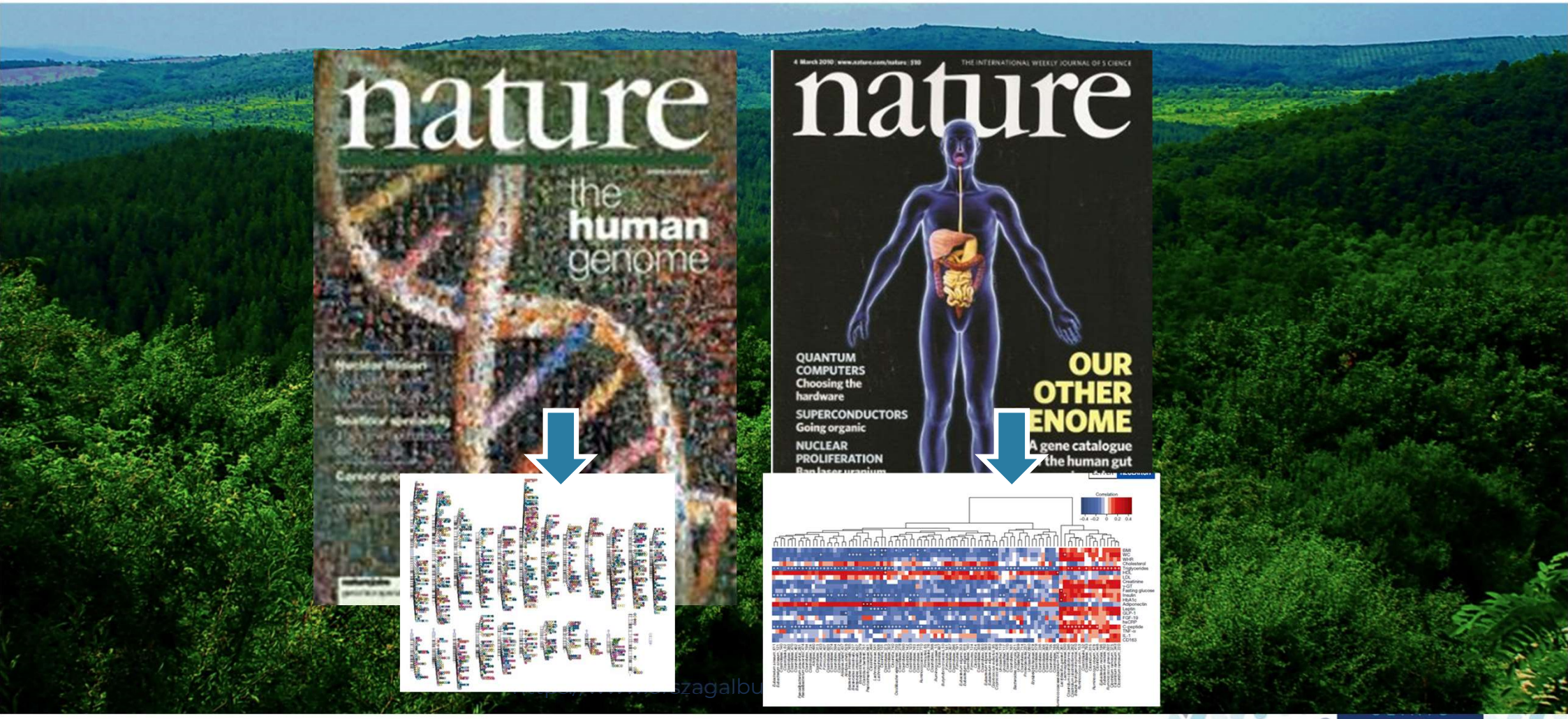
A bél microbiome és a bél-barrier két olyan faktor, ami meghatározza, hogy az energia-transzfer a bél agy tengelyen milyen hatásfokú lesz



MIND CLINIC

1. Introduction
2. **Microbiome**
3. Barrier and inflammation
4. Clinical practice

major milestones in the understanding of the human organism as an ecosystem



The human microbiome

Our bodies are **MORE MICROBE THAN HUMAN**

Our microbes have **360X** more protein DNA than us

The human body contains

4930

different individual species of **MICROORGANISMS**

Our microbiota weighs **1-2 KG**

60-80 % of the **IMMUNE SYSTEM** is in the gut

Laid out in one line, our microbes could get **2,5x** around the earth's equator

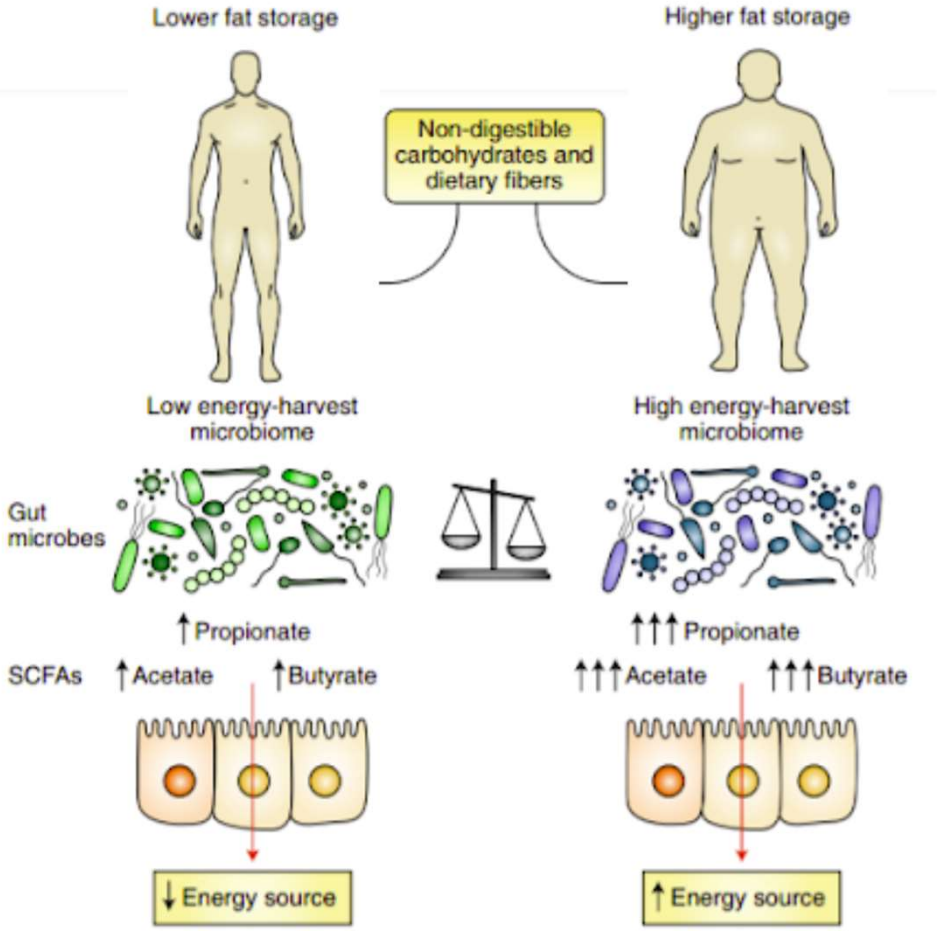
MIND CLINIC

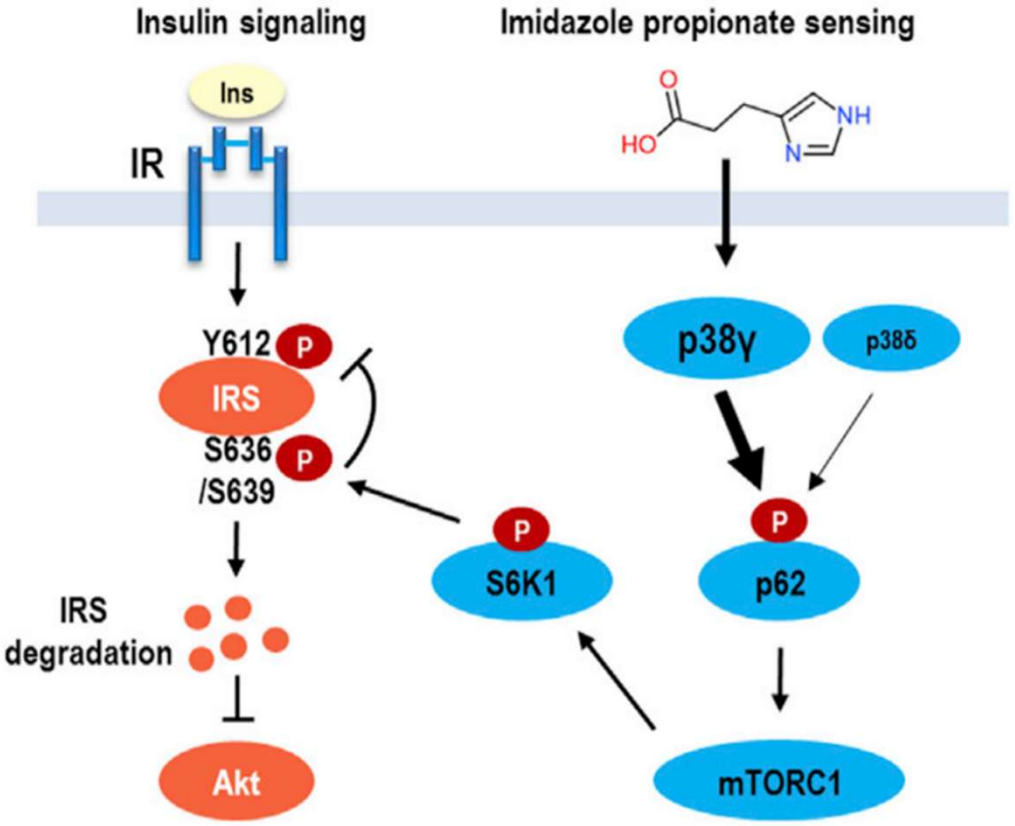
The basis of microbiome-associated diseases is typically an injury linked to environmental factors, which turns the genetic predisposition into a disease

https://jury.co.nz/tag - /wildflower-meadows/

https://wallhere.com/en/wallpaper/524152 - wasteland

Gut flora-derived propionate sensing and IR





Cell, 175: 4, P947-961, 2018

LETTERS
https://doi.org/10.1038/s41588-019-0350-x

nature genetics

Causal relationships among the gut microbiome, short-chain fatty acids and metabolic diseases

Serena Sanna[1,10]*, Natalie R. van Zuydam[2,3,10], Anubha Mahajan[2,3,10], Alexander Kurilshikov[1], Arnau Vich Vila[1,4], Urmo Võsa[1], Zlatan Mujagic[5], Ad A. M. Masclee[5], Daisy M. A. E. Jonkers[5], Marije Oosting[6], Leo A. B. Joosten[6], Mihai G. Netea[6], Lude Franke[1], Alexandra Zhernakova[1], Jingyuan Fu[1,7], Cisca Wijmenga[1,8,11]* and Mark I. McCarthy[2,3,9,11]*

Microbiome-wide association studies on large population cohorts have highlighted associations between the gut microbiome and complex traits, including type 2 diabetes (T2D) and obesity[1]. However, the causal relationships remain largely unresolved. We leveraged information from 952 normoglycemic individuals for whom genome-wide genotyping, gut metagenomic sequence and fecal short-chain fatty acid (SCFA) levels were available[2], then combined this information with genome-wide-association summary statistics for 17 metabolic and anthropometric traits. Using bidirectional Mendelian randomization (MR) analyses to assess causality[3], we found that the host-genetic-driven increase in gut production of the SCFA butyrate was associated with improved insulin response after an oral glucose-tolerance test ($P = 9.8 \times 10^{-5}$), whereas abnormalities in the production or absorption of another SCFA, propionate, were causally related to an increased risk of T2D ($P = 0.004$). These data provide evidence of a causal effect of the gut microbiome on metabolic traits and support the use of MR as a means to elucidate causal relationships from microbiome-wide association findings.

and fecal-transplantation studies have shown that insulin sensitivity increases in obese subjects with metabolic syndrome after the transfer of gut microbiota from lean donors[4,5,9,10]. Although the specific microbiome features identified as being responsible for these effects differ among studies, one consistent finding in T2D subjects is a shift in the microbiome composition away from species able to produce butyrate. Butyrate and other SCFAs, such as acetate and propionate, are produced by gut bacterial fermentation of undigested food components. After absorption by the colonocytes, these SCFAs either are used locally as fuel for colonic mucosal epithelial cells or enter the portal bloodstream[11]. Although the bulk of evidence suggests that increased SCFA production benefits the host by exerting antiobesity and antidiabetic effects[4,10,12–14], some in vitro and in vivo studies have indicated that overproduction or accumulation of SCFAs in the bowel may also lead to obesity, owing to increased energy accumulation[15,16]. Resolution of these conflicting data requires a detailed understanding of the causal relationships among gut-microbiome composition, SCFA abundance and host energy metabolism.

Using an MR approach[3], we set out to identify whether any bac-

1. Introduction
2. Microbiome
3. **Barrier and inflammation**
4. Clinical practice

Interview

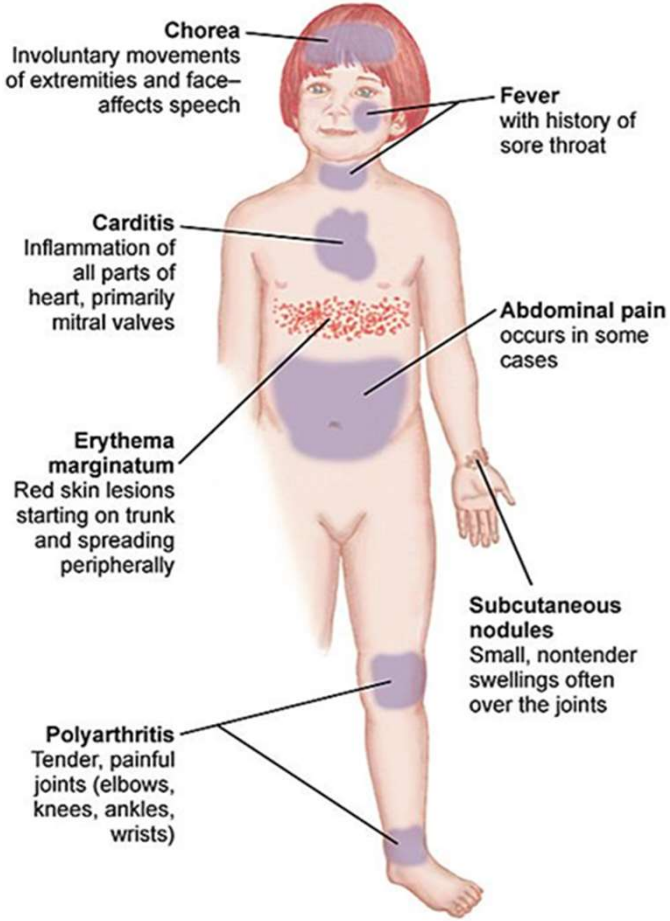
Marco van Basten: 'It was a very big fall and a really dark time'

Donald McRae

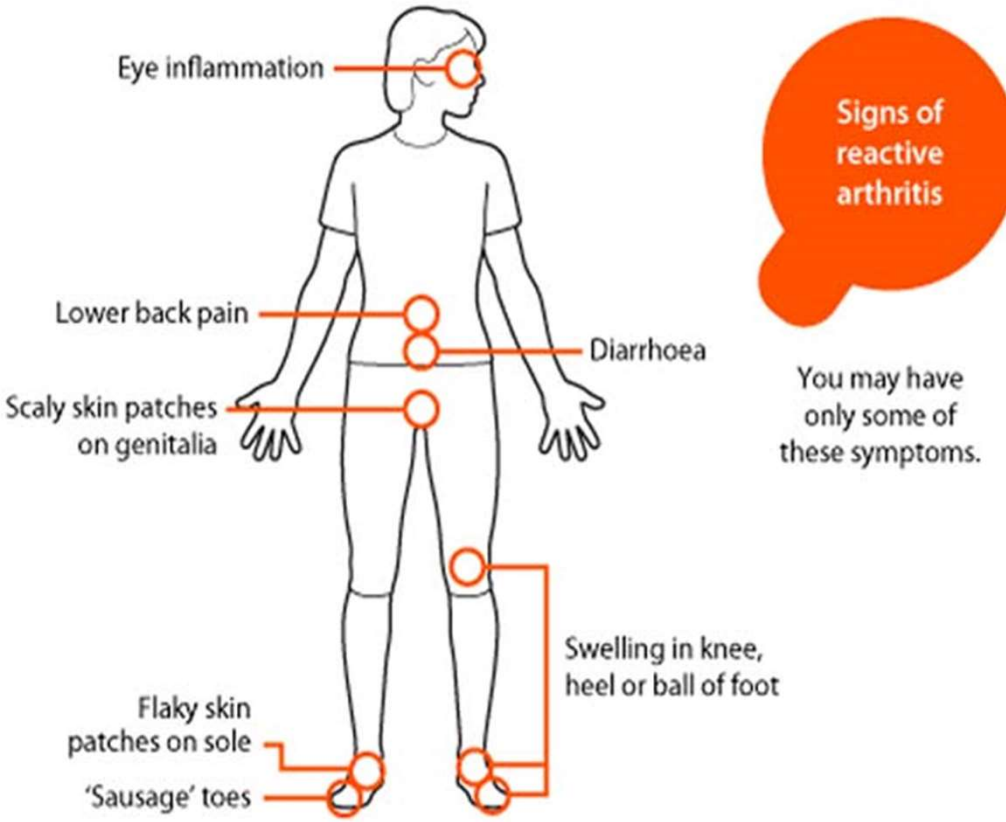
'After a lot of problems with operations I was limping. I couldn't do anything without pain. I was a little afraid,' says Marco van Basten. Photograph: Judith Jockel/The Observer

Dutch great played on until, aged 28, he could stand the pain from his damaged ankle no more. 'I went from football's highest level down to the lowest level of personal unhappiness'

Classical microbiome associated disease models



Rheumatoid fever



Reiter's syndrome

Elife. 2013 Nov 5;2:e01202. doi: 10.7554/eLife.01202.

Expansion of intestinal Prevotella copri correlates with enhanced susceptibility to arthritis.

Scher JU[1], Sczesnak A, Longman RS, Segata N, Ubeda C, Bielski C, Rostron T, Cerundolo V, Pamer EG, Abramson SB, Huttenhower C, Littman DR.

Author information

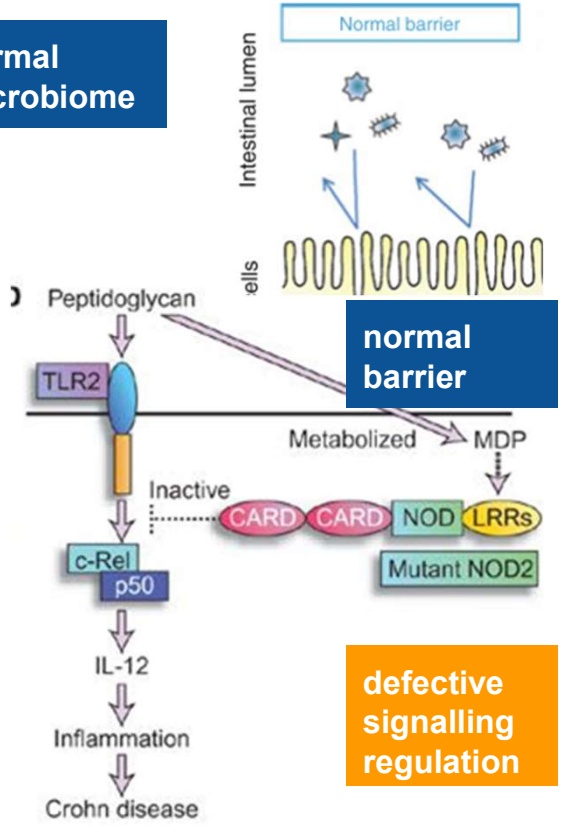
Abstract

Rheumatoid arthritis (RA) is a prevalent systemic autoimmune disease, caused by a combination of genetic and environmental factors. Animal models suggest a role for intestinal bacteria in supporting the systemic immune response required for joint inflammation. Here we performed 16S sequencing on 114 stool samples from rheumatoid arthritis patients and controls, and shotgun sequencing on a subset of 44 such samples. We identified the presence of Prevotella copri as strongly correlated with disease in new-onset untreated rheumatoid arthritis (NORA) patients. Increases in Prevotella abundance correlated with a reduction in Bacteroides and a loss of reportedly beneficial microbes in NORA subjects. We also identified unique Prevotella genes that correlated with disease. Further, colonization of mice revealed the ability of P. copri to dominate the intestinal microbiota and resulted in an increased sensitivity to chemically induced colitis. This work identifies a potential role for P. copri in the pathogenesis of RA. DOI: http://dx.doi.org/10.7554/eLife.01202.001.

Pathogen Associated Molecular Patterns (PAMP's) and IBD

Crohn's disease

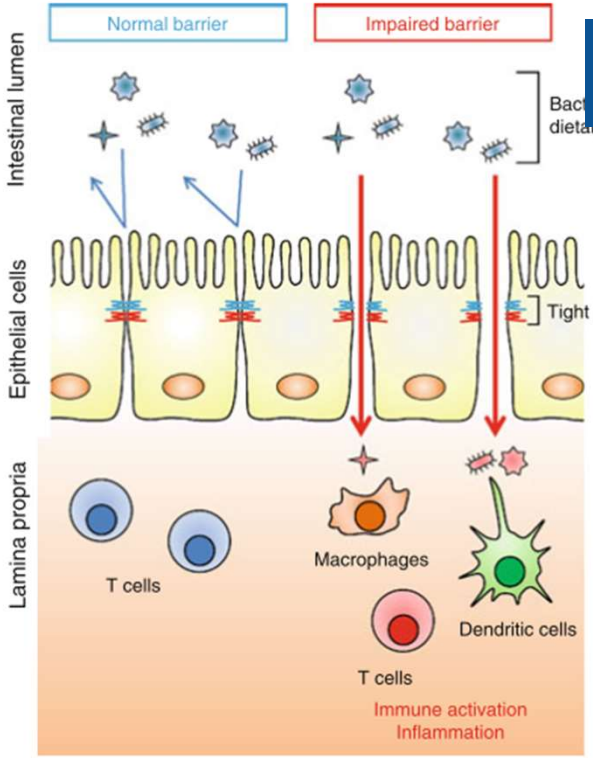
normal microbiome



normal barrier

defective signalling regulation

Ulcerative colitis

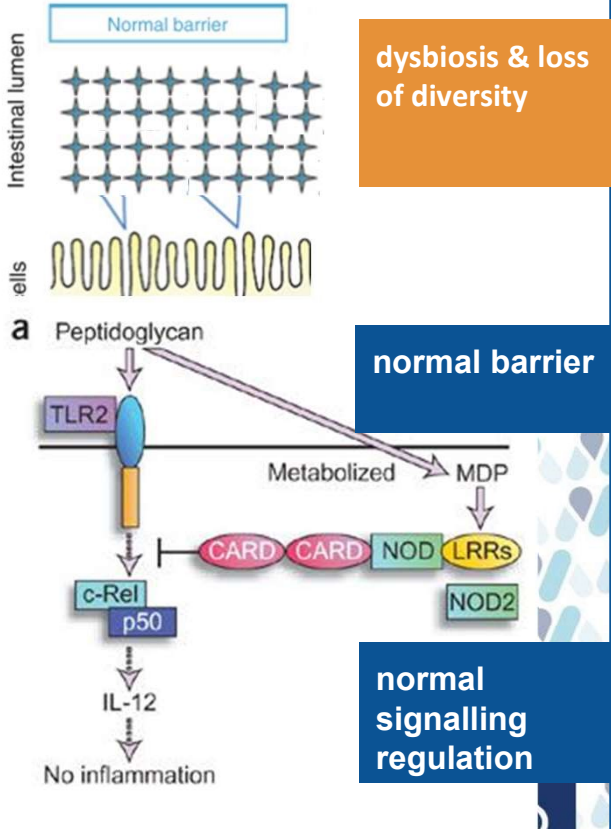


normal microbiome

defective barrier

normal signalling regulation

IBS



dysbiosis & loss of diversity

normal barrier

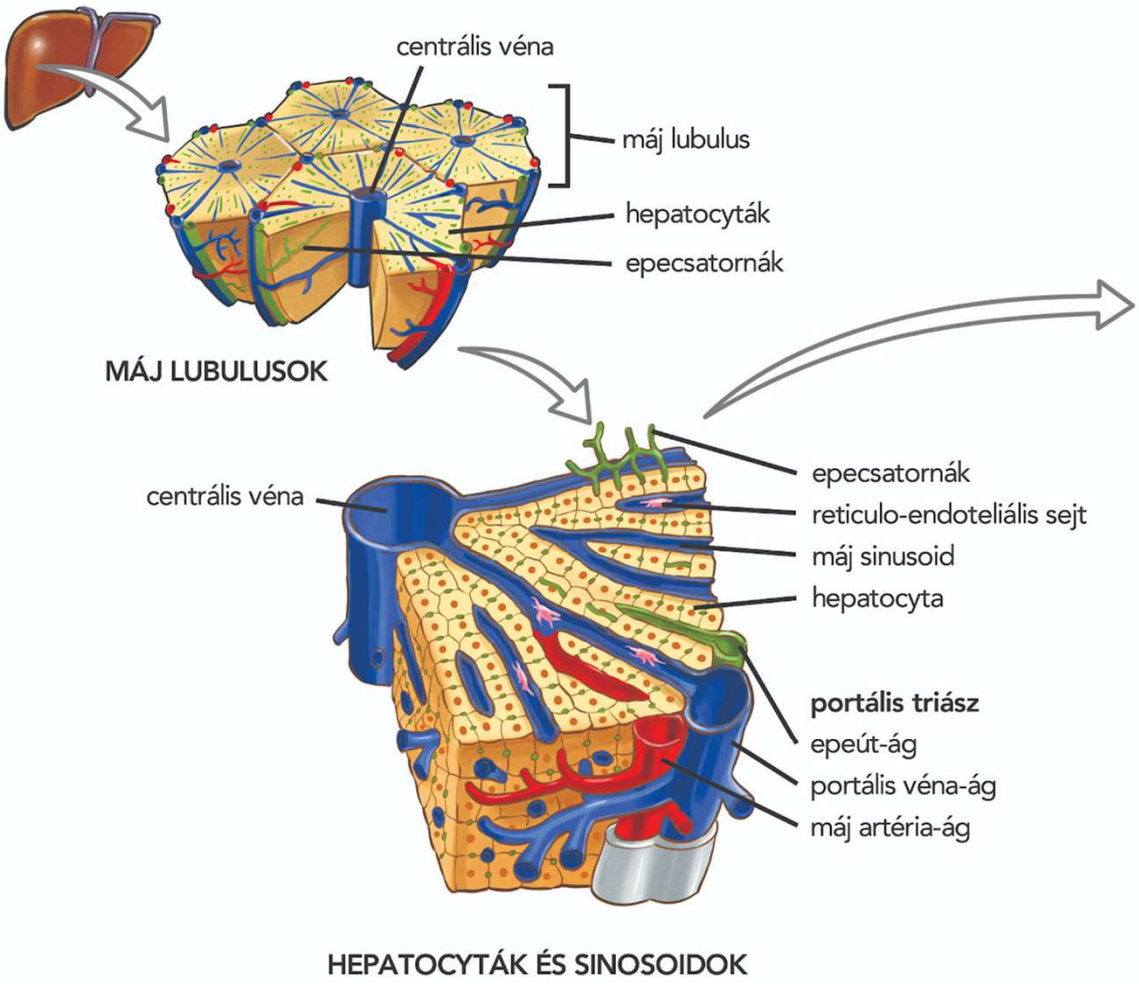
normal signalling regulation

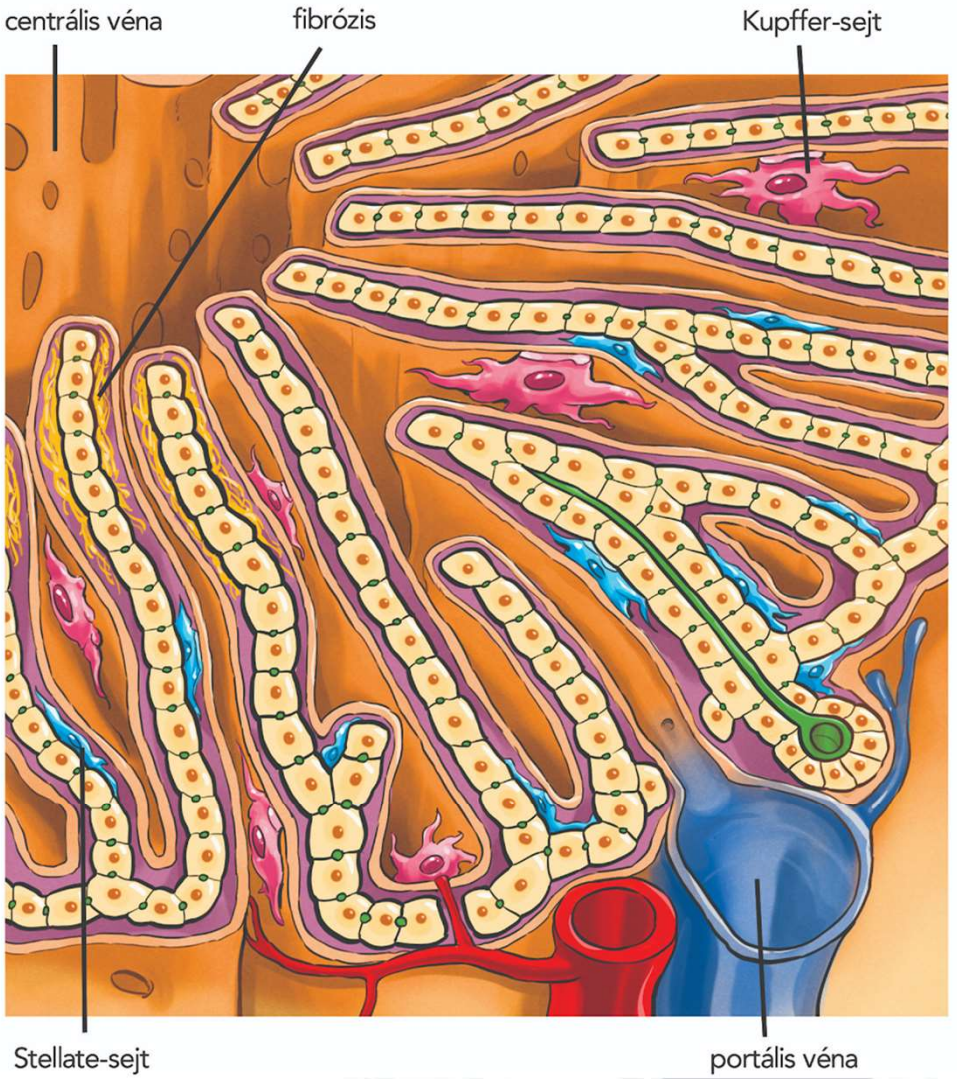
Nature Immunology **volume5**, pages776–778 (2004)

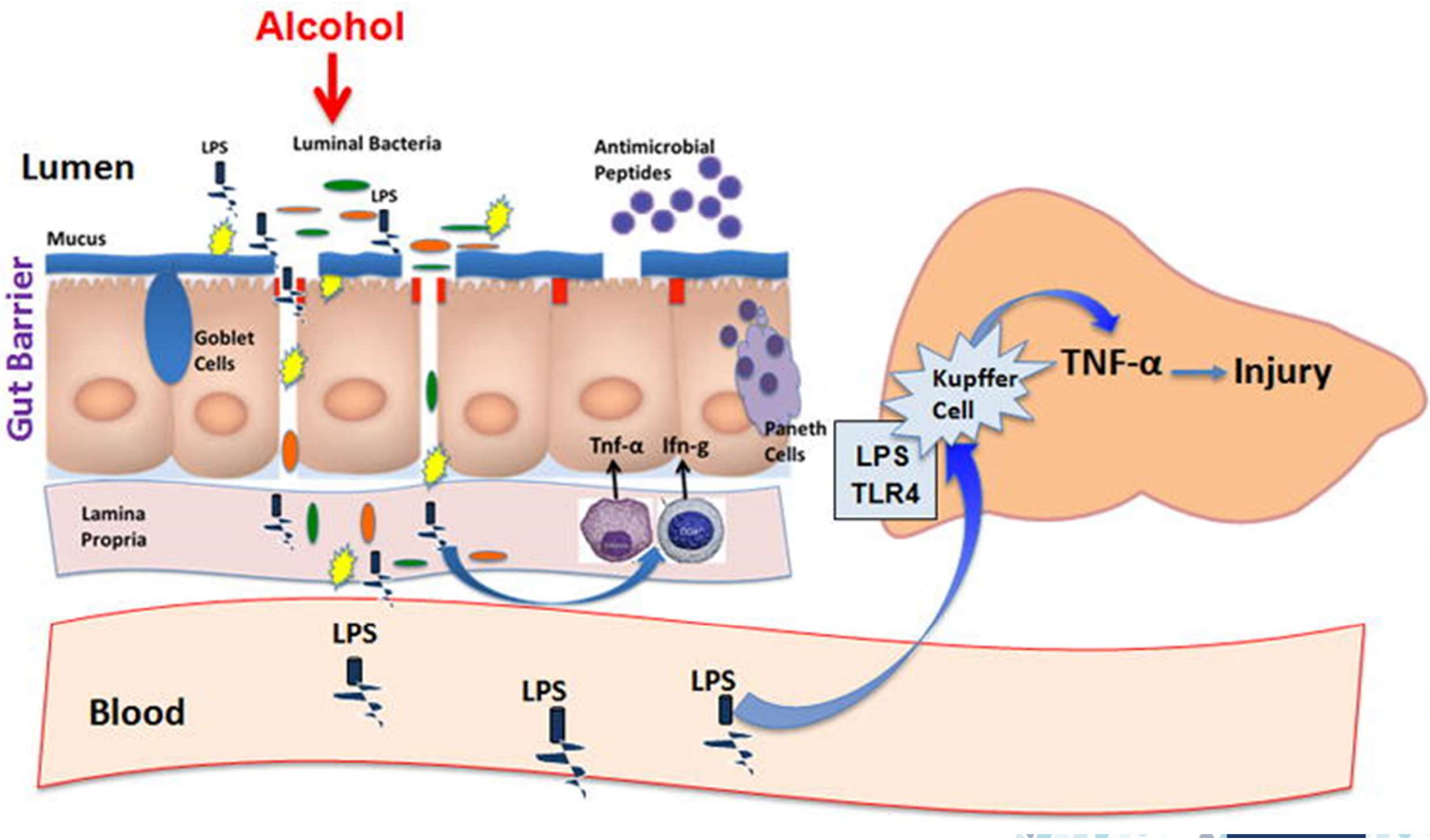

Franz Beckenbauer
1945 - 2024

Sinusoid circulation

KLINIKAI ONKOLÓGIA | 2019;6(1):25–33.






Alcohol
Lumen
LPS
Luminal Bacteria
LPS
Antimicrobial Peptides
Mucus
Gut Barrier
Goblet Cells
Paneth Cells
Tnf-α
Ifn-g
Lamina Propria
Kupffer Cell
TNF-α
Injury
LPS
TLR4
LPS
LPS
LPS
Blood

Hepatology. 2016 Mar;63(3):764-75. doi: 10.1002/hep.28356. Epub 2016 Jan 13.

The severity of nonalcoholic fatty liver disease is associated with gut dysbiosis and shift in the metabolic function of the gut microbiota.

Boursier J[1,2], Mueller O[3], Barret M[4], Machado M[5], Fizanne L[2], Araujo-Perez F[6], Guy CD[7], Seed PC[3,6], Rawls JF[3], David LA[3], Hunault G[2], Oberti F[1,2], Calès P[1,2], Diehl AM[5].

Author information

Abstract

Several animal studies have emphasized the role of gut microbiota in nonalcoholic fatty liver disease (NAFLD). However, data about gut dysbiosis in human NAFLD remain scarce in the literature, especially studies including the whole spectrum of NAFLD lesions. We aimed to evaluate the association between gut dysbiosis and severe NAFLD lesions, that is, nonalcoholic steatohepatitis (NASH) and fibrosis, in a well-characterized population of adult NAFLD. Fifty-seven patients with biopsy-proven NAFLD were enrolled. Taxonomic composition of gut microbiota was determined using 16S ribosomal RNA gene sequencing of stool samples. Thirty patients had F0/F1 fibrosis stage at liver biopsy (10 with NASH), and 27 patients had significant F≥2 fibrosis (25 with NASH). Bacteroides abundance was significantly increased in NASH and F≥2 patients, whereas Prevotella abundance was decreased. Ruminococcus abundance was significantly higher in F≥2 patients. By multivariate analysis, Bacteroides abundance was independently associated with NASH and Ruminococcus with F≥2 fibrosis. Stratification according to the abundance of these two bacteria generated three patient subgroups with increasing severity of NAFLD lesions. Based on imputed metagenomic profiles, Kyoto Encyclopedia of Genes and Genomes pathways significantly related to NASH and fibrosis F≥2 were mostly related to carbohydrate, lipid, and amino acid metabolism.

CONCLUSION NAFLD severity associates with gut dysbiosis and a shift in metabolic function of the gut microbiota. We identified Bacteroides as independently associated with NASH and Ruminococcus with significant fibrosis. Thus, gut microbiota analysis adds information to classical predictors of NAFLD severity and suggests novel metabolic targets for pre-/probiotics therapies.

Non-alcoholic fatty liver and the gut microbiota.

Bashiardes S[1], Shapiro H[1], Rozin S[1], Shibolet O[2], Elinav E[1].

Author information

Mol Metab. 2016 Jun 14;5(9):782-94.

Abstract

BACKGROUND: Non-alcoholic fatty liver (NAFLD) is a common, multi-factorial, and poorly understood liver disease whose incidence is globally rising. NAFLD is generally asymptomatic and associated with other manifestations of the metabolic syndrome. Yet, up to 25% of NAFLD patients develop a progressive inflammatory liver disease termed non-alcoholic steatohepatitis (NASH) that may progress towards cirrhosis, hepatocellular carcinoma, and the need for liver transplantation. In recent years, several lines of evidence suggest that the gut microbiome represents a significant environmental factor contributing to NAFLD development and its progression into NASH. Suggested microbiome-associated mechanisms contributing to NAFLD and NASH include dysbiosis-induced deregulation of the gut endothelial barrier function, which facilitates systemic bacterial translocation, and intestinal and hepatic inflammation. Furthermore, increased microbiome-modulated metabolites such as lipopolysaccharides, short chain fatty acids (SCFAs), bile acids, and ethanol, may affect liver pathology through multiple direct and indirect mechanisms.

SCOPE OF REVIEW: Herein, we discuss the associations, mechanisms, and clinical implications of the microbiome's contribution to NAFLD and NASH. Understanding these contributions to the development of fatty liver pathogenesis and its clinical course may serve as a basis for development of therapeutic microbiome-targeting approaches for treatment and prevention of NAFLD and NASH.

MAJOR CONCLUSIONS: Intestinal host-microbiome interactions play diverse roles in the pathogenesis and progression of NAFLD and NASH. Elucidation of the mechanisms driving these microbial effects on the pathogenesis of NAFLD and NASH may enable to identify new diagnostic and therapeutic targets of these common metabolic liver diseases. This article is part of a special issue on microbiota.

Atherosclerosis. 2017 Feb 9;259:75-82. doi: 10.1016/j.atherosclerosis.2017.02.003. [Epub ahead of print]

Hs-CRP and all-cause, cardiovascular, and cancer mortality risk: A meta-analysis.

Li Y[1], Zhong X[1], Cheng G[2], Zhao C[1], Zhang L[1], Hong Y[1], Wan Q[1], He R[1], Wang Z[1].

Author information

Abstract

BACKGROUND AND AIMS: Inconsistent findings have been reported on the association between high-sensitivity C-reactive protein (hs-CRP) and mortality risk. The objective of this meta-analysis was to investigate the association of elevated baseline hs-CRP levels with all-cause, cardiovascular, and cancer mortality risk in the general population.

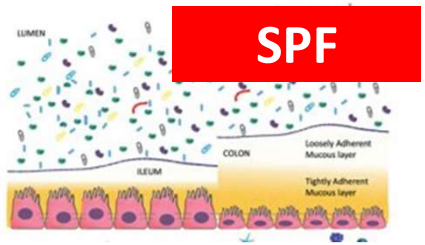
METHODS: PubMed and Embase were systematically searched for studies published from inception to October 2016. Prospective observational studies were eligible if they reported the effects of elevated baseline hs-CRP levels on cancer-related, cardiovascular or all-cause mortality in the general population. The pooled adjusted risk ratio (RR) with 95% confidence interval (CI) comparing the highest to the lowest category of hs-CRP levels was used as association measures.

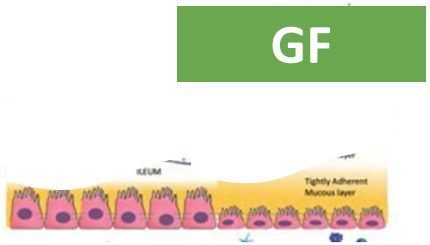
RESULTS: A total of 83,995 participants from 14 studies were identified. When comparing the highest to the lowest category of hs-CRP levels, the pooled RR was 1.25 (95% CI 1.13-1.38) for cancer-related mortality, 2.03 (95% CI 1.65-2.50) for cardiovascular mortality, and 1.75 (1.55-1.98) for all-cause mortality, respectively. Subgroup analysis showed that the effect of elevated hs-CRP levels on cancer-related mortality was observed in men (RR 1.26; 95% CI 1.11-1.43) but not in women (RR 1.03; 95% CI 0.83-1.27).

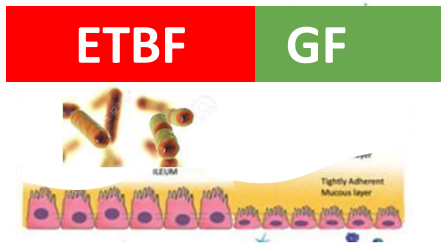
CONCLUSIONS: Elevated hs-CRP levels can independently predict risk of all-cause, cardiovascular mortality in the general population. However, the gender differences in the predictive role of hs-CRP on cancer mortality should to be further investigated.

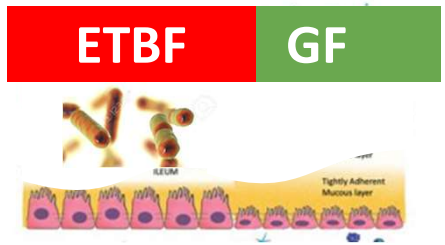
MIND
CLINIC

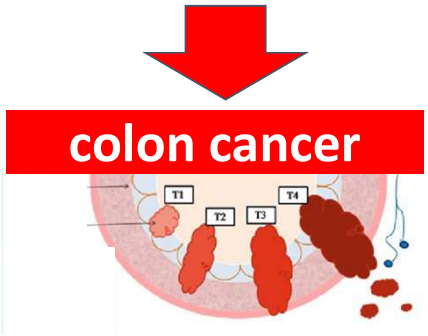
Colon cancer - genetic disorder?

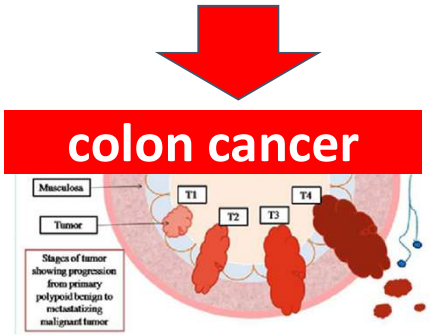










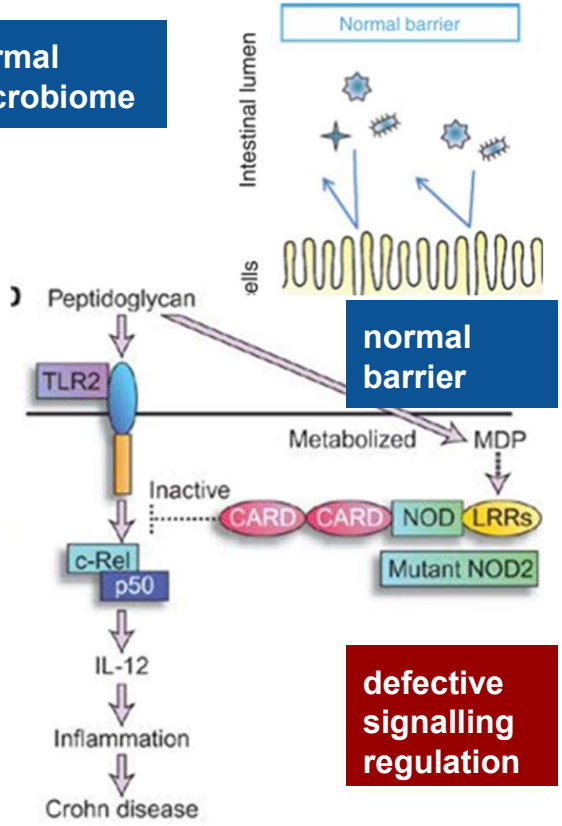


Cell Host & Microbe, VOLUME 23, ISSUE 2, P203-214.E5, FEBRUARY 14, 2018

MIND CLINIC

Pathogen Associated Molecular Patterns (PAMP's) & CANCER

Tumor free

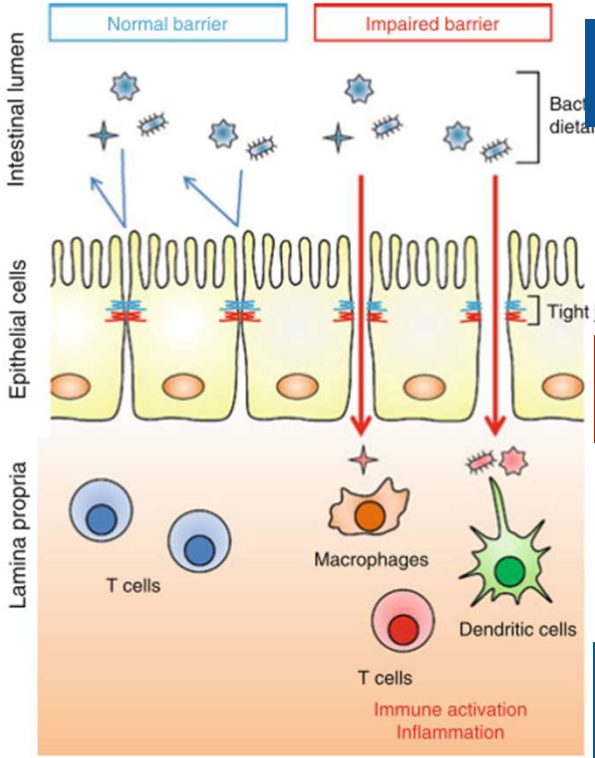


normal microbiome

normal barrier

defective signalling regulation

tumor free

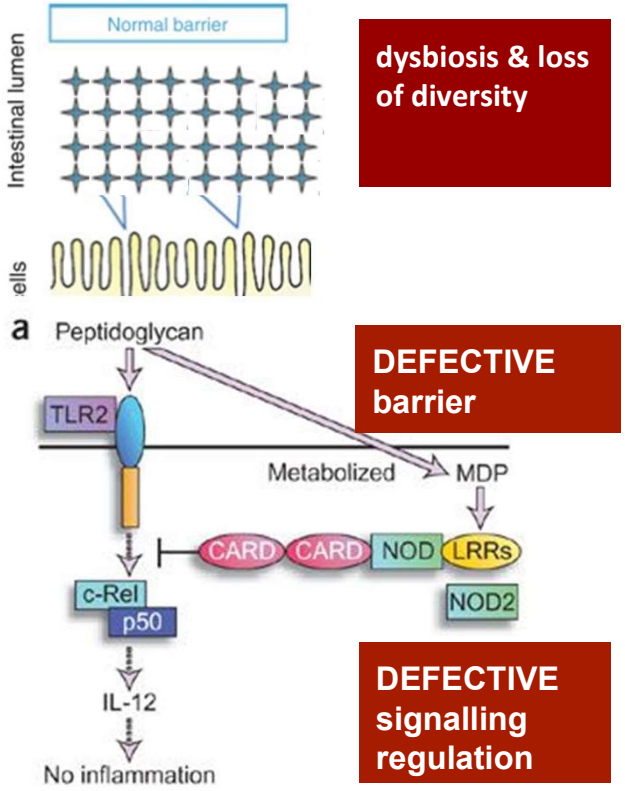


normal microbiome

defective barrier

normal signalling regulation

CANCER



dysbiosis & loss of diversity

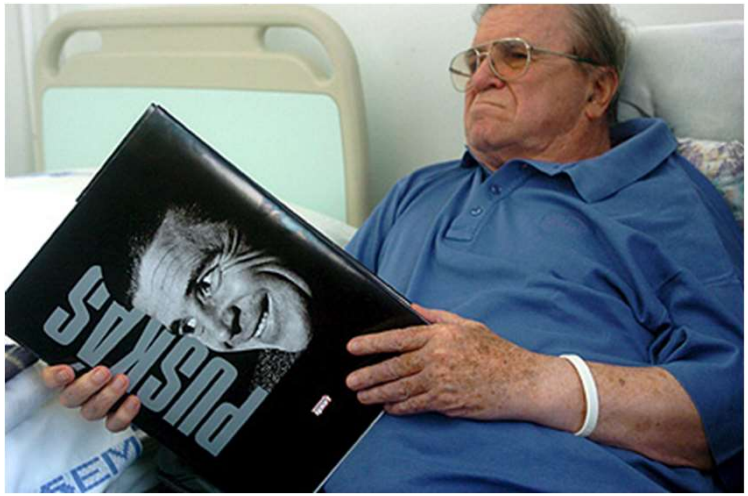
DEFECTIVE barrier

DEFECTIVE signalling regulation

Nature Immunology **volume5**, pages776–778 (2004)

MiND and MOOD and Microbiome

- **Alzheimer's and Parkinson's** .
- **Peripheral neuropathies** linked to the "metabolic syndrome" of T2DM.
- **Atherosclerosis** leading to stroke
- Some forms of **migraine**
- Some **autoimmune neuropathies** and **"neuro-inflammation"** ...and the list to be explored is long!



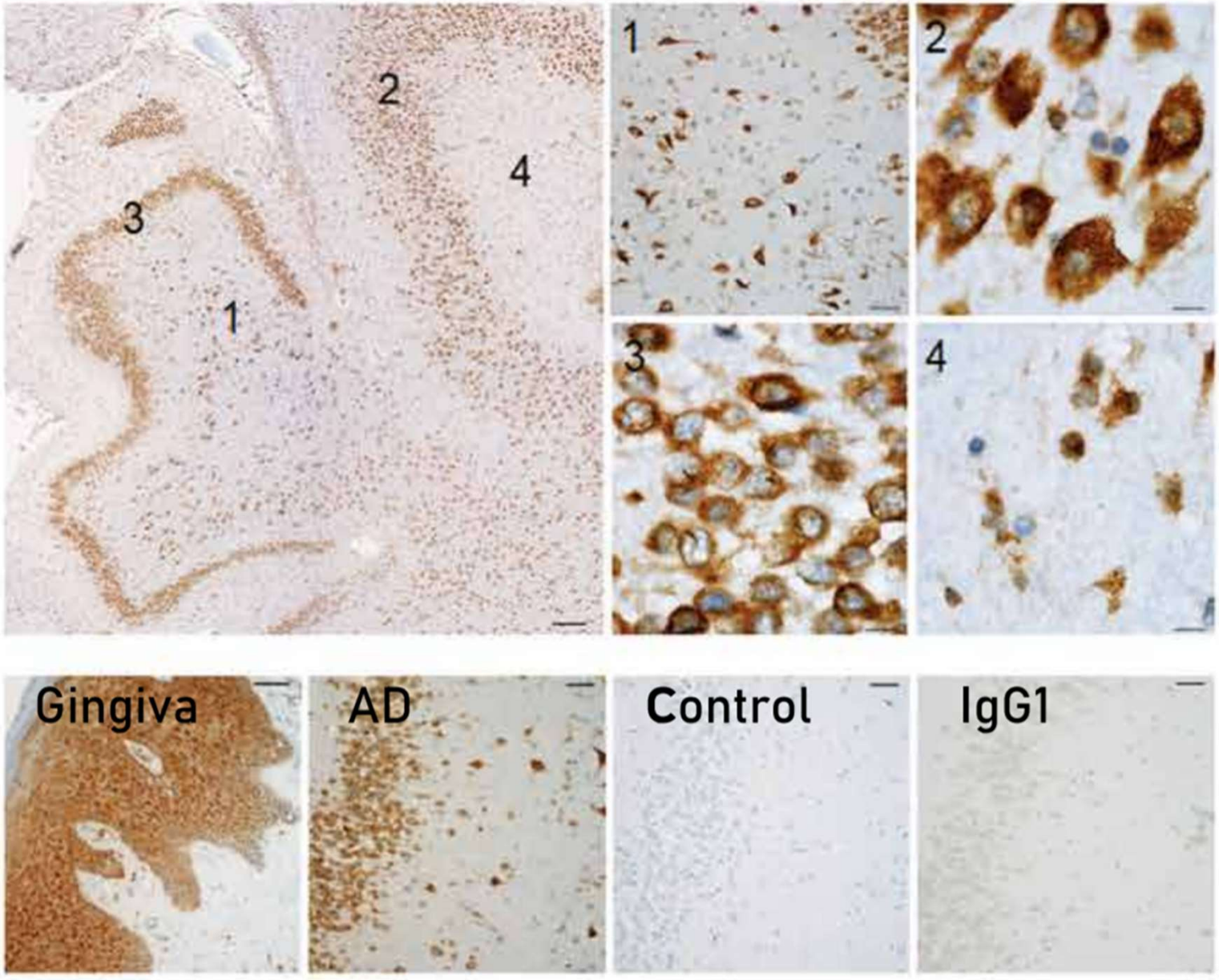
SCIENCE ADVANCES | RESEARCH ARTICLE

HEALTH AND MEDICINE

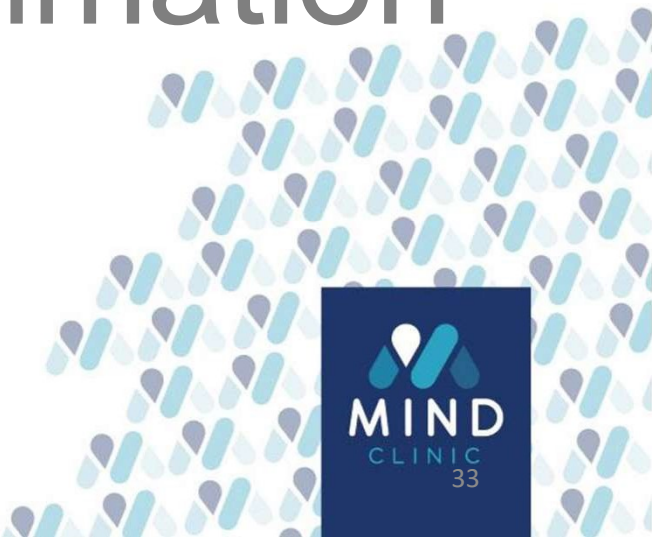
Porphyromonas gingivalis in Alzheimer's disease brains: Evidence for disease causation and treatment with small-molecule inhibitors

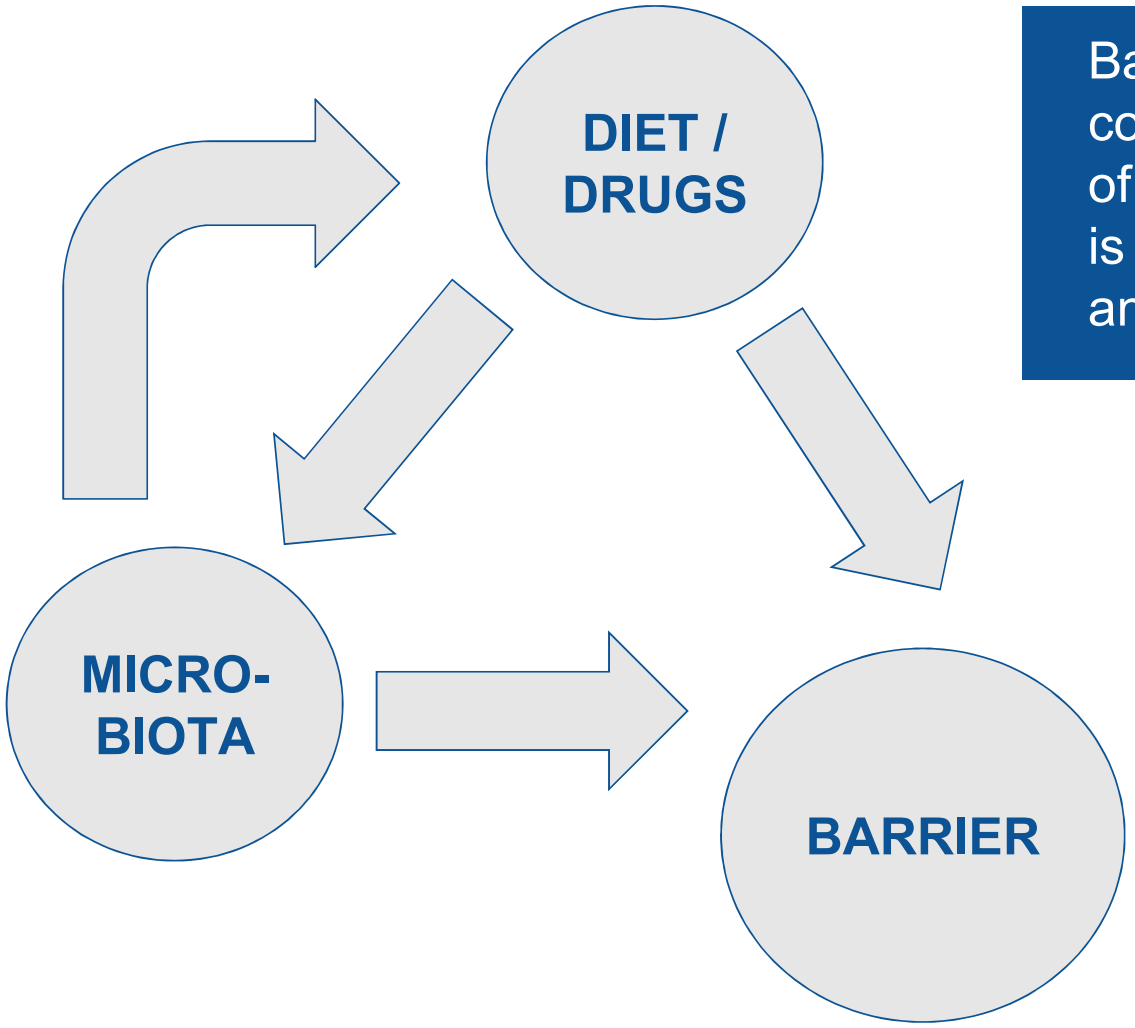
Stephen S. Dominy[1]*†, Casey Lynch[1]*, Florian Ermini[1], Malgorzata Benedyk[2,3], Agata Marczyk[2], Andrei Konradi[1], Mai Nguyen[1], Ursula Haditsch[1], Debasish Raha[1], Christina Griffin[1], Leslie J. Holsinger[1], Shirin Arastu-Kapur[1], Samer Kaba[1], Alexander Lee[1], Mark I. Ryder[4], Barbara Potempa[5], Piotr Mydel[2,6], Annelie Hellvard[3,6], Karina Adamowicz[2], Hatice Hasturk[7,8], Glenn D. Walker[9], Eric C. Reynolds[9], Richard L. M. Faull[10], Maurice A. Curtis[11,12], Mike Dragunow[11,13], Jan Potempa[2,5]*

Porphyromonas gingivalis, the keystone pathogen in chronic periodontitis, was identified in the brain of Alzheimer's disease patients. Toxic proteases from the bacterium called gingipains were also identified in the brain of Alzheimer's patients, and levels correlated with tau and ubiquitin pathology. Oral *P. gingivalis* infection in mice resulted in brain colonization and increased production of $A\beta_{1-42}$, a component of amyloid plaques. Further, gingipains were neurotoxic in vivo and in vitro, exerting detrimental effects on tau, a protein needed for normal neuronal function. To block this neurotoxicity, we designed and synthesized small-molecule inhibitors targeting gingipains. Gingipain inhibition reduced the bacterial load of an established *P. gingivalis* brain infection, blocked $A\beta_{1-42}$ production, reduced neuroinflammation, and rescued neurons in the hippocampus. These data suggest that gingipain inhibitors could be valuable for treating *P. gingivalis* brain colonization and neurodegeneration in Alzheimer's disease.

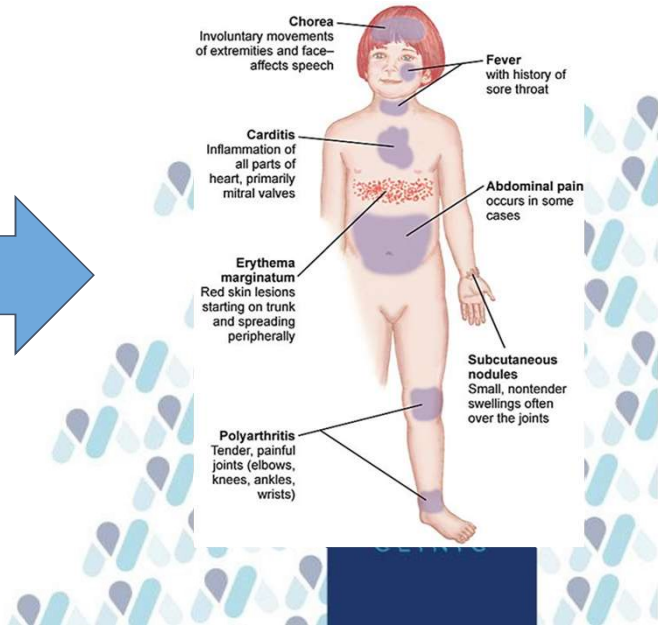

1
2
3
4
1
2
3
4
Gingiva
AD
Control
IgG1

1. Introduction
2. Microbiome
3. Barrier and inflammation
4. **Clinical practice**





Barrier function damage is the final common way in pathogenesis of diseases. Clinical manifestations is defined by nature of antigen and the MHC status of the host.



probiotic strategy: low diversity => long term administration

It will NOT restore beta diversity but will prevent invasive, inflammatory species to grow => just like good quality grass in a garden

BUT IT IS REVERSIBLE

doi:10.1038/nature12820

Diet rapidly and reproducibly alters the human gut microbiome

Nature 2014 Jan 23;505(7484):559-63

Lawrence A. David[1,2]†, Corinne F. Maurice[1], Rachel N. Carmody[1], David B. Gootenberg[1], Julie E. Button[1], Benjamin E. Wolfe[1], Alisha V. Ling[3], A. Sloan Devlin[4], Yug Varma[4], Michael A. Fischbach[4], Sudha B. Biddinger[3], Rachel J. Dutton[1] & Peter J. Turnbaugh[1]

- Diet
- Physical activity
- Alcohol abstinence
- sleep hygiene
- probiotics

Long-term dietary intake influences the structure and activity of the trillions of microorganisms residing in the human gut[1–5], but it remains unclear how rapidly and reproducibly the human gut microbiome responds to short-term macronutrient change. Here we show that the short-term consumption of diets composed entirely of animal or plant products alters microbial community structure and overwhelms inter-individual differences in microbial gene expression. The animal-based diet increased the abundance of bile-tolerant microorganisms (*Alistipes*, *Bilophila* and *Bacteroides*) and decreased the levels of Firmicutes that metabolize dietary plant polysaccharides (*Roseburia*, *Eubacterium rectale* and *Ruminococcus bromii*). Microbial activity mirrored differences between herbivorous and carnivorous mammals[2], reflecting trade-offs between carbohydrate and protein fermentation. Foodborne microbes from both diets transiently colonized the gut, including bacteria, fungi and even viruses. Finally, increases in the abundance and activity of *Bilophila wadsworthia* on the animal-based diet support a link between dietary fat, bile acids and the outgrowth of microorganisms capable of triggering inflammatory bowel disease[6]. In concert, these results demonstrate that the gut microbiome can rapidly respond to altered diet, potentially facilitating the diversity of human dietary lifestyles.

MIND CLINIC

Köszönöm a figyelmet

- Research from the 1980s demonstrated that **pets and their owners share common intestinal bacteria** (Caugant et al., 1984).
- Studies in infants and young children have shown that early life exposure to household furry pets increases richness and diversity of the human gut microbiome (Azad et al., 2013; Tun et al., 2017).
- A study published last year in The New England Journal of Medicine found that Amish children in Indiana who grew up close to barnyard animals had far lower rates of asthma.
- Research has shown that dog ownership raised the levels of different classes of bacterial species in the indoor environment

MIND
BRAIN - GUT CENTER

mailbox@schwab.nu

BIOM
TEAM