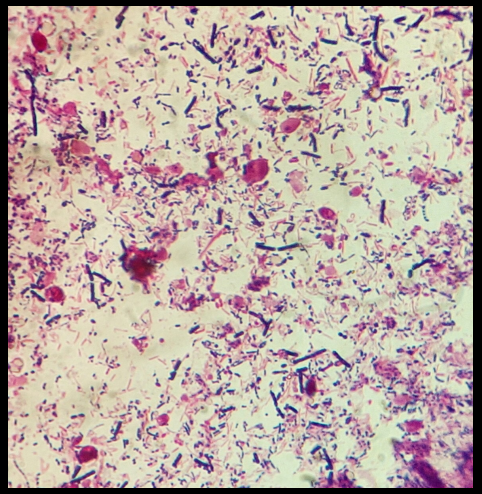
La MICROBIOTA: Mites i Realitats.



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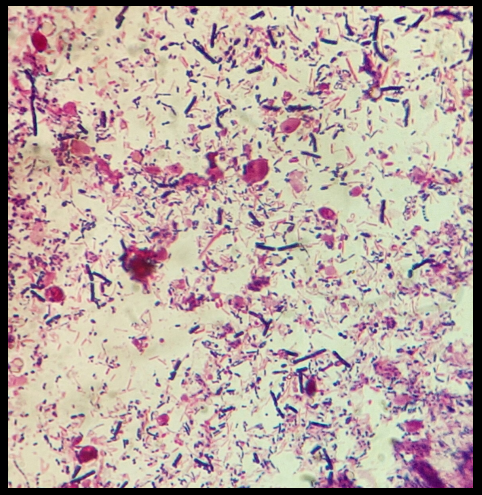
Clínic Barcelona | Universitat de Barcelona

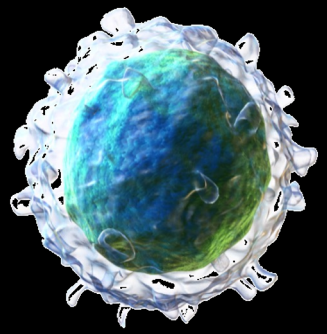
Climent Casals-Pascual, MD PhD

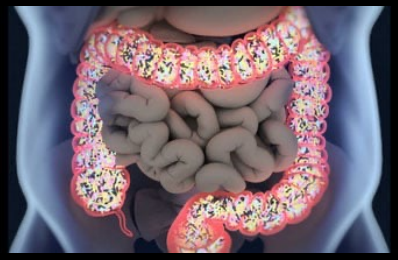
La microbiota ho explica tot?

Maria Branyes

De què parlarem?







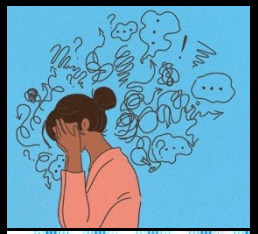
EL MICROBIOMA COM A ÒRGAN IMMUNOMODULADOR

Al final de la xerrada, haureu de ser capaços d'explicar.....

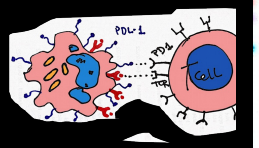
Com un tros de bròquil pot educar el nostre sistema immunitari?

Per quina raó menjar de nit ens dona inflamació crònica?

Per quina raó fracassen les dietes i per què l'índex glucèmic cal redefinir-lo?



Com pot ser que la microbiota afecti els trastorns d'angoixa o la depressió?



Com la microbiota afecta I pot millorar el tractament del càncer?

A l'inici...

NIH HUMAN MICROBIOME PROJECT

JF Kennedy , 1962 *"We choose to go to the moon in this decade ..(...), not because they are easy, but because it is hard."*

1990

Hubble

$2.700.000.000

Per a que us feu una idea...

=

People also ask

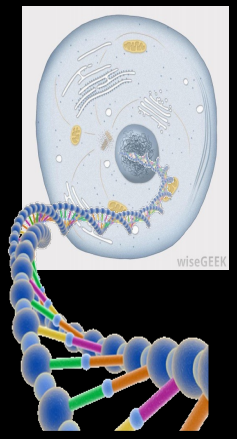
How much is FC Barcelona worth?

How much does it cost to buy FC Barcelona?

So, the building, equipment and staff plus the fact that it will produce star players, I think that it would be worth around 70 million. Also, we have to remember Camp Nou, Barcelona's stadium. When Camp Nou was built, it cost **1.7 billion euros**. Altogether, Barcelona costs **2.62 billion euros**.

Quin va ser el repte?

HUMAN GENOME PROJECT



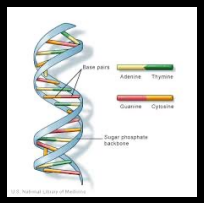
2 metres reals

d'informació

ACTCGGTCCA... $3x10^9$ lletres

Què vàrem aprendre del HGP?

2003



99.6% idèntics

98% of DNA is brossa

Les granotes tenen el doble d'ADN que els humans

Si som tan semblants genèticament,
Com s'explica
tanta variabilitat biològica?

No estem sols...

...tenim el nostre microbioma

Que és un ecosistema que coexisteix i co-evoluciona amb nosaltres

MICROBIOTA INTESTINAL

És un ÒRGAN:

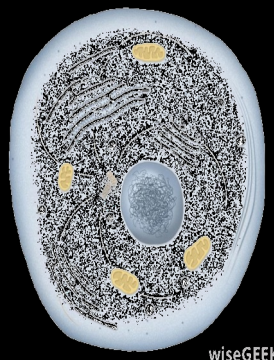
1,3 kg of bacteris

El que importa no és la quantitat sinó la seva diversitat

MICROBIOTA:

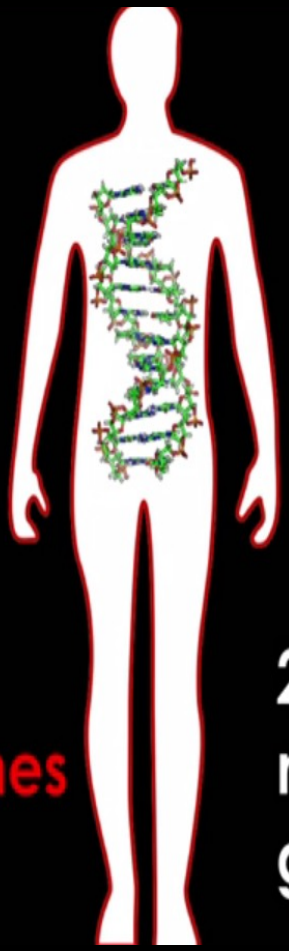
Cèl.lules humanes = Cèl.lules bacterianes

1 : 1



Som TAN BACTERIANS COM HUMANS ...

MICROBIOTA : GENÈTICAMENT ENCARA ESTEM MÉS DILUÏTS!



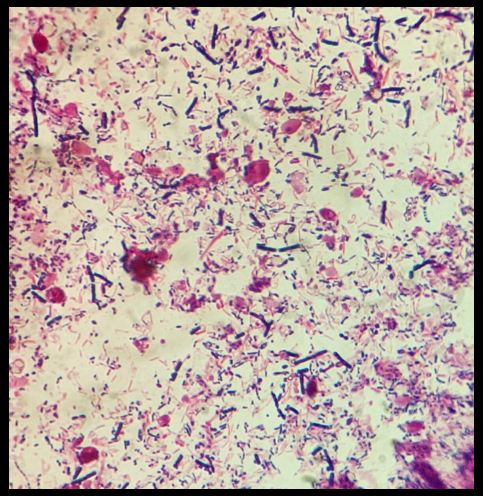
1: 100

Farmaco-microbiòmica

2008

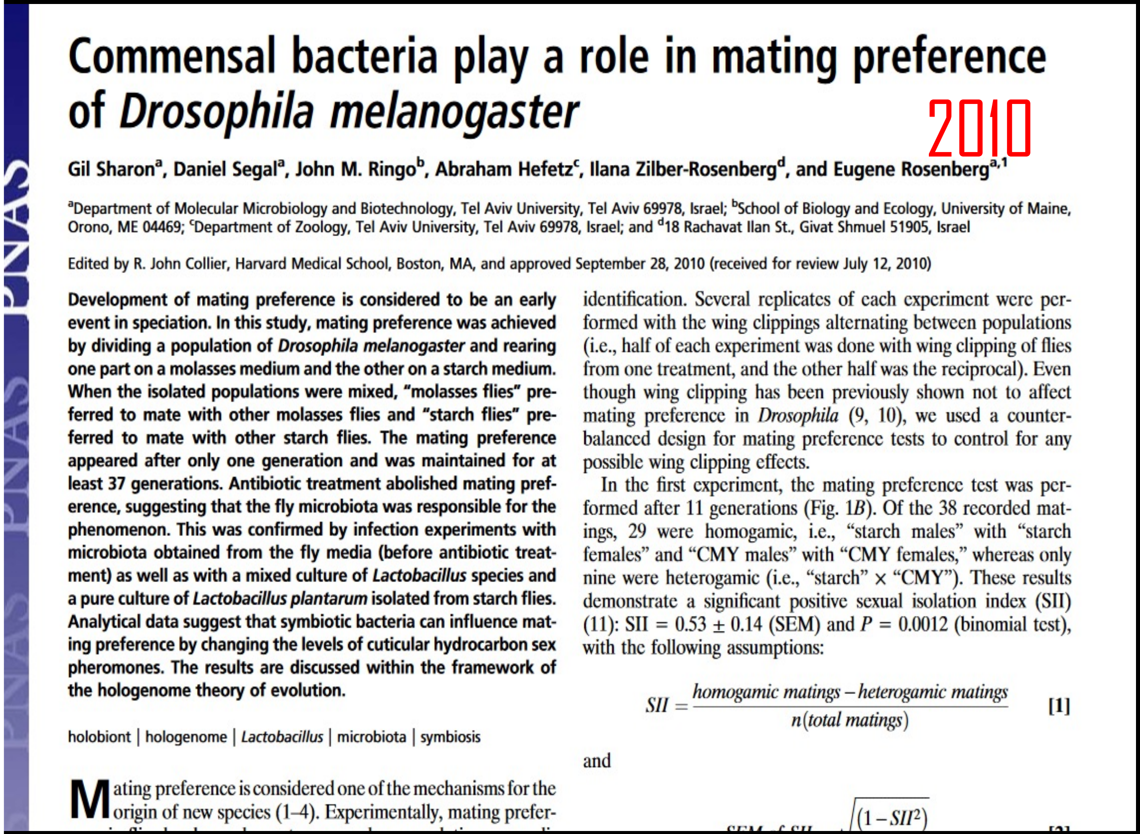
NIH HUMAN MICROBIOME PROJECT

Quins bacteris conté una MICROBIOTA SANA?



$115.000.000

I això per a què serveix?



Commensal bacteria play a role in mating preference of *Drosophila melanogaster*

2010

Gil Sharon[a], Daniel Segal[a], John M. Ringo[b], Abraham Hefetz[c], Ilana Zilber-Rosenberg[d], and Eugene Rosenberg[a,1]

[a]Department of Molecular Microbiology and Biotechnology, Tel Aviv University, Tel Aviv 69978, Israel; [b]School of Biology and Ecology, University of Maine, Orono, ME 04469; [c]Department of Zoology, Tel Aviv University, Tel Aviv 69978, Israel; and [d]18 Rachavat Ilan St., Givat Shmuel 51905, Israel

Edited by R. John Collier, Harvard Medical School, Boston, MA, and approved September 28, 2010 (received for review July 12, 2010)

Development of mating preference is considered to be an early event in speciation. In this study, mating preference was achieved by dividing a population of *Drosophila melanogaster* and rearing one part on a molasses medium and the other on a starch medium. When the isolated populations were mixed, "molasses flies" preferred to mate with other molasses flies and "starch flies" preferred to mate with other starch flies. The mating preference appeared after only one generation and was maintained for at least 37 generations. Antibiotic treatment abolished mating preference, suggesting that the fly microbiota was responsible for the phenomenon. This was confirmed by infection experiments with microbiota obtained from the fly media (before antibiotic treatment) as well as with a mixed culture of *Lactobacillus* species and a pure culture of *Lactobacillus plantarum* isolated from starch flies. Analytical data suggest that symbiotic bacteria can influence mating preference by changing the levels of cuticular hydrocarbon sex pheromones. The results are discussed within the framework of the hologenome theory of evolution.

holobiont | hologenome | *Lactobacillus* | microbiota | symbiosis

Mating preference is considered one of the mechanisms for the origin of new species (1–4). Experimentally, mating prefer-

identification. Several replicates of each experiment were performed with the wing clippings alternating between populations (i.e., half of each experiment was done with wing clipping of flies from one treatment, and the other half was the reciprocal). Even though wing clipping has been previously shown not to affect mating preference in *Drosophila* (9, 10), we used a counter-balanced design for mating preference tests to control for any possible wing clipping effects.

In the first experiment, the mating preference test was performed after 11 generations (Fig. 1*B*). Of the 38 recorded matings, 29 were homogamic, i.e., "starch males" with "starch females" and "CMY males" with "CMY females," whereas only nine were heterogamic (i.e., "starch" × "CMY"). These results demonstrate a significant positive sexual isolation index (SII) (11): SII = 0.53 ± 0.14 (SEM) and P = 0.0012 (binomial test), with the following assumptions:

$$SII = \frac{homogamic\ matings - heterogamic\ matings}{n(total\ matings)} \quad [1]$$

and

$$\ldots \sqrt{(1 - SII^2)} \ldots \quad [2]$$

el teu sex-appeal depèn de la teva microbiota..

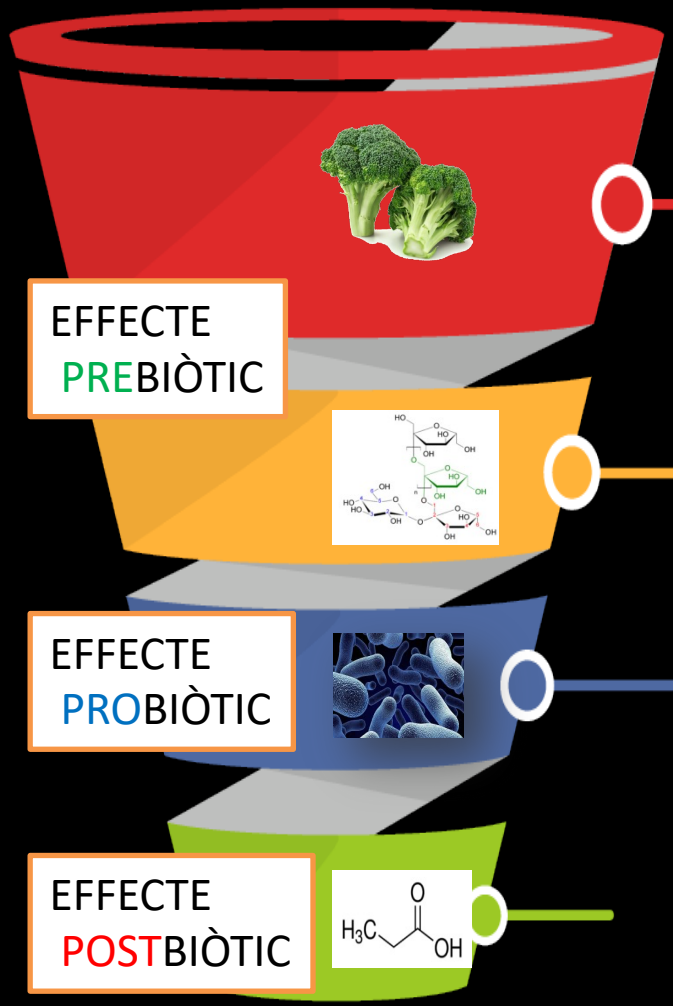
I com no, LA DIETA!!

NUTRICIÓ

MICROBIOTA INTESTINAL

SALUT

Com funciona el microbioma?



El que mengem...

EFFECTE PREBIÒTIC

El que queda de la digestió de la fibra...

EFFECTE PROBIÒTIC

El que mengen els nostres bacteris...

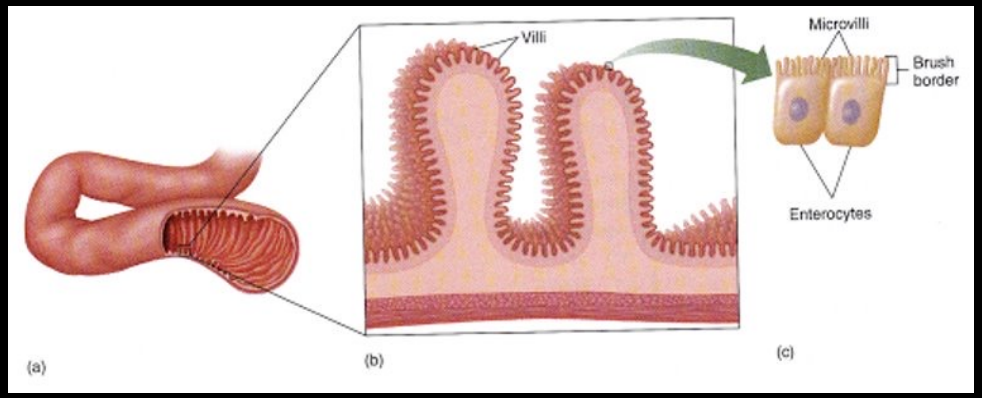
EFFECTE POSTBIÒTIC

El que ens donen els bacteris...SCFA

Impacte sobre la salut

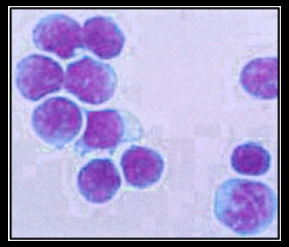
Com interacciona la microbioota intestinal amb el Sistema immune?

nutrients



Intestinal surface

Tissue	Total cell number ($\times 10^9$)
Bone marrow	50
Lamina propria of the gut	**30**

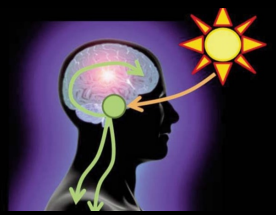


Lymphocytes in làina propria

De la fibra a la immuno tolerància....

Fibra
Pectin
Resistant starch
Cellulose
Fructan

Ritmes circadians



Per què no s'ha de menjar de nit?

Lower healthy ageing
Dose response effect

JAMA Network | Open

Original Investigation | Public Health

Rotating Night Shift Work and Healthy Aging After 24 Years of Follow-up in the Nurses' Health Study

Hongying Shi, PhD; Tianyi Huang, ScD, MSc; Eva S. Schernhammer, MD, DrPH; Qi Sun, MD, ScD; Molin Wang, PhD

Abstract

IMPORTANCE Rotating night shift work is associated with higher mortality. Whether it is also associated with overall health among those who survive to older ages remains unclear.

OBJECTIVE To examine whether rotating night shift work is associated with healthy aging after 24 years of follow-up in the Nurses' Health Study, a cohort study among registered female nurses.

DESIGN, SETTING, AND PARTICIPANTS For this cohort study, a compos phenotype was ascertained among 46 318 participants who were aged 4 major chronic diseases in 1988 when the history of night shift work was a analysis in which cognitive function decline was considered in the healthy nurses were involved. Data were analyzed from March 1 to September 30

EXPOSURES Duration of rotating night shift work.

MAIN OUTCOMES AND MEASURES Healthy aging was defined as reach and being free of 11 major chronic diseases, memory impairment, physical mental health.

RESULTS Of 46 318 female nurses (mean [SD] age at baseline, 55.4 [6.1] achieved healthy aging after 24 years of follow-up. After adjusting for esta confounders, compared with women who never worked rotating night sh healthy aging decreased significantly with increasing duration of night sh were 0.96 (95% CI, 0.89-1.03) for 1 to 5 years, 0.92 (95% CI, 0.79-1.07) fo (95% CI, 0.69-0.91) for 10 or more years of night shift work (P = .001 for t not differ substantially by age and lifestyles and was consistent for 4 indivi aging. Results were similar in a secondary analysis, with an odds ratio of 0 P < .001 for trend) comparing 10 or more years of night shift work vs no ni

CONCLUSIONS AND RELEVANCE In this cohort study, rotating night shi decreased probability of healthy aging among US female nurses. These da excess night shift work is a significant health concern that may also lead to among older individuals.

JAMA Network Open. 2022;5(5):e2210450.
Corrected on June 1, 2022. doi:10.1001/jamanetworkopen.2022.10450

Key Points

Question Is rotating night shift work prospectively associated with healthy aging that simultaneously considers chronic diseases, cognitive and physical function, and mental health?

Findings In a large cohort study of

MAIN OUTCOMES AND MEASURES Healthy aging was defined as reaching at least 70 years of age and being free of 11 major chronic diseases, memory impairment, physical limitation, or deteriorated mental health.

RESULTS Of 46 318 female nurses (mean [SD] age at baseline, 55.4 [6.1] years), 3695 (8.0%) achieved healthy aging after 24 years of follow-up. After adjusting for established and potential confounders, compared with women who never worked rotating night shifts, the odds of achieving healthy aging decreased significantly with increasing duration of night shift work. The odds ratios were 0.96 (95% CI, 0.89-1.03) for 1 to 5 years, 0.92 (95% CI, 0.79-1.07) for 6 to 9 years, and 0.79 (95% CI, 0.69-0.91) for 10 or more years of night shift work (P = .001 for trend). This association did not differ substantially by age and lifestyles and was consistent for 4 individual dimensions of healthy aging. Results were similar in a secondary analysis, with an odds ratio of 0.73 (95% CI, 0.60-0.89; P < .001 for trend) comparing 10 or more years of night shift work vs no night shift work.

CONCLUSIONS AND RELEVANCE In this cohort study, rotating night shift work was associated with decreased probability of healthy aging among US female nurses. These data support the notion that excess night shift work is a significant health concern that may also lead to deteriorated overall health among older individuals.

JAMA Network Open. 2022;5(5):e2210450.
Corrected on June 1, 2022. doi:10.1001/jamanetworkopen.2022.10450

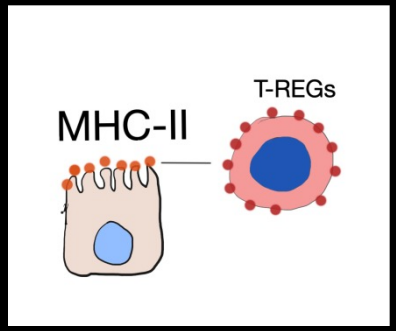
associated with increased mortality
findings of this study further sugge
that shift work is also associated wi
worse overall health among women
survive to older ages.

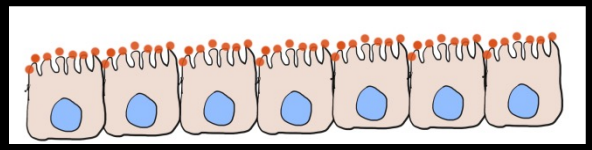
+ Supplemental content

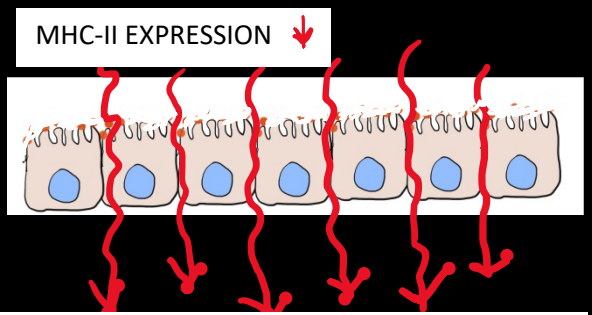
Author affiliations and article information
listed at the end of this article.

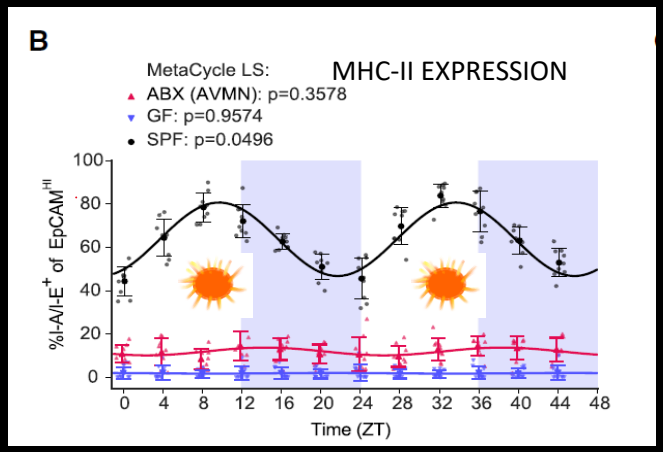
Ritmes circadians i microbiota intestinal

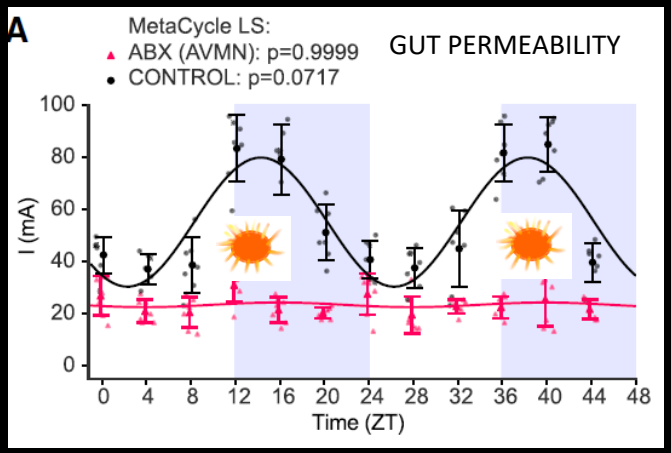
Per què no s'ha de menjar de nit?











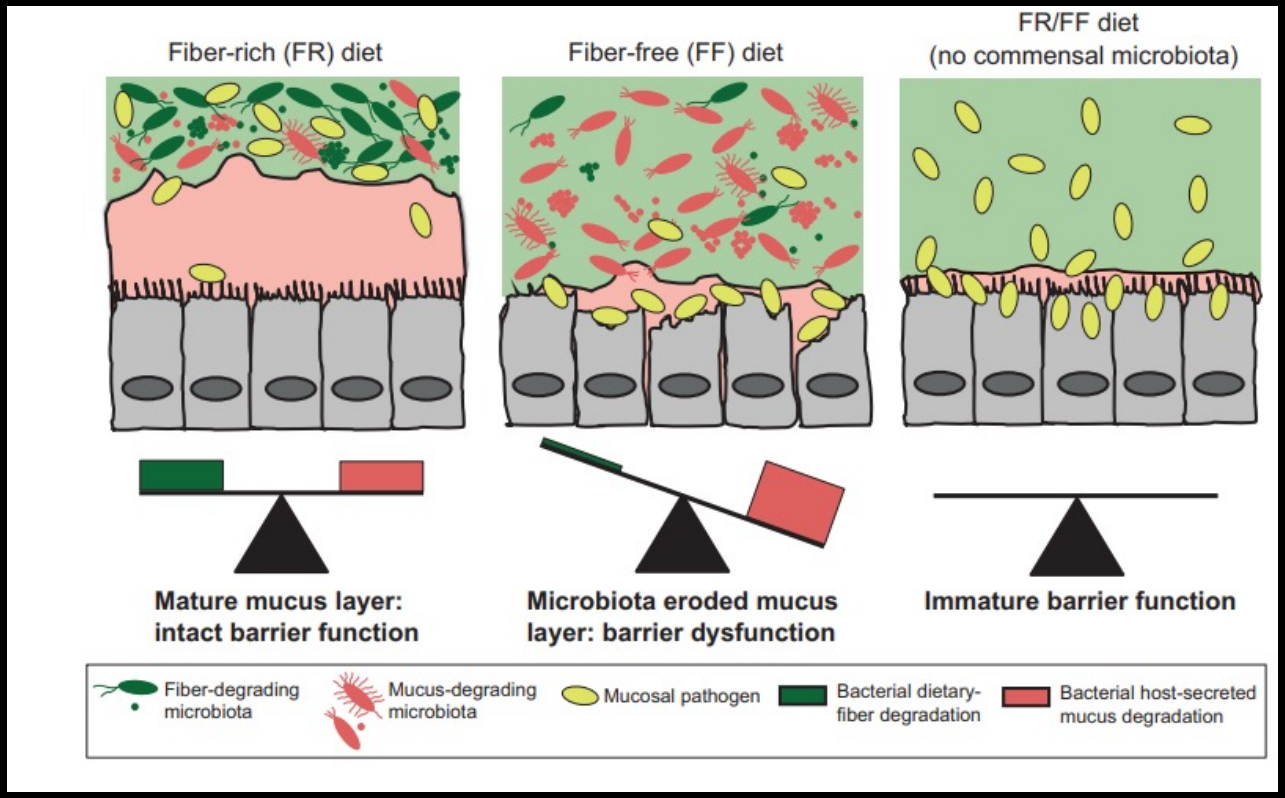
Alerta:

No hi ha bacteris bons o dolents...

Article

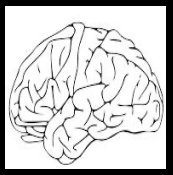
Cell

A Dietary Fiber-Deprived Gut Microbiota Degrades the Colonic Mucus Barrier and Enhances Pathogen Susceptibility



BRAIN-GUT AXIS

El segon cervell?

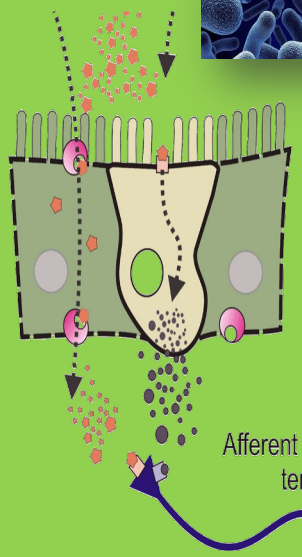


En le budell tenim:

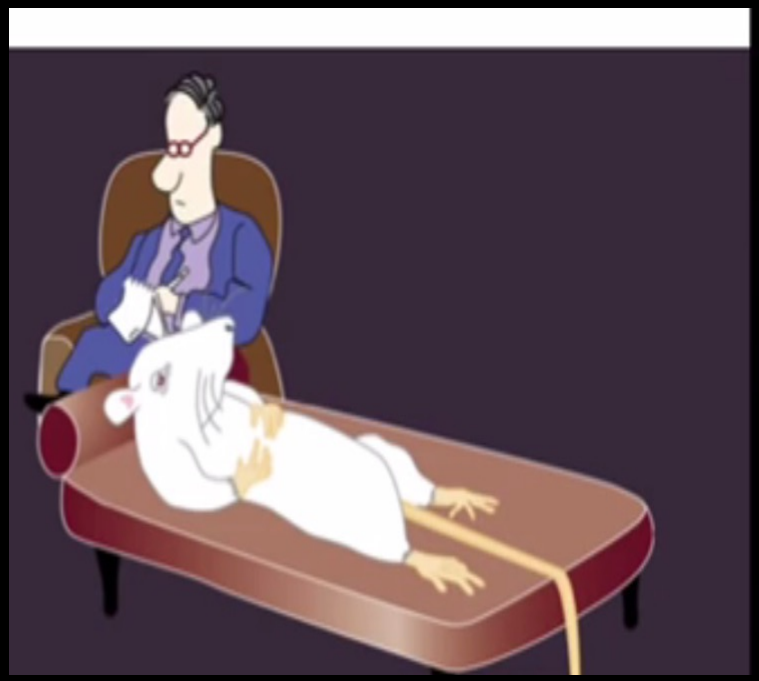
10^8 neurones (cervell: 10^{11})

80% of immune cells!

90% de la serotonina
(en el cervell<10% producció)

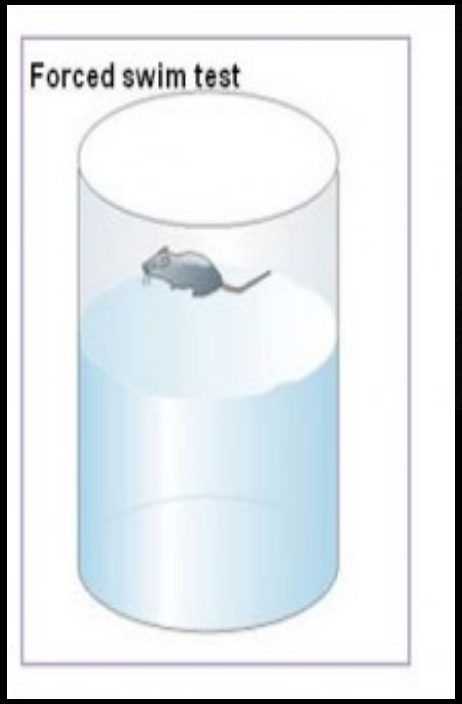


BRAIN-GUT AXIS: psicobiòtics

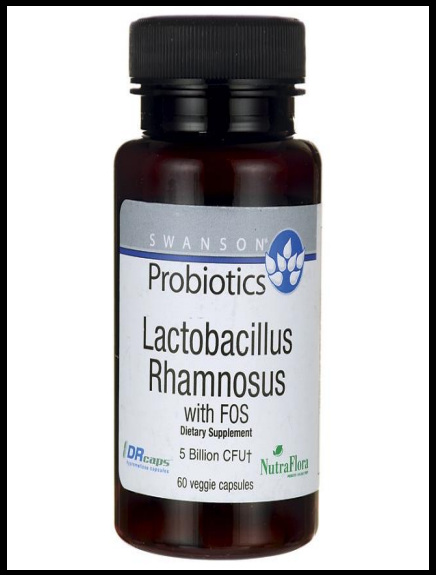


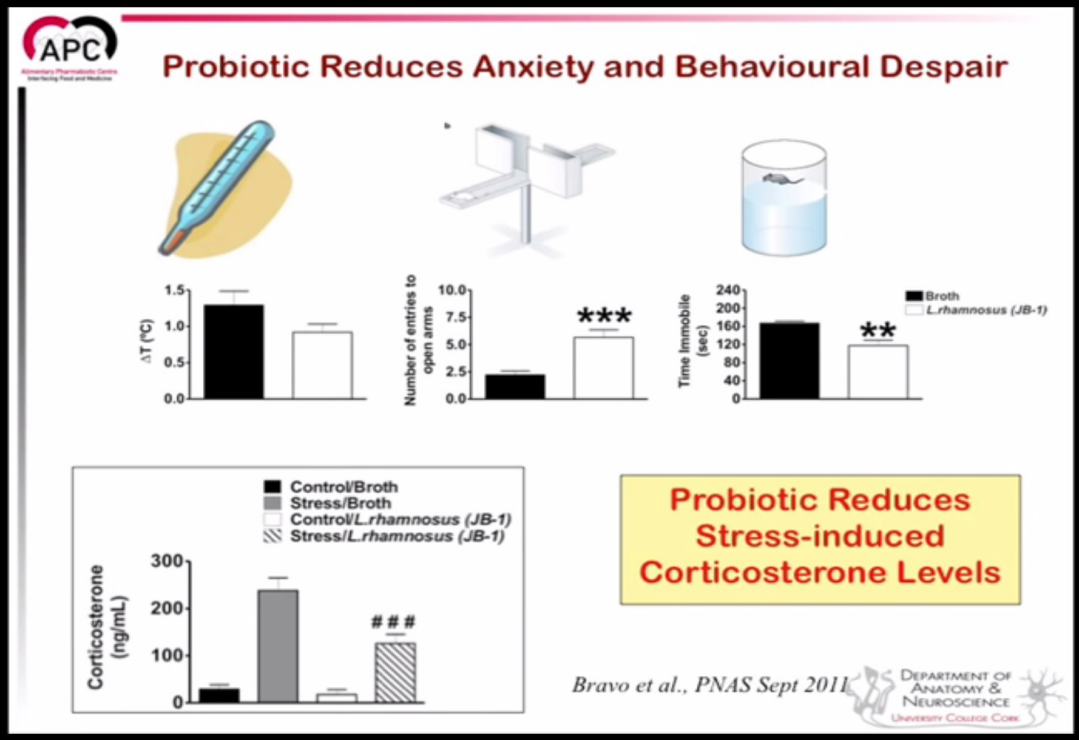
BRAIN-GUT AXIS

PODEM ESTUDIAR LA DEPRESSIÓ EN MODEL ANIMALS?

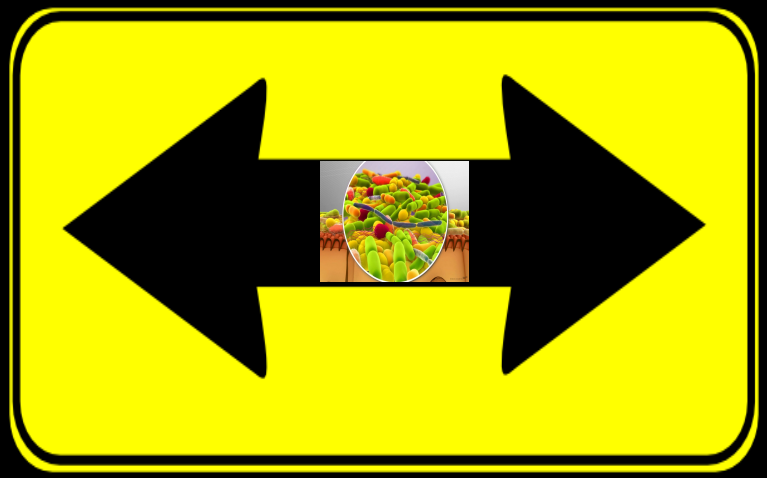


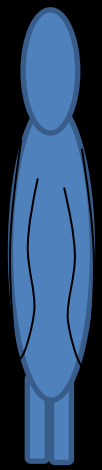
BRAIN-GUT AXIS

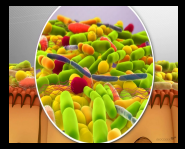


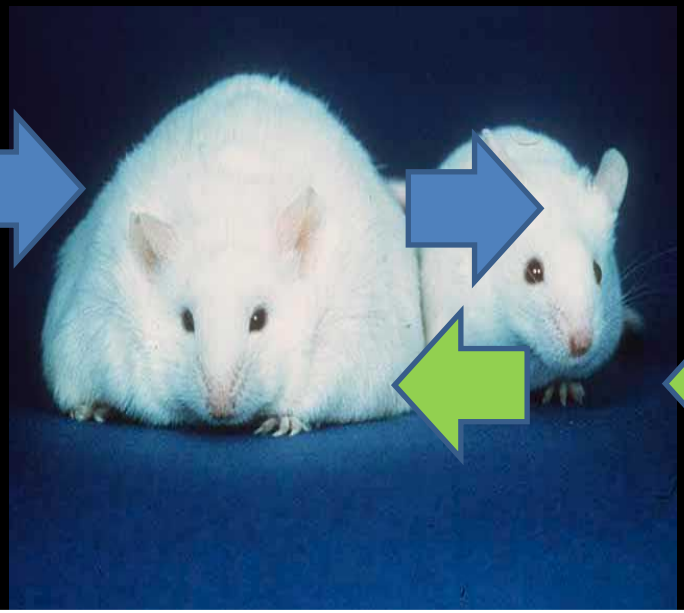


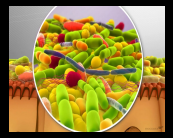
OBESITAT

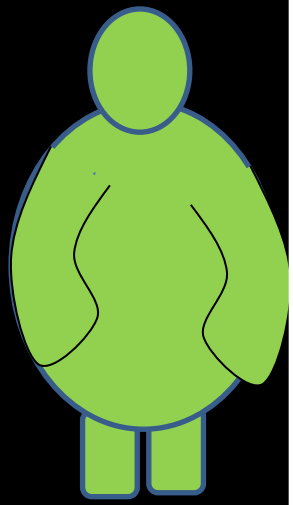








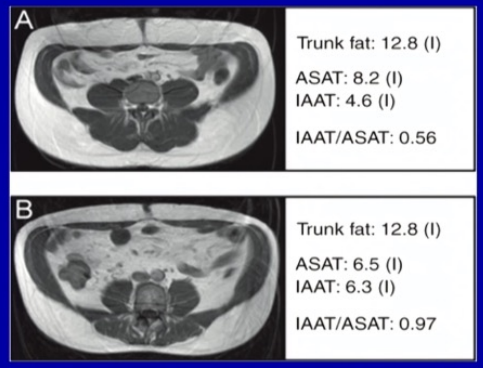




OBESITY

People tend to regain their weight after a successful

Relation between visceral and subcutaneous obesity:
TOFI (thin on the outside, fat on the inside)



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Body-Weight Fluctuations and Outcomes in Coronary Disease

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ABSTRACT

BACKGROUND
Body-weight fluctuation is a risk factor for death and coronary events in patients without cardiovascular disease. It is not known whether variability in body weight affects outcomes in patients with coronary artery disease.

METHODS
We determined intraindividual fluctuations in body weight from baseline weight and follow-up visits and performed a post hoc analysis of the Treating to New Targets trial, which involved assessment of the efficacy and safety of lowering low-density lipoprotein cholesterol levels with atorvastatin. The primary outcome was any coronary event (a composite of death from coronary heart disease, nonfatal myocardial infarction, resuscitated cardiac arrest, revascularization, or angina). Secondary outcomes were any cardiovascular event (a composite of any coronary event, a cerebrovascular event, peripheral vascular disease, or heart failure), death, myocardial infarction, or stroke.

RESULTS
Among 9509 participants, after adjustment for risk factors, baseline lipid levels, mean body weight, and weight change, each increase of 1 SD in body-weight variability (measured according to average successive variability and used as a time-dependent covariate) was associated with an increase in the risk of any coronary event (2091 events; hazard ratio, 1.04; 95% confidence interval [CI], 1.01 to 1.07; P=0.01), any cardiovascular event (2727 events; hazard ratio, 1.04; 95% CI, 1.02 to 1.07; P<0.001), and death (487 events; hazard ratio, 1.09; 95% CI, 1.07 to 1.12; P<0.001). Among patients in the quintile with the highest variation in body weight, the risk of a coronary event was 64% higher, the risk of a cardiovascular event 85% higher, death 124% higher, myocardial infarction 117% higher, and stroke 136% higher than it was among those in the quintile with the lowest variation in body weight in adjusted models.

CONCLUSIONS
Among participants with coronary artery disease, fluctuation in body weight was associated with higher mortality and a higher rate of cardiovascular events independent of traditional cardiovascular risk factors. (Funded by Pfizer; ClinicalTrials.gov number, NCT00327691.)

OBESITY

Published: 24 November 2016

Persistent microbiome alterations modulate the rate of post-dieting weight regain

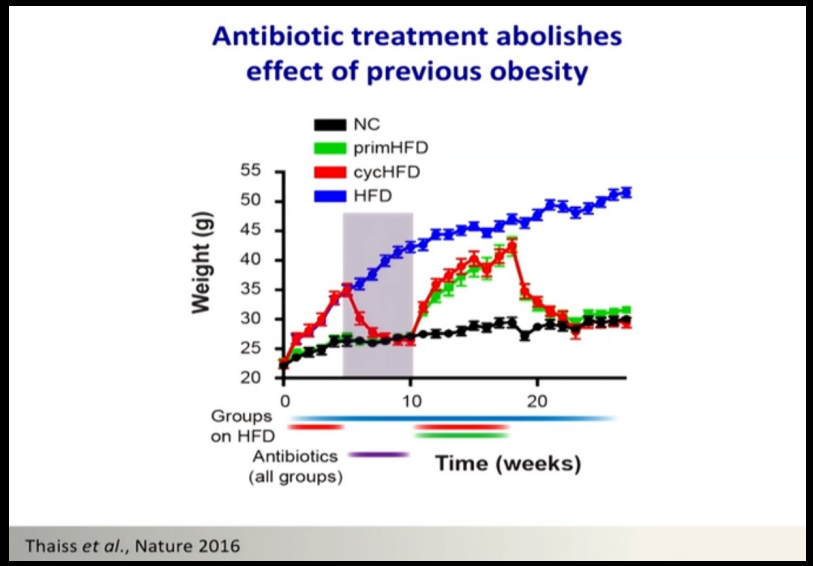
Christoph A. Thaiss, Shlomik Itav, Daphna Rothschild, Mariska T. Meijer, Maayan Levy, Claudia Moresi, Lenka Dohnalová, Sofia Braverman, Shachar Rozin, Sergey Malitsky, Mally Dori-Bachash, Yael Kuperman, Inbal Biton, Arieh Gertler, Alon Harmelin, Hagit Shapiro, Zamir Halpern, Asaph Aharoni, Eran Segal & Eran Elinav

Nature **540**, 544–551(2016) | Cite this article

6725 Accesses | **207** Citations | **780** Altmetric | Metrics

Abstract

In tackling the obesity pandemic, considerable efforts are devoted to the development of effective weight reduction strategies, yet many dieting individuals fail to maintain a long-term weight reduction, and instead undergo excessive weight regain cycles. The mechanisms driving recurrent post-dieting obesity remain largely elusive. Here we identify an intestinal microbiome signature that persists after successful dieting of obese mice and contributes to faster weight regain and metabolic aberrations upon re-exposure to obesity-promoting conditions. Faecal transfer experiments show that the accelerated weight regain phenotype can be transmitted to germ-free mice. We develop a machine-learning algorithm that enables personalized microbiome-based prediction of the extent of post-dieting weight regain. Additionally, we find that the microbiome contributes to diminished post-dieting flavonoid levels and reduced energy expenditure, and demonstrate that flavonoid-based 'post-biotic' intervention ameliorates excessive secondary weight gain. Together, our data highlight a possible microbiome contribution to accelerated post-dieting weight regain, and suggest that microbiome-targeting approaches may help to diagnose and treat this common disorder.



Conclusions:

Hi ha"memòria d'obesitat" que depèn de la microbiota intestinal

Efecte "yo-yo" es pot prevenir o modificar amb antibiòtics

Effecte "yo-yo" es pot eliminar amb alguns post-biòtics: flavonoids (apigenin i naringin)

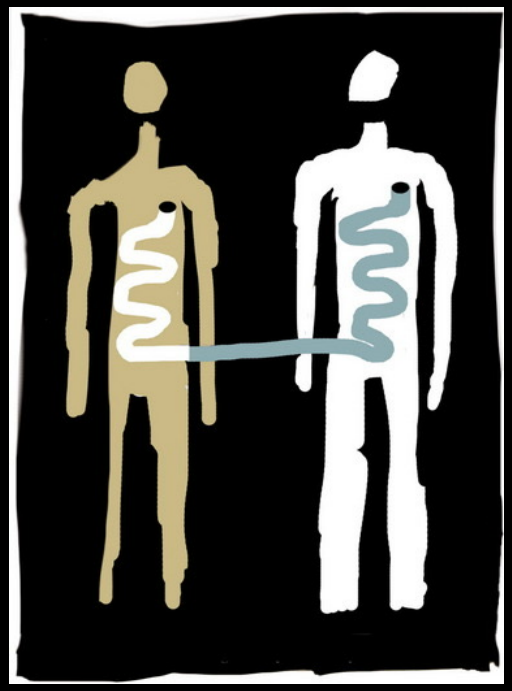
julivert

pomelo


THEORY
INTO
PRACTICE

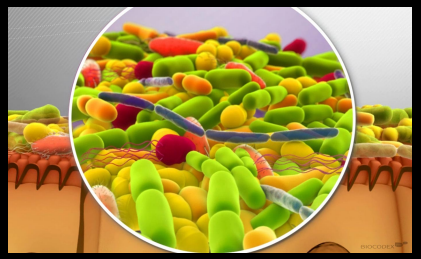
Què tenen en comú aquests animals?

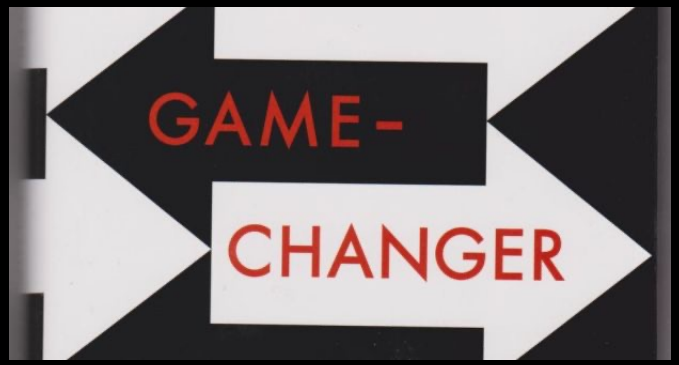


Trasplantament Fecal

Com funciona?





The NEW ENGLAND JOURNAL of MEDICINE

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Duodenal Infusion of Donor Feces for Recurrent *Clostridium difficile*

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ABSTRACT

BACKGROUND

Recurrent *Clostridium difficile* infection is difficult to treat, and failure rates for antibiotic therapy are high. We studied the effect of duodenal infusion of donor feces in patients with recurrent *C. difficile* infection.

METHODS

We randomly assigned patients to receive one of three therapies: an initial vancomycin regimen (500 mg orally four times per day for 4 days), followed by bowel lavage and subsequent infusion of a solution of donor feces through a nasoduodenal tube; a standard vancomycin regimen (500 mg orally four times per day for 14 days); or a standard vancomycin regimen with bowel lavage. The primary end point was the resolution of diarrhea associated with *C. difficile* infection without relapse after 10 weeks.

RESULTS

The study was stopped after an interim analysis. Of 16 patients in the infusion group, 13 (81%) had resolution of *C. difficile*–associated diarrhea after the first infusion. The 3 remaining patients received a second infusion with feces from a different donor, with resolution in 2 patients. Resolution of *C. difficile* infection occurred in 4 of 13 patients (31%) receiving vancomycin alone and in 3 of 13 patients (23%) receiving vancomycin with bowel lavage ($P<0.001$ for both comparisons with the infusion group). No significant differences in adverse events among the three study groups were observed except for mild diarrhea and abdominal cramping in the infusion group on the infusion day. After donor-feces infusion, patients showed increased fecal bacterial diversity, similar to that in healthy donors, with an increase in Bacteroidetes species and clostridium clusters IV and XIVa and a decrease in Proteobacteria species.

CONCLUSIONS

The infusion of donor feces was significantly more effective for the treatment of recurrent *C. difficile* infection than the use of vancomycin. (Funded by the Netherlands Organization for Health Research and Development and the Netherlands Organization for Scientific Research; Netherlands Trial Register number, NTR1177.)

From the Departments of Internal Medicine (E.N., A.V., M.N., P.S.), Microbiology (C.E.V.), Gastroenterology (J.F.W.M.B., J.J.K.), and Cardiology (J.G.P.T.) and the Clinical Research Unit (M.G.W.D.), Academic Medical Center, University of Amsterdam, Amsterdam; the Laboratory of Microbiology, Wageningen University, Wageningen (S.F., E.G.Z., W.M.V.); the Department of Experimental and Medical Microbiology, Leiden University Medical Center, Leiden (E.J.K.); and the Department of Gastroenterology, Hagaziekenhuis, The Hague (J.J.K.) — all in the Netherlands; and the Department of Bacteriology and Immunology, Medical Faculty, University of Helsinki, Helsinki (W.M.V.). Address reprint requests to Dr. Keller at the Academic Medical Center, Department of Gastroenterology, Meibergdreef 9, 1105 AZ Amsterdam, the Netherlands, or at keller@hagaziekenhuis.nl.

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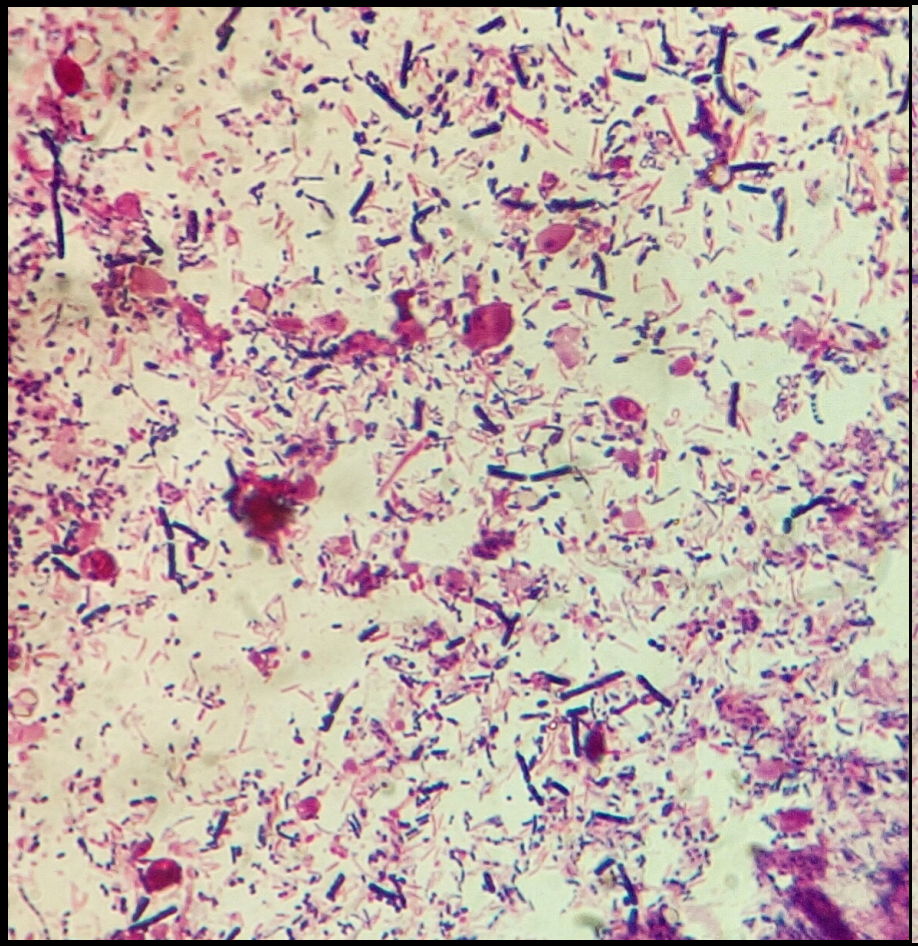
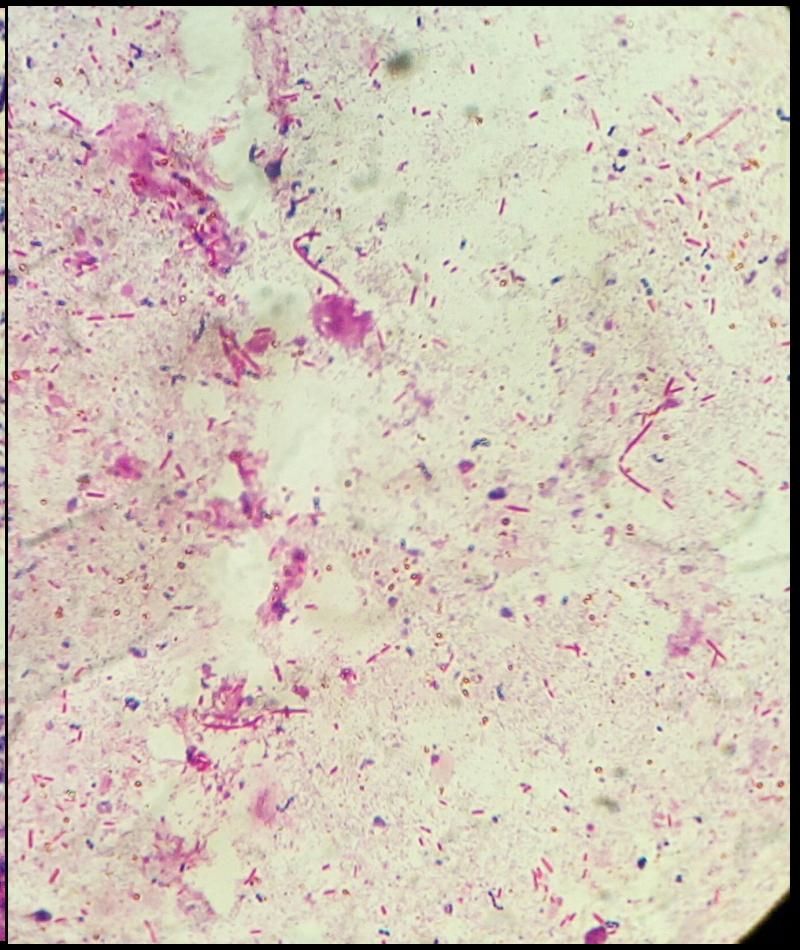
FEMTA NORMAL



FEMTA DISBIÒTICA/ C. DIFFICILE



OBESITAT

JAMA Network | Open

Original Investigation | Diabetes and Endocrinology

Effects of Fecal Microbiome Transfer in Adolescents With Obesity
The Gut Bugs Randomized Controlled Trial

Karen S. W. Leong, MBBS; Thilini N. Jayasinghe, PhD; Brooke C. Wilson, MSc; José G. B. Derraik, PhD; Benjamin B. Albert, PhD; Valentina Chiavaroli, PhD; Darren M. Svirskis, PhD; Kathryn L. Beck, PhD; Cathryn A. Conlon, PhD; Yannan Jiang, PhD; William Schierding, PhD; Tommi Vatanen, PhD; David J. Holland, PhD; Justin M. O'Sullivan, PhD; Wayne S. Cutfield, MD

Abstract

IMPORTANCE Treatment of pediatric obesity is challenging. Preclinical studies in mice indicated that weight and metabolism can be altered by gut microbiome manipulation.

OBJECTIVE To assess efficacy of fecal microbiome transfer (FMT) to treat adolescent obesity and improve metabolism.

DESIGN, SETTING, AND PARTICIPANTS This randomized, double-masked, placebo-controlled trial (October 2017-March 2019) with a 26-week follow-up was conducted among adolescents aged 14 to 18 years with a body mass index (BMI; calculated as weight in kilograms divided by height in meters squared) of 30 or more in Auckland, New Zealand. A total of 87 individuals took part—565 individuals responded to advertisements, 328 were ineligible, and 150 declined participation. Clinical data were analyzed from September 2019 to May 2020.

INTERVENTIONS Single course of oral encapsulated fecal microbiome from 4 healthy lean donors of the same sex or saline placebo.

MAIN OUTCOMES AND MEASURES Primary outcome was BMI standard deviation score at 6 weeks using intention-to-treat analysis. Secondary outcomes included body composition, cardiometabolic parameters, well-being, and gut microbiome composition.

RESULTS Eighty-seven participants (59% female adolescents, mean [SD] age 17.2 [1.4] years) were randomized 1:1, in groups stratified by sex, to FMT (42 participants) or placebo (45 participants). There was no effect of FMT on BMI standard deviation score at 6 weeks (adjusted mean difference [aMD] −0.026; 95% CI −0.074, 0.022). Reductions in android-to-gynoid-fat ratio in the FMT vs placebo group were observed at 6, 12, and 26 weeks, with aMDs of −0.021 (95% CI, −0.041 to −0.001), −0.023 (95% CI, −0.043 to −0.003), and −0.029 (95% CI, −0.049 to −0.008), respectively. There were no observed effects on insulin sensitivity, liver function, lipid profile, inflammatory markers, blood pressure, total body fat percentage, gut health, and health-related quality of life. Gut microbiome profiling revealed a shift in community composition among the FMT group, maintained up to 12 weeks. In post-hoc exploratory analyses among participants with metabolic syndrome at baseline, FMT led to greater resolution of this condition (18 to 4) compared with placebo (13 to 10) by 26 weeks (adjusted odds ratio, 0.06; 95% CI, 0.01-0.45; P = .007). There were no serious adverse events recorded throughout the trial.

CONCLUSIONS AND RELEVANCE In this randomized clinical trial of adolescents with obesite, there was no effect of FMT on weight loss in adolescents with obesity, although a reduction in abdominal adiposity was observed. Post-hoc analyses indicated a resolution of undiagnosed metabolic

Key Points

Question Will fecal microbiome transfer (FMT) lead to weight loss among adolescents with obesity?

Findings In this randomized clinical trial of 87 adolescents with obesity, FMT alone did not lead to weight loss at 6 weeks. FMT alone was associated with a reduction in android-to-gynoid-fat ratio sustained for at least 26 weeks, particularly in female adolescents; changes in the overall gut microbiome composition; and a resolution of metabolic syndrome by 26 weeks in participants who had this undiagnosed condition at baseline.

Meaning FMT alone is not an effective treatment for weight loss but may reduce visceral adiposity and improve health.

+ Visual Abstract

+ Supplemental content

Author affiliations and article information are listed at the end of this article.

Obesitat i Diabetis

1981 Índex glucèmic

We measure the glycemic curve after giving a set amount of carbohydrates

Glycemic index of foods: a physiological basis for carbohydrate exchange[1-3]

David J. A. Jenkins,[4] D.M., Thomas M. S. Wolever,[5] M.Sc., Rodney H. Taylor,[6] M.R.C.P., Helen Barker, B.Sc.,[6] S.R.D., Hashmein Fielden,[6] S.R.N., Janet M. Baldwin,[6] M.R.C.P., Allen C. Bowling,[5] Hillary C. Newman,[5] B.A., Alexandra L. Jenkins,[5] and David V. Goff,[5] M.Biol.

ABSTRACT To determine the effect of different foods on the blood glucose, 62 commonly eaten foods and sugars were fed individually to groups of 5 to 10 healthy fasting volunteers. Blood glucose levels were measured over 2 h, and expressed as a percentage of the area under the glucose response curve when the same amount of carbohydrate was taken as glucose. The largest rises were seen with vegetables (70 ± 5%), followed by breakfast cereals (65 ± 5%), cereals and biscuits (60 ± 3%), fruit (50 ± 5%), dairy products (35 ± 1%), and dried legumes (31 ± 3%). A significant negative relationship was seen between fat ($p < 0.01$) and protein ($p < 0.001$) and postprandial glucose rise but not with fiber or sugar content. *Am. J. Clin. Nutr.* 34: 362–366, 1981.

KEY WORDS Carbohydrate exchange, dietary carbohydrate, dietary fiber, blood glucose, diabetes

A significant negative relationship was seen between fat ($r = -0.386$, $p < 0.01$) and protein ($r = -0.523$, $p < 0.001$) content of the foods and the glycemic index (**Fig. 2**). There was, however, no relationship between glycemic index and dietary fiber or sugar content.

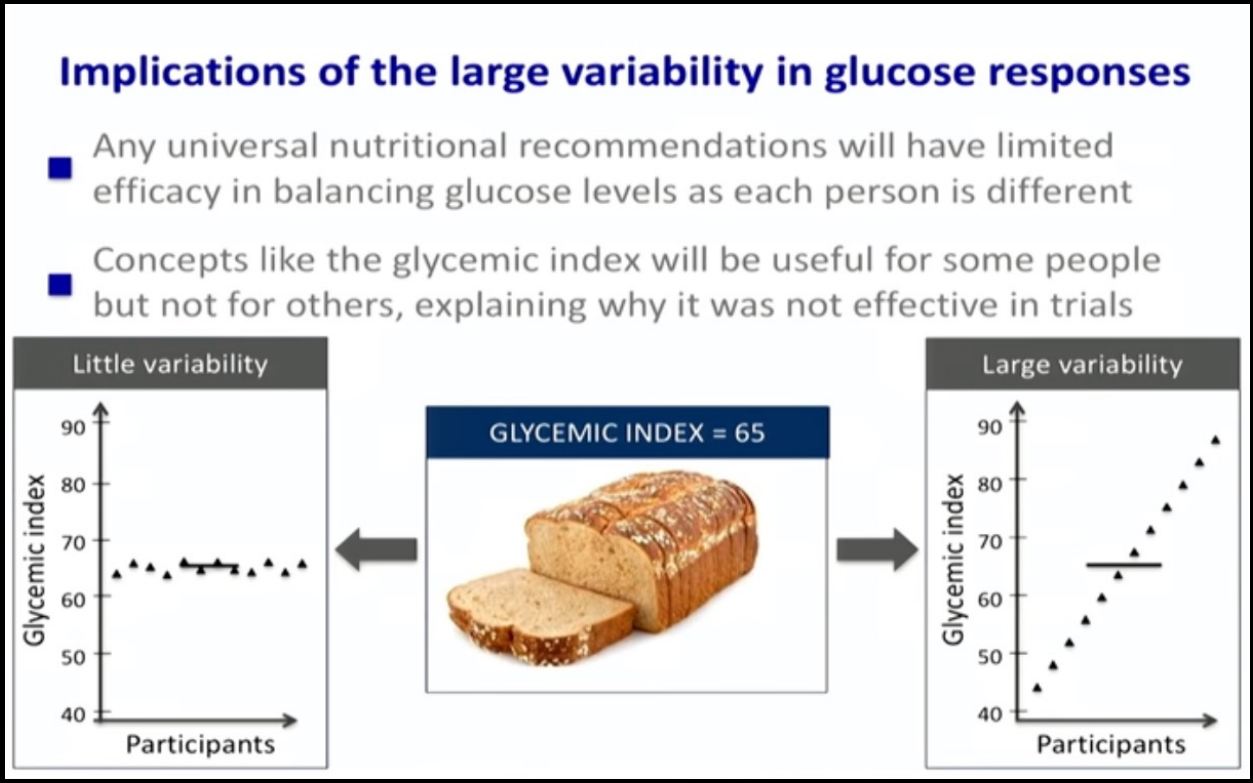
TABLE 1
Glycemic index: the area under the blood glucose response curve for each food expressed as a percentage of the area after taking the same amount of carbohydrate as glucose (result are means of 5 to 10 individuals)

Grain, cereal products			Vegetables			Fruit		
Buckwheat	51 ± 10*	(5)	Broad beans (25)¶	79 ± 16	(6)	Apples (golden delicious)	39 ± 3†	(6)
Bread (white)	69 ± 5†	(10)	Frozen peas	51 ± 6†	(6)	Banana	62 ± 9*	(6)
Bread (wholemeal)	72 ± 6†	(10)	Root Vegetables			Oranges	40 ± 3†	(6)
Millet	71 ± 10‡	(5)	Beetroot (25)¶	64 ± 16	(5)	Orange juice	46 ± 6†	(6)
Pastry	59 ± 6*	(5)	Carrots (25)¶	92 ± 20	(5)	Raisins	64 ± 11‡	(6)
Rice (brown)	66 ± 5†	(7)	Parsnips (25)¶	97 ± 19	(5)			
Rice (white)	72 ± 9§	(7)	Potato (instant)	80 ± 13	(8)	Sugars		
Spaghetti (wholemeal)	42 ± 4†	(6)	Potato (new)	70 ± 8*	(8)	Fructose	20 ± 5†	(5)
Spaghetti (white)	50 ± 8|	(6)	Potato (sweet)	48 ± 6†	(5)	Glucose	100 ±	(35)
Sponge cake	46 ± 6†	(5)	Swede (25)¶	72 ± 8‡	(5)	Maltose	105 ± 12	(6)
Sweetcorn	59 ± 11§	(5)	Yam	51 ± 12§	(5)	Sucrose	59 ± 10§	(5)
			Dried legumes			Dairy products		
Breakfast cereals			Beans (tinned, baked)	40 ± 3†	(7)	Ice cream	36 ± 8|	(5)
All-Bran	51 ± 5†	(6)	Beans (butter)	36 ± 4†	(6)	Milk (skim)	32 ± 5†	(6)
Cornflakes	80 ± 6‡	(6)	Beans (haricot)	31 ± 6†	(6)	Milk (whole)	34 ± 6†	(6)
Meusli	66 ± 9§	(6)	Beans (kidney)	29 ± 8†	(6)	Yoghurt	36 ± 4†	(5)
Porridge Oats	49 ± 8|	(6)	Beans (soya)	15 ± 5†	(7)			
Shredded Wheat	67 ± 10‡	(6)	Beans (tinned, soya)	14 ± 2†	(7)	Miscellaneous		
Wheatabix	75 ± 10‡	(6)	Peas (blackeye)	33 ± 4†	(6)	Fish fingers	38 ± 6†	(5)
			Peas (chick)	36 ± 5†	(6)	Honey	87 ± 8	(6)
Biscuits			Peas (marrowfat)	47 ± 3†	(6)	Lucozade	95 ± 10	(5)
Digestives	59 ± 7*	(6)	Lentils	29 ± 3†	(7)	Mars bar	68 ± 12‡	(6)
Oatmeal	54 ± 4†	(6)				Peanuts (25)¶	13 ± 6†	(5)
Rich Tea	55 ± 4†	(6)				Potato crisps	51 ± 7†	(6)
Ryvita	69 ± 10‡	(7)				Sausages	28 ± 6†	(5)
Water	63 ± 9*	(6)				Tomato soup	38 ± 9*	(5)

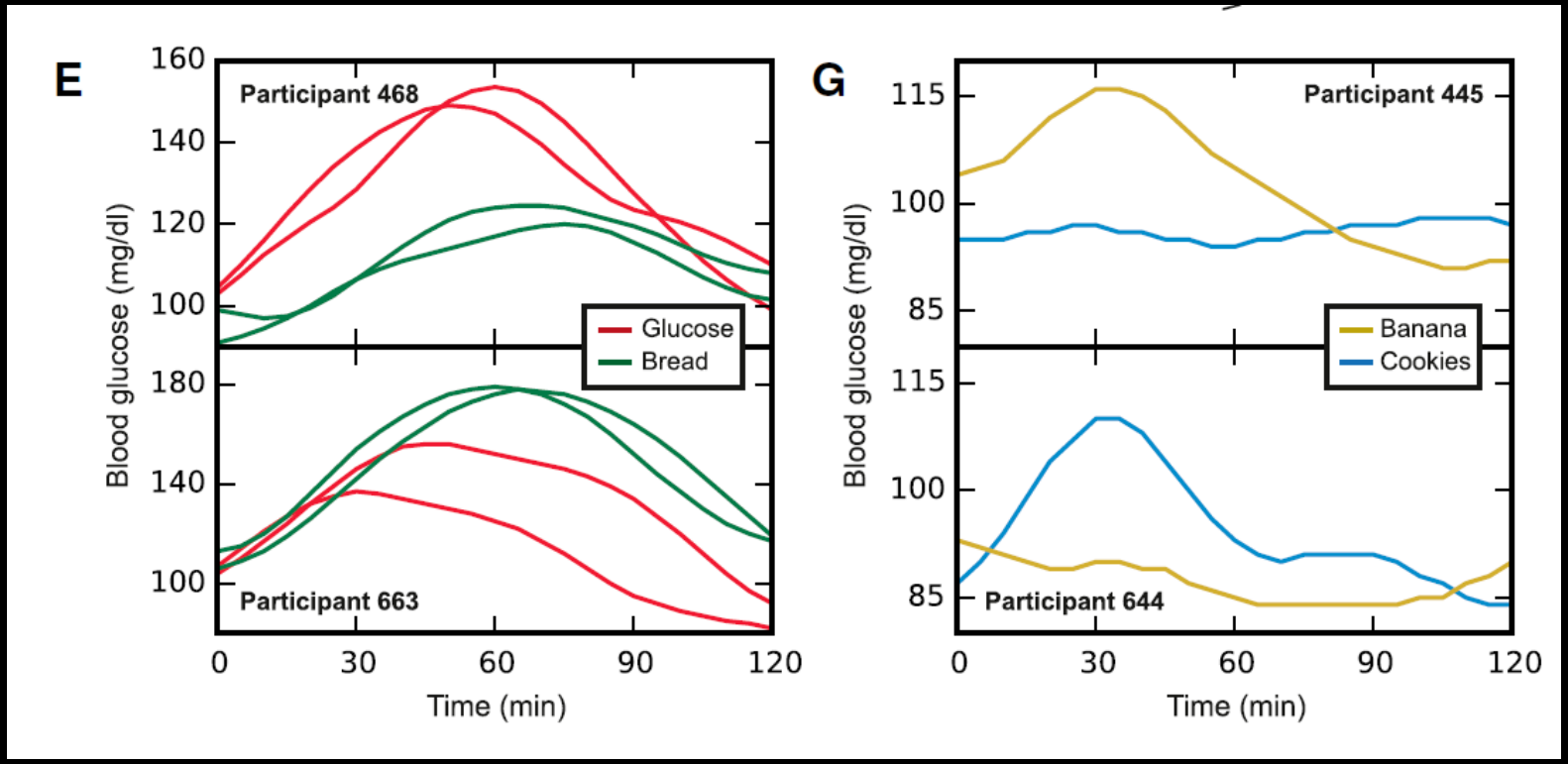
Significance of difference from equivalent glucose load: * = $p < 0.01$; † = $p < 0.001$; ‡ = $p < 0.05$; § = $p < 0.02$; | = $p < 0.002$; ¶ Only 25 g carbohydrate portion given.

CONTROL de la glucèmia

Possibles limitacions de l'índex glucèmic?



CONTROL de la glucèmia



CONTROL de la glucèmia

ÍNDEX GLUCÈMIC

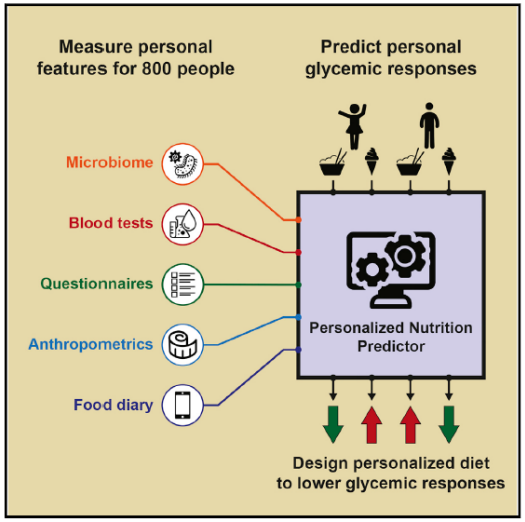
REDEFINIT

Article

Cell

Personalized Nutrition by Prediction of Glycemic Responses

Graphical Abstract



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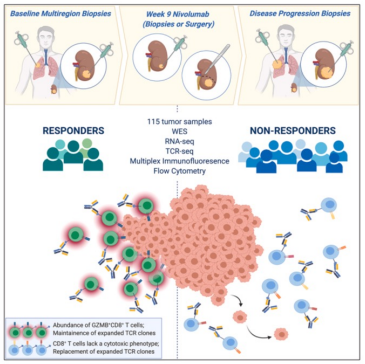
In Brief

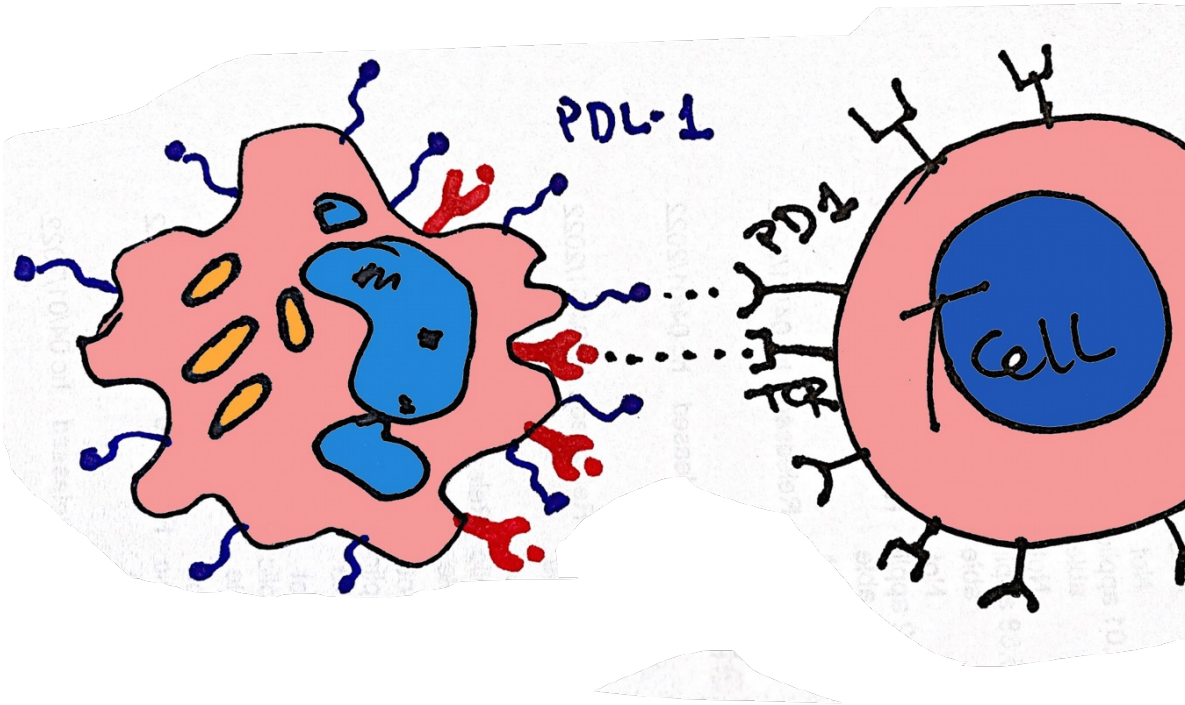
People eating identical meals present high variability in post-meal blood glucose response. Personalized diets created with the help of an accurate predictor of blood glucose response that integrates parameters such as dietary habits, physical activity, and gut microbiota may successfully lower post-meal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

Highlights

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

CÀNCER & MICROBIOMA





Immune checkpoint inhibitor. Checkpoint proteins, such as PD-L1 on tumor cells and PD-1 on T cells, help keep immune responses in check. The binding of PD-L1 to PD-1 keeps T cells from killing tumor cells in the body (left panel). Blocking the binding of PD-L1 to PD-1 with an immune checkpoint inhibitor (anti-PD-L1 or anti-PD-1) allows the T cells to kill tumor cells (right panel).

The Nobel Prize in Physiology or Medicine 2018

James P. Allison
Tasuku Honjo

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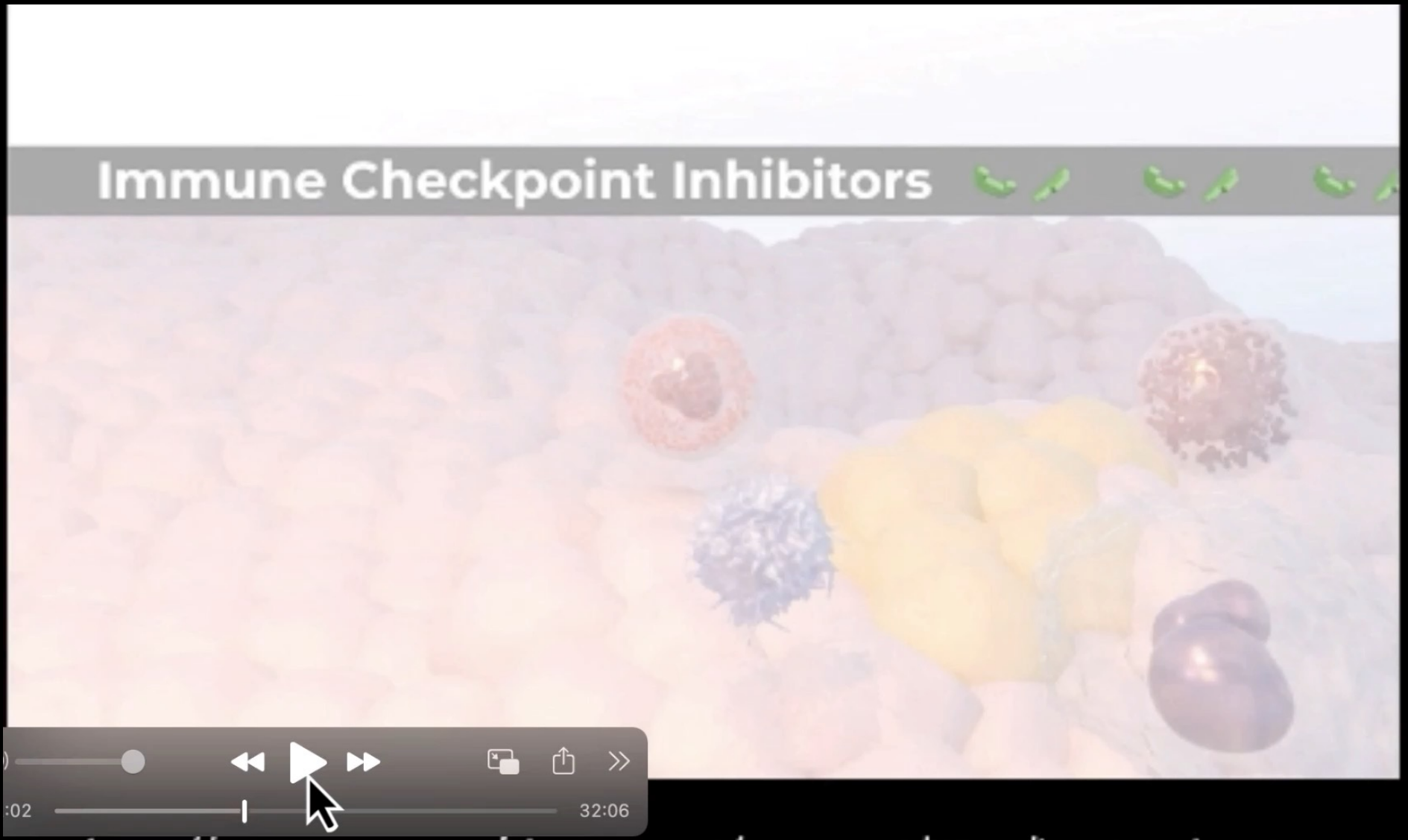

https://www.cancer.gov/about-cancer/treatment/types/immunotherapy