16–17 Ottobre 2026

PERCORSO ALLEANZA

PSICOBIOMA E ASSE INTESTINO-CERVELLO DALLA RICERCA ALL'AMBULATORIO NUTRIZIONALE

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diseases

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Review

Exploring the Exposome Spectrum: Unveiling Endogenous and Exogenous Factors in Non-Communicable Chronic Diseases

Laura Di Renzo [1], Paola Gualtieri [1], Giulia Frank [2,3,*], Rossella Cianci [4,5,*], Mario Caldarelli [4,5], Giulia Leggeri [1], Glauco Raffaelli [2,3], Erica Pizzocaro [2,3], Michela Cirillo [3] and Antonino De Lorenzo [1]

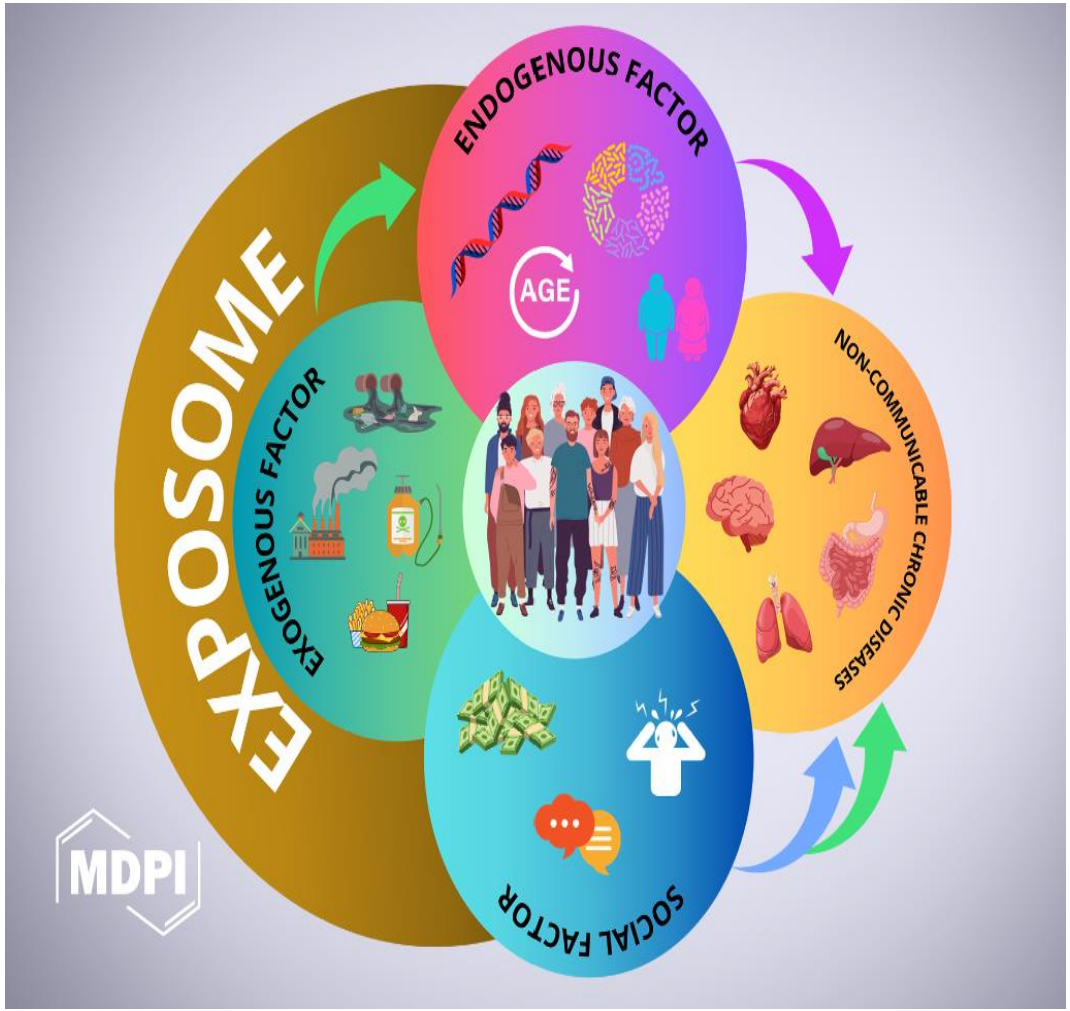
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Abstract: The exposome encompasses all endogenous and exogenous exposure individuals encounter throughout their lives, including biological, chemical, physical, psychological, relational, and socioeconomic factors. It examines the duration and intensity of these types of exposure and their complex interactions over time. This interdisciplinary approach involves various scientific disciplines, particularly toxicology, to understand the long-term effects of toxic exposure on health. Factors like air pollution, racial background, and socioeconomic status significantly contribute to diseases such as metabolic, cardiovascular, neurodegenerative diseases, infertility, and cancer. Advanced analytical methods measure contaminants in biofluids, food, air, water, and soil, but often overlook the cumulative risk of multiple chemicals. An exposome analysis necessitates sophisticated tools and methodologies to understand health interactions and integrate findings into precision medicine for better disease diagnosis and treatment. Chronic exposure to environmental and biological stimuli can lead to persistent low-grade inflammation, which is a key factor in chronic non-communicable diseases (NCDs), such as obesity, cardiometabolic disorders, cancer, respiratory diseases, autoimmune conditions, and depression. These NCDs are influenced by smoking, unhealthy diets, physical inactivity, and alcohol abuse, all shaped by genetic, environmental, and social factors. Dietary patterns, especially ultra-processed foods, can exacerbate inflammation and alter gut microbiota. This study investigates the exposome's role in the prevention, development, and progression of NCDs, focusing on endogenous and exogenous factors.

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Citation: Di Renzo, L.; Gualtieri, P.; Frank, G.; Cianci, R.; Caldarelli, M.; Leggeri, G.; Raffaelli, G.; Pizzocaro, E.; Cirillo, M.; De Lorenzo, A. Exploring the Exposome Spectrum: Unveiling Endogenous and Exogenous Factors in Non-Communicable Chronic Diseases. *Diseases* **2024**, *12*, 176. https://doi.org/10.3390/diseases12080176

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Review

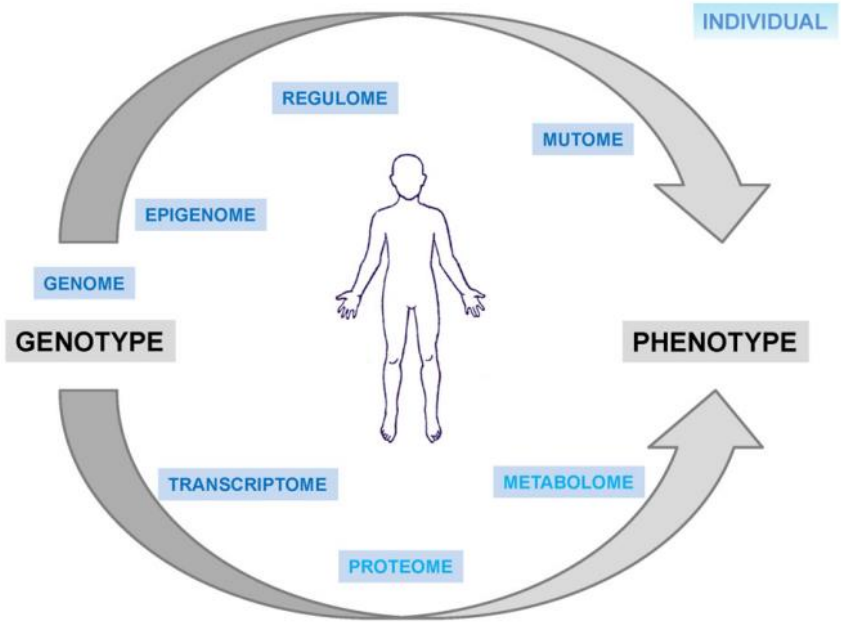
Foodomics as part of the host-microbiota-exposome interplay

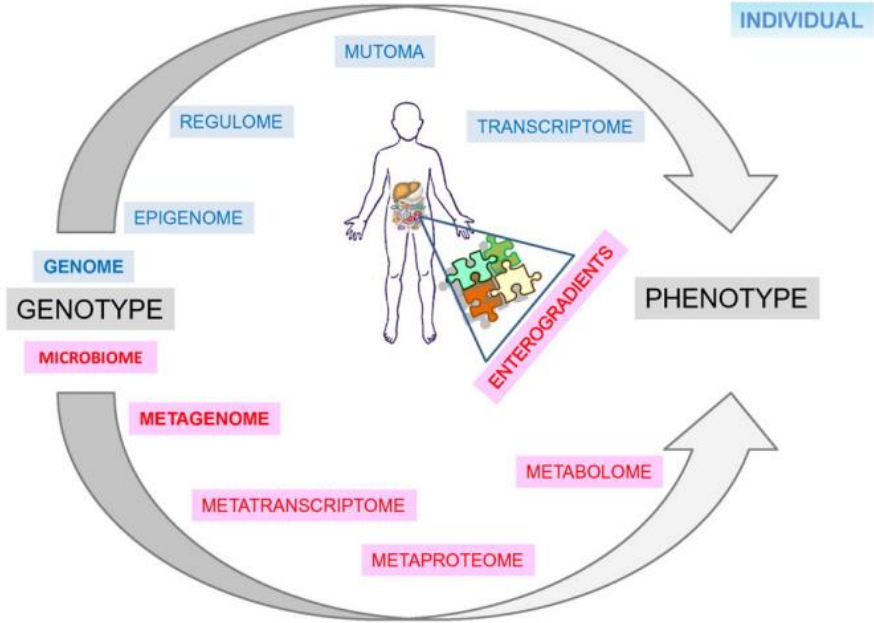
Lorenza Putignani [a,*], Bruno Dallapiccola [b]

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Interazioni genotipo-fenotipo e ruolo del microbioma

Strategie -omiche e meta-omiche rivelano il flusso tra genoma e fenotipo, evidenziando la regolazione molecolare del DNA e la sua traduzione in proteine. L'integrazione del microbioma amplia questa prospettiva nel contesto del "superorganismo", chiarendo la relazione funzionale ospite-microbiota





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REVIEW ARTICLE OPEN

Pesticide exposure and the microbiota-gut-brain axis

Rie Matsuzaki[1,2], Eoin Gunnigle[1], Violette Geissen[3], Gerard Clarke[1,4], Jatin Nagpal[1,5] and John F. Cryan[1,2]✉

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The gut microbiota exist within a dynamic ecosystem shaped by various factors that includes exposure to xenobiotics such as pesticides. It is widely regarded that the gut microbiota plays an essential role in maintaining host health, including a major influence on the brain and behaviour. Given the widespread use of pesticides in modern agriculture practices, it is important to assess the long-term collateral effects these xenobiotic exposures have on gut microbiota composition and function. Indeed, exposure studies using animal models have shown that pesticides can induce negative impacts on the host gut microbiota, physiology and health. In tandem, there is a growing body of literature showing that the effects of pesticide exposure can be extended to the manifestation of behavioural impairments in the host. With the increasing appreciation of the microbiota-gut-brain axis, in this review we assess whether pesticide-induced changes in gut microbiota composition profiles and functions could be driving these behavioural alterations. Currently, the diversity of pesticide type, exposure dose and variation in experimental designs hinders direct comparisons of studies presented. Although many insights presented, the mechanistic connection between the gut microbiota and behavioural changes remains insufficiently explored. Future experiments should therefore focus on causal mechanisms to examine the gut microbiota as the mediator of the behavioural impairments observed in the host following pesticide exposure.

The ISME Journal (2023) 17:1153–1166; https://doi.org/10.1038/s41396-023-01450-9

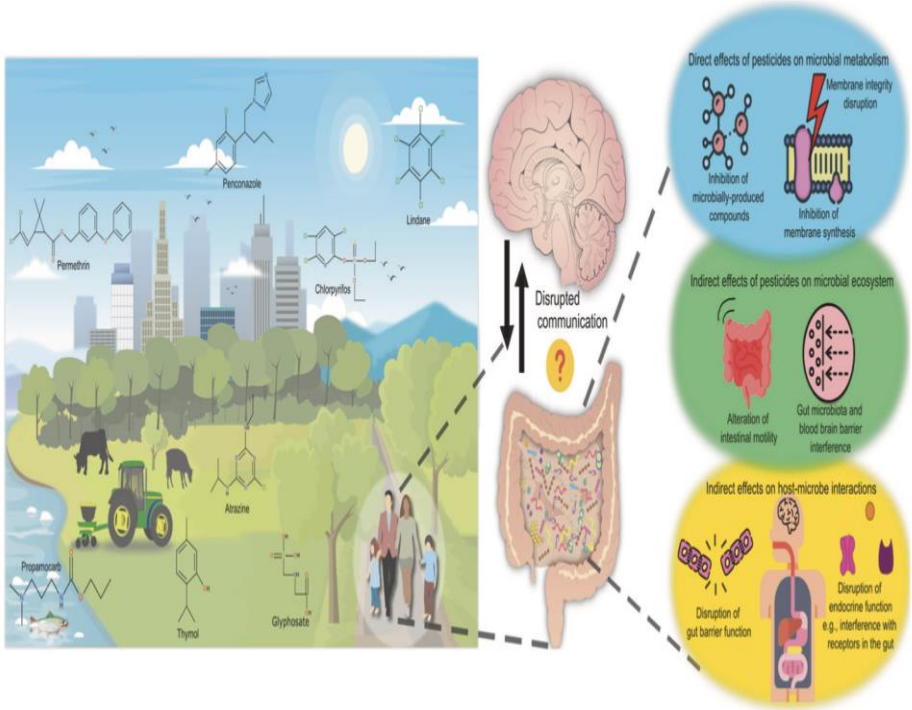
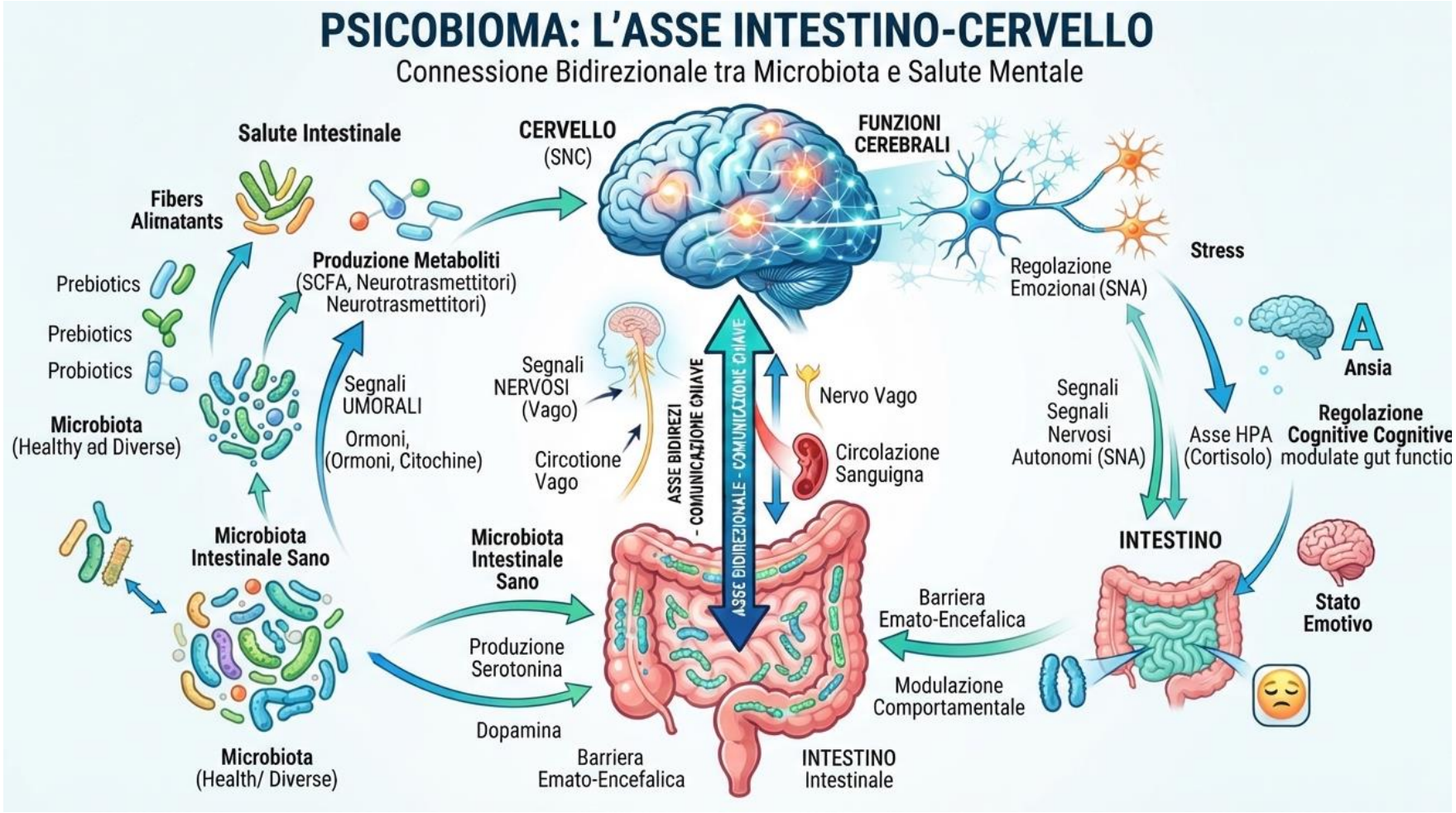


Fig. 1 Proposed mechanisms of environmental pesticides impacting the microbiota-gut-brain axis. The residue of pesticides in the environment (air, soil, water and food) can enter the body of the host leading to disrupted communication between the gut and the brain. While the mechanisms are still being uncovered, some of the potential routes include, solely or in combination of, direct effects on microbial metabolism, indirect effects of pesticides on microbial communities in the gut and indirect effects on host-microbe interactions, which may explain the disrupted communication as seen from behavioural impairments. The icons used in this figure were designed by adriansyah, Flat icons, Freepik and Kalashnyk on https://www.flaticon.com/ and the chemical structure was drawn using BIOVIA, Dassault Systèmes, BIOVIA Draw 2022, San Diego: Dassault Systèmes, 2023.


PSICOBIOMA: L'ASSE INTESTINO-CERVELLO
Connessione Bidirezionale tra Microbiota e Salute Mentale
Salute Intestinale
Fibers Alimatants
Prebiotics
Prebiotics
Probiotics
Microbiota
(Healthy ad Diverse)
Produzione Metaboliti
(SCFA, Neurotrasmettitori)
Neurotrasmettitori)
Segnali
UMORALI
Ormoni,
(Ormoni, Citochine)
CERVELLO
(SNC)
FUNZIONI
CEREBRALI
Regolazione
Emozionai (SNA)
Stress
Ansia
Segnali
NERVOSI
(Vago)
Circotione
Vago
ASSE BIDIREZIONALE - COMUNICAZIONE CHIAVE
Nervo Vago
Circolazione
Sanguigna
Segnali
Segnali
Nervosi
Autonomi (SNA)
Asse HPA
(Cortisolo)
Regolazione
Cognitive Cognitive
modulate gut functio
Microbiota
Intestinale Sano
Microbiota
Intestinale
Sano
INTESTINO
Barriera
Emato-Encefalica
Stato
Emotivo
Produzione
Serotonina
Modulazione
Comportamentale
Dopamina
Microbiota
(Health/ Diverse)
Barriera
Emato-Encefalica
INTESTINO
Intestinale

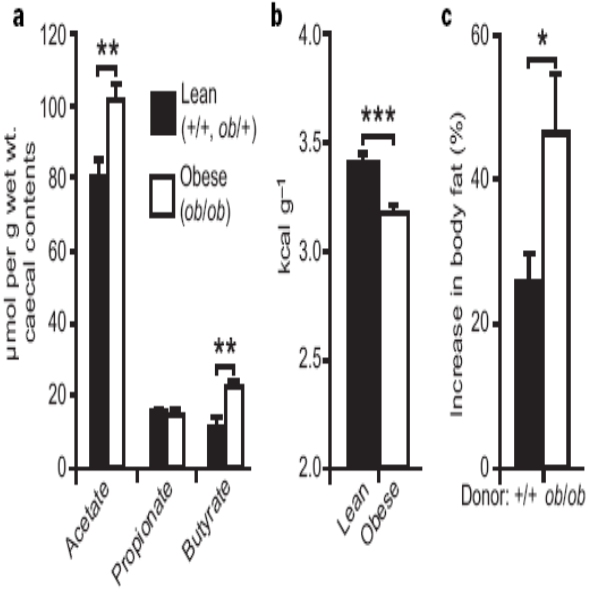
PHYSIOLOGY

Obesity and gut flora

Matej Bajzer and Randy J. Seeley



Figure 1 | Home for an abundant microbiological flora. The human gut and (inset) a scanning electron micrograph of part of the small intestine, with some bacterial inhabitants picked out in green.



An obesity-associated gut microbiome with increased capacity for energy harvest

Peter J. Turnbaugh[1], Ruth E. Ley[1], Michael A. Mahowald[1], Vincent Magrini[2], Elaine R. Mardis[1,2] & Jeffrey I. Gordon[1]

nutrients

MDPI

Review

Gut Feelings: How Microbiota Might Impact the Development and Course of Anorexia Nervosa

Jochen Seitz *, Brigitte Dahmen, Lara Keller and Beate Herpertz-Dahlmann

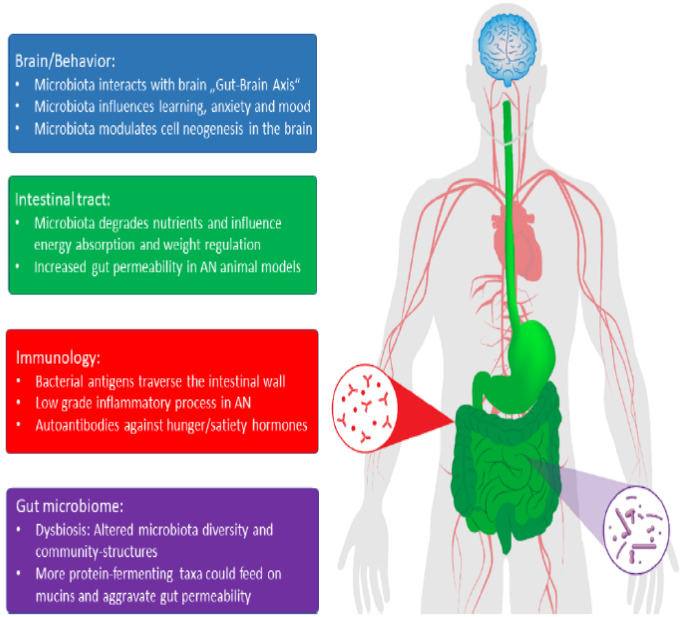


Figure 1. Effects of gut microbial products on brain functions and behavior in the intestinal tract and on immunological processes in patients with AN. (Adapted from [30]).

nutrients

MDPI

Review

Eating Disorders and Gastrointestinal Diseases

Antonella Santonicola [1,†], Mario Gagliardi [1,†], Michele Pier Luca Guarino [2], Monica Siniscalchi [1], Carolina Ciacci [1] and Paola Iovino [1,*]

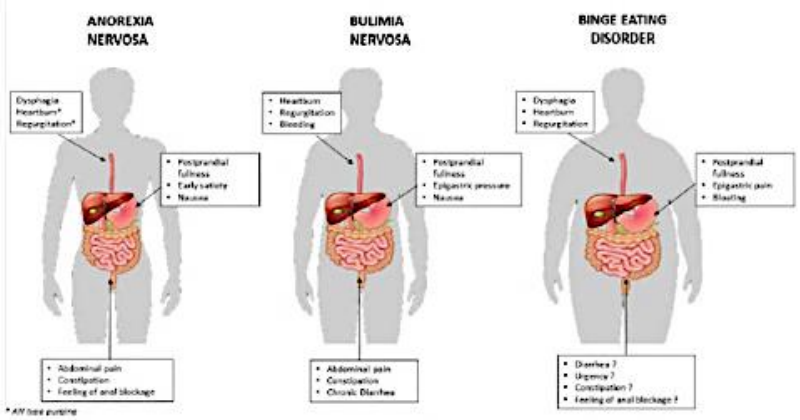


Figure 2. The most prevalent GI symptoms are shown taking into account different anatomical regions from esophagus to intestine in the three main ED disorders: AN, BN and BED.

Anorexia nervosa (AN)
a) restriction of energy intake leading to a significantly low body weight in the context of age, sex, developmental trajectory and physical health b) intense fear of gaining weight or becoming fat even though being at a significantly low weight. c) c) disturbance in the way in which one's body weight or shape is perceived **Specify if:** • **restrictive type:** over the past three months, the person has had no episodes recurrent compulsive binges or emptying practices (that is vomiting self-induced or abuse / misuse of laxatives, diuretics, or enemas). This subtype describes cases in which weight loss is achieved essentially through diets, fasts and / or excessive exercise • **bulimic / purgative type:** over the past three months, the person has had recurrent episodes of compulsive bingeing or emptying practices (that is vomiting self-induced or abuse / misuse of laxatives, diuretics, or enemas)
Bulimia nervosa (BN)
a) recurrent episodes of binge eating (at least once a week for 3 months), characterized by eating in a discrete period of time (e.g. within any 2 hour period), an amount of food that is definitely larger than what most individuals would eat and by a lack of control over eating during the episodes b) recurrent inappropriate compensatory behaviors to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, or other medications; fasting; or excessive exercise
Binge-Eating Disorder (BED)
Recurrent episodes of binge eating (at least once a week for 3 months) associated with three or more of the following: a) Eating much more rapidly than normal b) Eating until feeling uncomfortably full c) Eating large amounts of food when not feeling physically hungry d) Eating alone because of feeling embarrassed by how much one is eating e) Feeling disgusted with oneself, depressed, or very guilty afterwards f) Uncontrolled eating behavior occurs in the media, at least once a week for three consecutive months. g) The uncontrolled feeding is not associated with the systematic use of inadequate compensatory behaviors (eg, use of purgatives, fasting, excessive exercise) and does not occur exclusively in the course of bulimia nervosa or anorexia nervosa.

Figure 1. The description of the three main types of Eating Disorders (adapted by the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V), released by the American Psychiatric Association [2].

RESEARCH ARTICLE

Microbiota in anorexia nervosa: The triangle between bacterial species, metabolites and psychological tests

Francesca Borgo[1], Alessandra Riva[1], Alberto Benetti[2], Maria Cristina Casiraghi[3], Sara Bertelli[2], Stefania Garbossa[1,2], Simona Anselmetti[2], Silvio Scarone[1,2], Antonio E. Pontiroli[1,2], Giulia Morace[1], Elisa Borghi[1]*

Fecal and systemic microbial metabolites

Fecal concentrations of total SCFAs, butyrate and propionate were significantly ($p<0.05$) decreased in AN samples, while acetate, *iso*-valerate and *iso*-butyrate were not different between groups (Table 4).

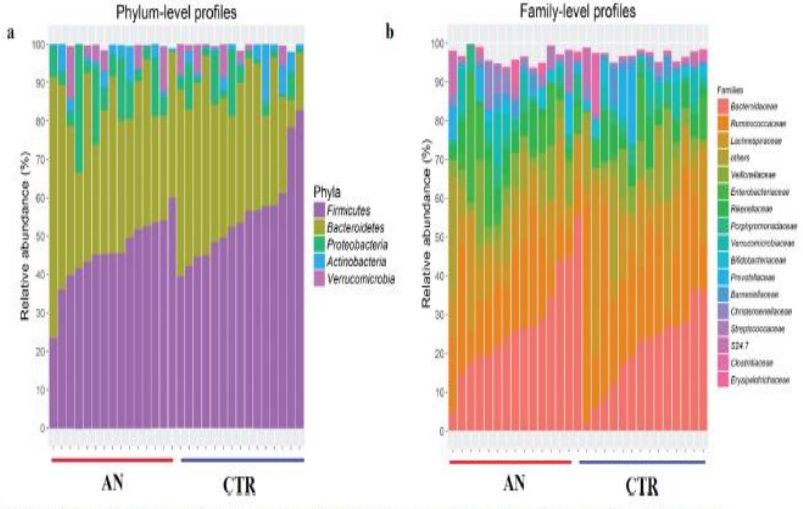


Fig 1. Relative abundance of common microbial taxa. Bar charts showing the relative abundance of the most represented microbial taxa, defined as having a mean relative abundance of >1%, in stool samples of anorexia nervosa (AN, n = 15) and control (CTR, n = 15) groups. Phylum-level (**a**) and family-level (**b**) taxon profiles are shown.

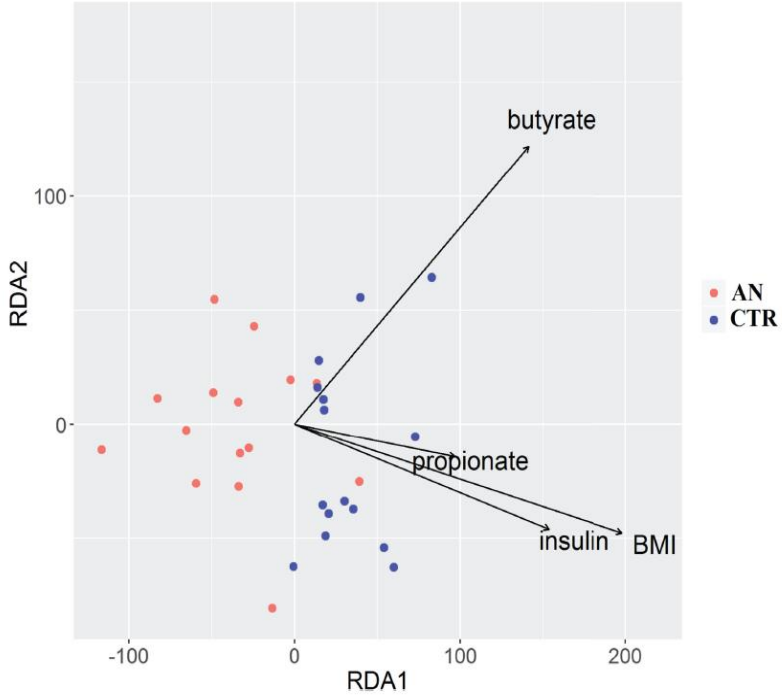


Fig 3. Distance-based redundancy analysis (db-RDA). Db-RDA plot shows correlations between gut microbiota composition and body mass index (BMI), insulin, propionate and butyrate. Only significant variables are represented ($p>0.05$, Permanova analysis). Arrows in the db-RDA biplot denote the magnitudes and directions of the variable effects. Controls are represented by blue dots and anorexic subjects by red dots.

Journal of Psychiatric Research 63 (2015) 1–9

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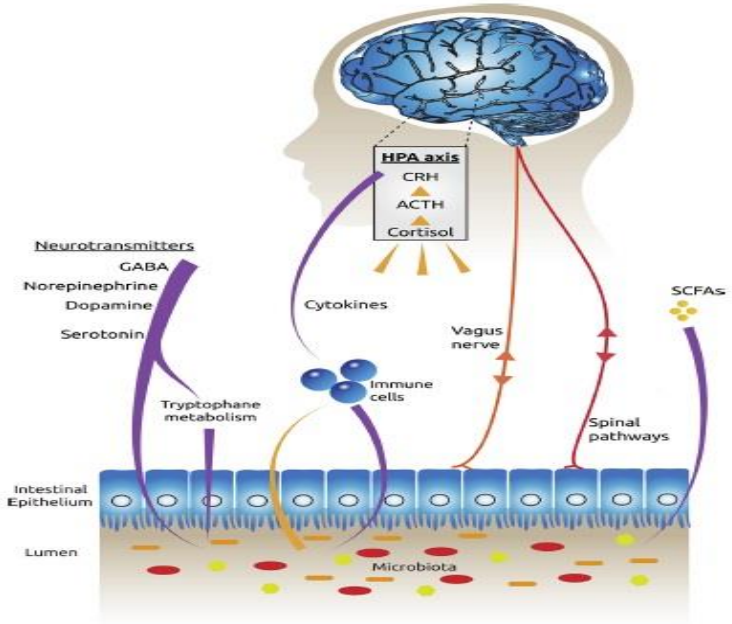
Journal of Psychiatric Research

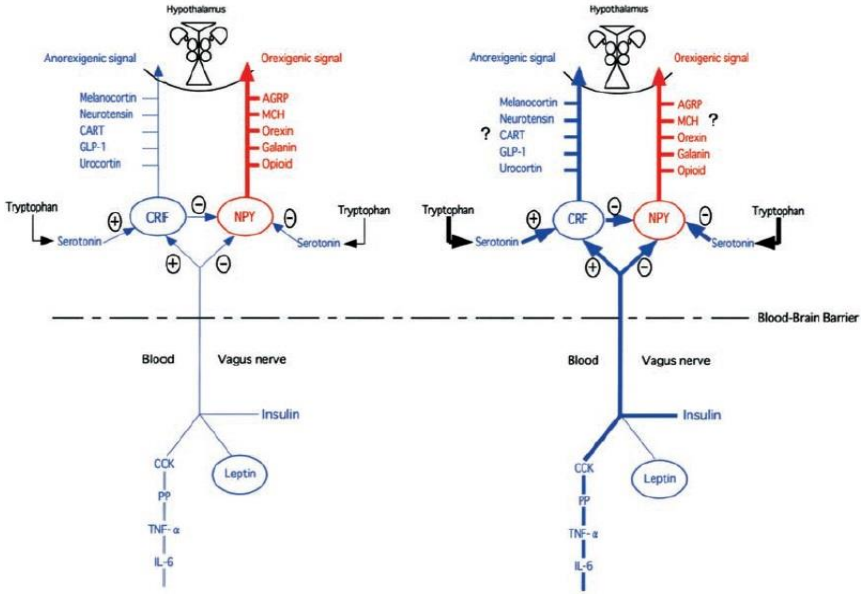
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Review

Collective unconscious: How gut microbes shape human behavior

Timothy G. Dinan [a, b, *], Roman M. Stilling [a, d], Catherine Stanton [a, b, c], John F. Cryan [a, d]





- The multiple bidirectional routes of communication between the brain and the gut microbiota. These routes include the **vagus nerve, the hypothalamic-pituitary-adrenal axis (HPA), cytokines produced by the immune system, tryptophan metabolism and production of short chain fatty acids.**

Behavioural Brain Research 356 (2019) 390–399

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Behavioural Brain Research

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Research report

Probiotics modify body weight together with anxiety states via pro-inflammatory factors in HFD-treated Syrian golden hamster

Ennio Avolio[a,b,*], Gilda Fazzari[a], Merylin Zizza[a], Antonino De Lorenzo[b], Laura Di Renzo[b], Raffaella Alò[a], Rosa Maria Facciolo[a], Marcello Canonaco[a]



3g/die:

- *Streptococcus thermophilus* (CNCM strain number I-1630) 1.5×10^{10} UFC
- *Lactobacillus bulgaricus* (CNCM strain numbers I-1632 and I-1519) 1.5×10^{10} UFC
- *Lactococcus lactis subsp lactis* (CNCM strain number I-1631) 1.5×10^{10} UFC *B*
- *lactis* (CNCM I-2494/DN-173 010) 1.2×10^{9} UFC *Lactobacillus acidophilus* 1.5×10^{10} UFC
- *Streptococcus thermophilus* 1.5×10^{10} UFC
- *Lactobacillus Plantarum* 1.5×10^{10} UFC
- *Bifidobacterium lactis* (CNCM I-2494) 1.5×10^{10} UFC *Lactobacillus Reuteri* (DSM 17938) $1.5 \times 1\ 1^{010}$ UFC

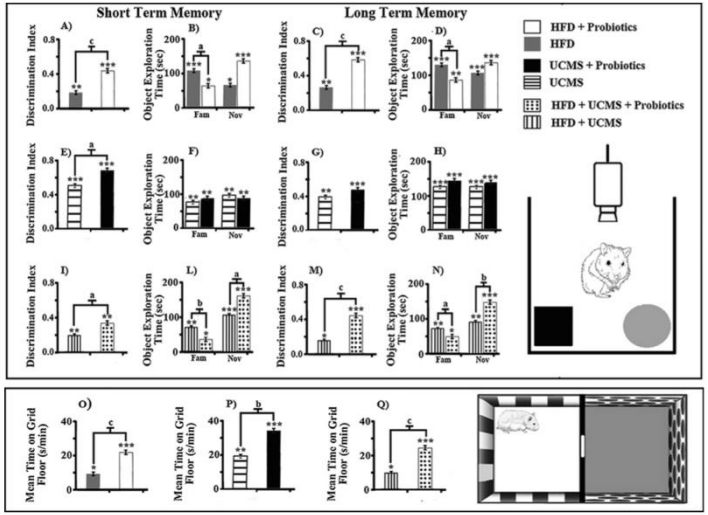


Fig. 2. Effects of probiotics on memory performances of hamsters.
Effects of treatments n = 6/treatment groups) on memory performances were evaluated in animals that received HFD + probiotics, HFD, UCMS + probiotics, UCMS, UCMS + HFD + probiotics, UCMS + HFD with respect to unstressed control (*) during Novel Object (A–N) plus Conditioned Place Preference Tests (O–Q). Animals treated with probiotics received a single daily dose directly dissolved in water (3 g/200 ml to each hamster per day). Each bar represents % mean ± S.E.M. of discrimination index (A, C, E, G, I, M) and object exploration time (B, D, F, H, L, N; short and long term memory) along with mean time on grid floor box (O, P, Q). Behavioral changes were determined by ANOVA plus a *post hoc* Newman–Keuls test when $p < 0.05$, $*^{a}p < 0.05$, $**^{b}p < 0.01$ and $***^{c}p < 0.001$.

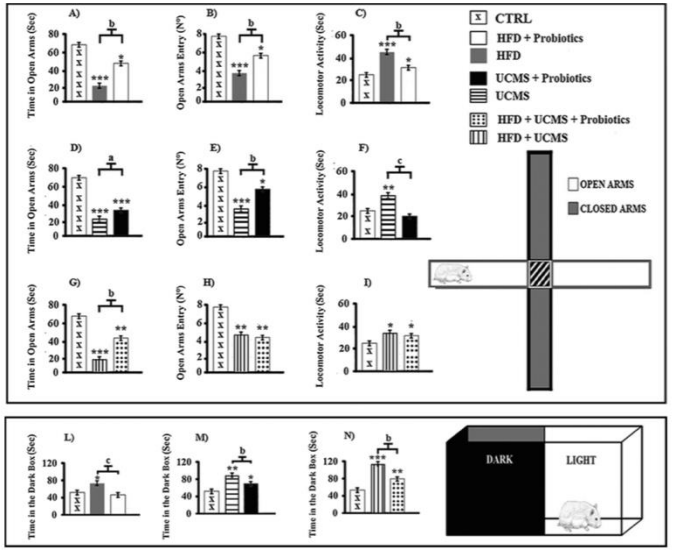


Fig. 1. Effects of probiotics on anxiety-like behaviors of hamsters.
Effects of treatments (n = 6/treatment groups) on anxiety-like behaviors were checked in animals that received HFD + probiotics, HFD, UCMS + probiotics, UCMS, UCMS + HFD + probiotics, UCMS + HFD with respect to unstressed controls (*) during EPM (A–I) plus LDT (L–N). Animals treated with probiotics received a single daily dose directly dissolved in water (3 g/200 ml to each hamster per day). Each bar represents % change (± S.E.M) with respect to unstressed controls of time spent in open arms (A, D, G), open arms entry (B, E, H), back- and forward locomotor activities (C, F, I) along with time spent in the dark box (L, M, N) by treated hamsters. Behavioral changes were determined by ANOVA plus a *post hoc* Newman–Keuls test when $*^{a}p < 0.05$, $**^{b}p < 0.01$ and $***^{c}p < 0.001$.

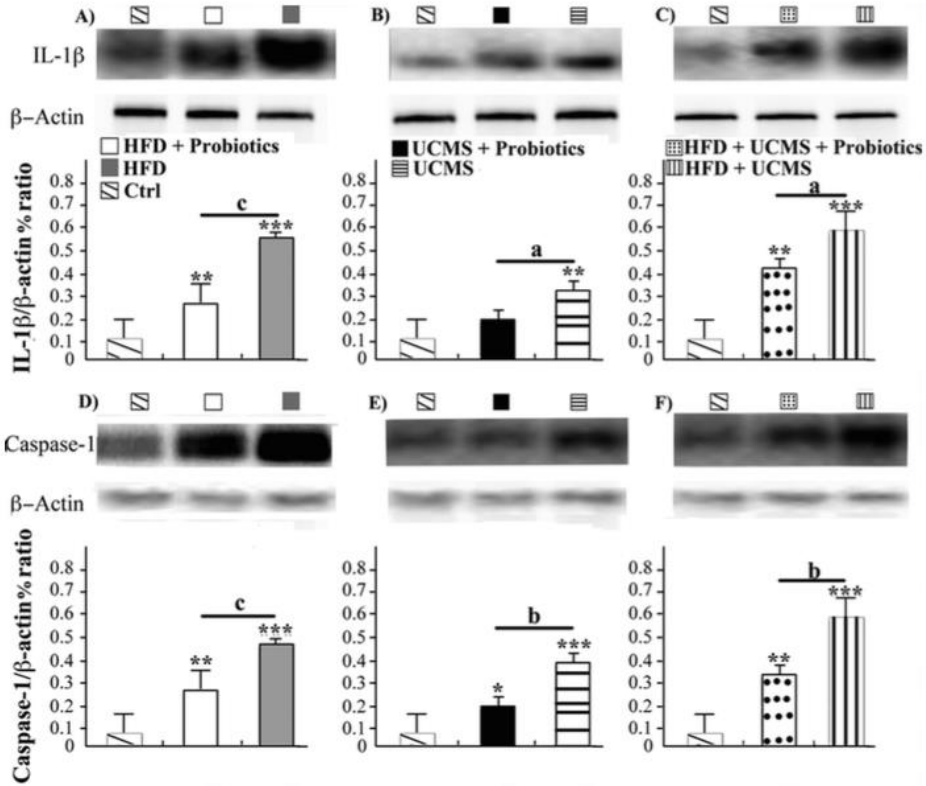
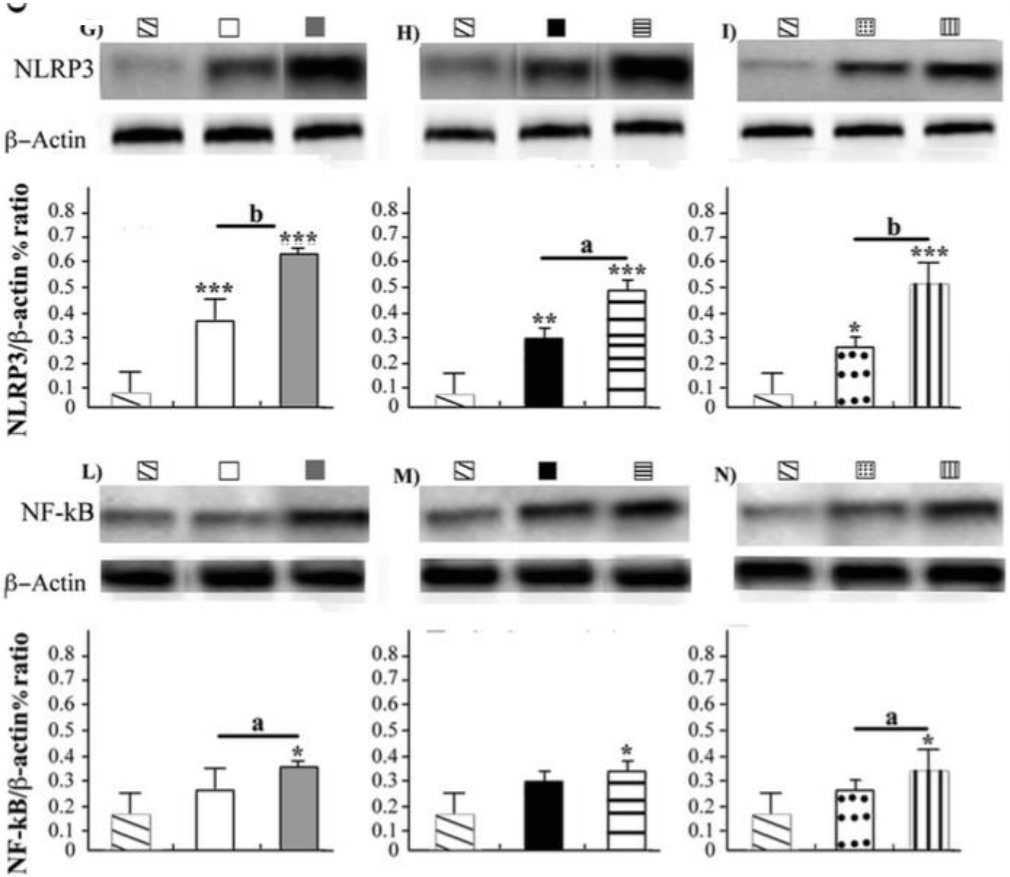


Fig. 4. Effects of probiotics on hypothalamic expression of IL-1β, Caspase-1, NLRP3 and NF-kB.

Immunoblotting was used to detect IL-1β (A–C), Caspase-1 (D–F), NLRP3 (G–I) and NF-kB (L–N) expression in the HTH of HFD + probiotics, HFD, UCMS + probiotics, UCMS, UCMS + HFD + probiotics, UCMSd + HFD with respect to unstressed controls. Quantification of immunoblotting results was expressed as ratio of IL-1β/β-Actin, Caspase-1/β-Actin, NLRP3/β-Actin and NF-kB/β-Actin ± s.e.m. Immunoblotting differences in animal groups (n = 6/group) were evaluated using a two-way ANOVA followed by a *post hoc* Newman-Keuls multiple range test when *p*-value $^{*,a}p < 0.05$, $^{**,b}p < 0.01$ and $^{***,c}p < 0.001$.


G)
H)
I)
NLRP3
β–Actin
NLRP3/β-actin % ratio
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
b
***
***
a
**
***
b
*
***
L)
M)
N)
NF-kB
β–Actin
NF-kB/β-actin% ratio
a
*
*
a
*

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Nutrition,
Metabolism &
Cardiovascular Diseases

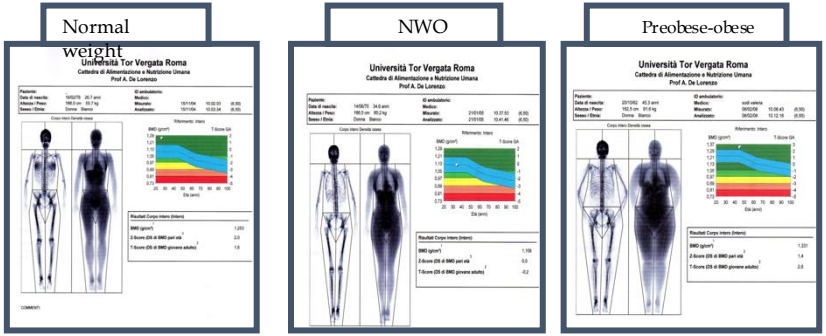
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Normal weight obese (NWO) women: An evaluation of a candidate new syndrome

A. De Lorenzo [a,b,*], R. Martinoli [a], F. Vaia [c], L. Di Renzo [a]

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3g/die:
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De Lorenzo *et al. J Transl Med* (2017) 15:135
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Journal of Translational Medicine

RESEARCH Open Access

Can psychobiotics intake modulate psychological profile and body composition of women affected by normal weight obese syndrome and obesity? A double blind randomized clinical trial

Antonino De Lorenzo[1,8*], Micaela Costacurta[2], Giuseppe Merra[3], Paola Gualtieri[4], Giorgia Cioccoloni[4], Massimiliano Marchetti[5,6], Dimitrios Varvaras[7], Raffaella Docimo[2] and Laura Di Renzo[1]

Table 4. Comparison of normal weight lean, normal weight obese and pre-obese/obese groups of women at 3 week POS treatment.

	NWL	NWO	PreOB/OB	
	Mean±SD (Min - Max)	Mean±SD (Min - Max)	Mean±SD (Min - Max)	p
Weight (Kg)	54.84±5.63 ^{a,b} (49.20 - 63.00)	63.10±3.27 (59.00 - 66.60)	63.35±0.16 (63.20 - 63.50)	<0.001
BMI (Kg/m²)	20.49±1.40 ^{a,b} (18.98 - 22.59)	23.80±0.85 ^{c} (22.92 - 24.88)	25.77±0.48 (25.32 - 26.23)	<0.001
Waist (cm)	67.98±1.68 ^{a,b} (63.80 - 75.00)	75.33±1.70 (70.00 - 81.00)	73.75±2.35 (71.50 - 76.00)	<0.001
Hip (cm)	93.23±4.35 ^{a,b} (87.50 - 97.00)	101.00±3.72 ^{c} (98.00 - 106.00)	106.75±0.78 (106.00 - 107.50)	<0.001
Resistance (Ohm)	598.33±69.50 ^{b} (497.00 - 690.00)	567.00±53.60 (513.00 - 636.00)	523.00±24.02 (500.00 - 546.00)	0.01
Reactance (Ohm)	65.89±8.46 (56.00 - 73.00)	59.67±8.75 ^{c} (51.00 - 71.00)	69.50±6.79 (63.00 - 76.00)	0.02
PA (°)	6.29±0.46 ^{b} (5.70 - 6.80)	6.03±0.97 ^{c} (5.10 - 7.30)	7.55±0.37 (7.20 - 7.90)	<0.001
TBW (L)	31.66±3.00 (29.70 - 36.90)	33.40±2.56 (31.10 - 36.80)	33.35±1.10 (32.30 - 34.40)	0.18
Total Body Water (%)	57.82±3.19 (52.40 - 60.40)	52.90±2.01 (50.60 - 55.30)	52.65±1.83 (50.90 - 54.40)	<0.001
ECW (L)	14.07±1.39 (12.60 - 16.10)	15.37±2.18 ^{c} (12.50 - 17.40)	13.20±0.84 (12.40 - 14.00)	0.01
ECW (%)	43.93±2.64 ^{b} (40.80 - 47.20)	45.87±4.29 ^{c} (40.30 - 50.10)	39.50±1.36 (38.20 - 40.80)	<0.001
ICW (L)	17.39±1.80 ^{b} (16.20 - 20.80)	18.03±1.47 ^{c} (16.10 - 19.40)	20.15±0.26 (19.90 - 20.40)	<0.001
ICW (%)	55.62±1.91 ^{b} (52.92 - 57.58)	54.12±4.37 ^{c} (49.85 - 59.81)	60.61±1.37 (59.30 - 61.92)	<0.001

Can psychobiotics intake modulate psychological profile and body composition of women affected by normal weight obese syndrome and obesity? A double blind randomized clinical trial

Table 1 Comparison of body composition parameters of normal weight lean, normal weight obese and pre-obese/obese groups between baseline and after 3 weeks POS treatment

	NWL		NWO		PreobOB	
	Baseline	POS	Baseline	POS	Baseline	POS
Age (years)	30.18 ± 2.04 (28.00–33.00)		40.00 ± 12.56 (27.00–56.00)		33.57 ± 10.57 (24.00–50.00)	
Height (cm)	164.64 ± 4.03 (160.00–170.00)		162.90 ± 6.60 (154.00–168.00)		158.33 ± 1.86 (155.60–160.00)	
Weight (kg)	55.55 ± 4.65[a,o] (50.50–62.50)	54.84 ± 5.63[χ,ŋ] (49.20–63.00)	63.50 ± 3.97[¥] (59.00–68.30)	63.10 ± 3.27 (59.00–66.60)	69.47 ± 6.07 (63.80–78.00)	63.35 ± 0.16[b] (63.20–63.50)
BMI (kg/m^2)	20.47 ± 1.04[a,o] (19.48–22.41)	20.49 ± 1.40[χ,ŋ] (18.98–22.59)	23.94 ± 0.93[¥] (22.74–24.88)	23.80 ± 0.84[a,¢] (22.92–24.88)	27.68 ± 1.92 (25.56–30.47)	25.78 ± 0.48[b] (25.32–26.23)
Waist (cm)	67.41 ± 4.22 (64.50–75.00)[o]	67.98 ± 4.60[χ,ŋ] (63.80–75.00)	74.25 ± 5.08 (69.50–79.00)	75.33 ± 4.70 (70.00–81.00)	80.17 ± 8.02 (73.00–91.00)	73.75 ± 2.35[b] (71.50–76.00)
Hip (cm)	94.09 ± 4.57 [a,o] (87.00–97.00)	93.23 ± 4.35 [χ,ŋ] (87.50–97.00)	103.25 ± 5.61[¥] (98.00–108.50)	101.00 ± 3.72[¢] (98.00–106.00)	107.73 ± 0.54 (107.00–108.20)	106.75 ± 0.78[b] (106.00–107.50)
TBFat (%)	26.55 ± 2.83[a,o] (24.10–29.00)	–	35.25 ± 1.44[¥] (33.90–36.60)	–	43.80 ± 0.00 (43.80–43.80)	–
TBFat (kg)	15.15 ± 0.01[a,o] (15.14–15.16)	–	21.73 ± 1.73[¥] (20.11–23.34)	–	34.10 ± 0.00 (34.10–34.10)	–
TBLean (kg)	40.17 ± 5.98 (34.99–45.35)	–	37.61 ± 0.57 (37.08–38.14)	–	41.26 ± 0.00 (41.26–41.26)	–
FM (kg)	11.35 ± 2.86[a,o] (8.10–15.10)	11.80 ± 3.12[c,χ,ŋ] (8.10–16.00)	18.00 ± 1.64 (15.90–19.70)	17.27 ± 1.80[a] (15.80–19.70)	18.65 ± 0.99 (17.70–19.60)	16.95 ± 0.99[b] (16.00–17.90)
FM (%)	20.30 ± 4.06[a,o] (16.10–26.40)	21.05 ± 4.43[c,χ,ŋ] (16.50–27.80)	28.37 ± 2.09 (26.90–31.20)	27.57 ± 2.58[a] (25.00–30.90)	29.10 ± 1.36 (27.80–30.40)	26.70 ± 1.46[b] (25.30–28.10)
FFM (kg)	44.23 ± 3.57 (42.20–50.00)	43.75 ± 4.06 (41.10–50.30)	45.50 ± 3.25 (43.10–49.90)	45.83 ± 3.32[a] (43.20–50.30)	45.50 ± 0.63 (44.90–46.10)	46.40 ± 0.84[b] (45.60–47.20)
FFM (%)	79.70 ± 4.06[a,o] (73.60–83.90)	78.95 ± 4.43[c,χ,ŋ] (72.20–83.50)	71.63 ± 2.09 (68.80–73.10)	72.43 ± 2.58[a] (69.10–75.00)	70.90 ± 1.36 (69.60–72.20)	73.30 ± 1.46[b] (71.90–74.70)

Results are expressed in mean value ± standard deviation, and minimum and maximum for each parameter. Values of $p < 0.05$ are considered significant

BMI body mass index, *ECW* extracellular water, *FM* fat mass, *FFM* fat free mass, *ICW* intracellular water, *NWL* normal weight lean, *NWO* normal weight obese, *PA* phase angle, *PreOB/OB* preobese–obese, *TB* total body, *TBW* total body water

[a] NWO T0 vs T1 $p < 0.05$; [b] PreOB/OB T0 vs T1 $p < 0.05$ [c] NWL T0 vs T1. For Anova-test at baseline $p < 0.05$; [¥] NWL vs NWO $p < 0.05$; [¥] NWO vs PreOB/OB $p < 0.05$; [*] NWL vs PreOB/OB $p < 0.05$. Anova-test after POS $p < 0.05$; [χ] NWL vs NWO $p < 0.05$; [¢] NWO vs PreOB/OB $p < 0.05$; [ŋ] NWL vs PreOB/OB $p < 0.05$

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Can psychobiotics intake modulate psychological profile and body composition of women affected by normal weight obese syndrome and obesity? A double blind randomized clinical trial

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	NWL		NWO		PreOB/OB	
	T0 Mean±SD (Min-Max)	T21 Mean±SD (Min-Max)	T0 Mean±SD (Min-Max)	T21 Mean±SD (Min-Max)	T0 Mean±SD (Min-Max)	T21 Mean±SD (Min-Max)
EDI-2_B	1.00±2.45 (0.00 - 10.00)	0.39±1.24 (0.00 - 5.00)	0.91±1.64 (0.00 - 7.00)	0.53±1.16 [a] (0.00 - 5.00)	3.64±4.12 (0.00 - 16.00)	2.50±2.89 [b] (0.00 - 10.00)
EDI-2_BD	5.28±6.09 (0.00 - 23.00)	4.94±6.01 (0.00 - 23.00)	9.29±7.81 (0.00 - 27.00)	7.50±7.18 [a] (0.00 - 25.00)	14.09±6.35 (2.00 - 24.00)	11.91±5.76 [b] (0.00 - 20.00)
EDI-2_I	1.39±2.20 (0.00 - 6.00)	1.22±1.90 (0.00 - 5.00)	3.26±4.29 (0.00 - 17.00)	1.62±2.85 [a] (0.00 - 10.00)	5.82±4.92 (1.00 - 19.00)	3.68±4.51 [b] (0.00 - 16.00)

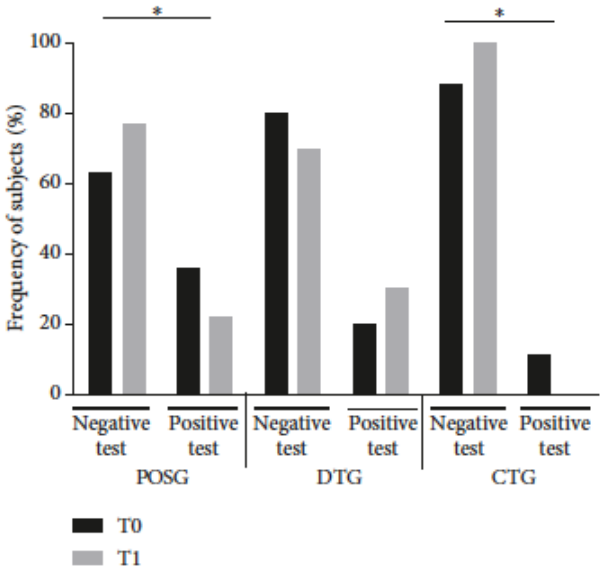


FIGURE 3: Frequency change of anxious subjects in POSG, DTG, and CTG after treatment. Frequency of anxiety was evaluated before and after treatment in POSG, DTG, and CTG. Negative test (nonanxious 619 subjects) if total score < 18 and positive test (anxious subjects) if total score ≥ 18. Statistical significance attributed to results with $^*p < 0.05$ between T0 and T1 by McNemar test. POSG: $p = 0.03^*$; DTG: $p = 0.10$; CTG: $p = 0.01^*$. POSG: psychobiotics oral suspension group; DTG: dietary treatment group; CTG: combined treatment group.

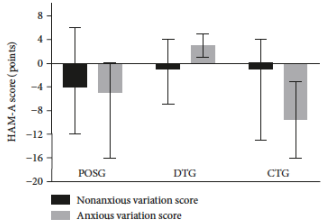


FIGURE 2: Hamilton anxiety rating scale (HAM-A) score variation before and after treatments in anxious and nonanxious subjects. Nonanxious subjects (negative test) if total score < 18 and anxious subjects (positive test) if total score ≥ 18. Variation score is shown as median, minimum, and maximum. 609 statistical significance attributed to results with $p < 0.05$ by Kruskal Wallis test. Anxious variation score among groups: $p = 0.10$ and nonanxious variation score among groups: $p = 0.67$. POSG: psychobiotics oral suspension group; DTG: dietary treatment group; CTG: combined treatment group.

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Clinical Study

Evidences of a New Psychobiotic Formulation on Body Composition and Anxiety

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Il contributo del nostro gruppo di ricerca ad oggi

> Nutrients. 2021 Jan 25;13(2):358. doi: 10.3390/nu13020358.

Potential Effects of a Modified Mediterranean Diet on Body Composition in Lipoedema

Laura Di Renzo [1], Giulia Cinelli [2 3], Lorenzo Romano [2], Samanta Zomparelli [2], Gemma Lou De Santis [2], Petronilla Nocerino [1], Giulia Bigioni [4], Lorenzo Arsini [4], Giuseppe Cenname [5], Alberto Pujia [6], Gaetano Chiricolo [6], Antonino De Lorenzo [1]

> Nutrients. 2023 Aug 20;15(16):3654. doi: 10.3390/nu15163654.

Modified Mediterranean-Ketogenic Diet and Carboxytherapy as Personalized Therapeutic Strategies in Lipedema: A Pilot Study

Laura Di Renzo [1], Paola Gualtieri [1], Samanta Zomparelli [2], Gemma Lou De Santis [3], Silvia Seraceno [2], Claudia Zuena [2], Giulia Frank [2 3], Rossella Cianci [4], Domenico Centofanti [5], Antonino De Lorenzo [1]

> Horm Mol Biol Clin Investig. 2021 Jul 29;42(4):351-355. doi: 10.1515/hmbci-2021-0027.

The association between serum vitamin D and mood disorders in a cohort of lipedema patients

Mohammad Al-Wardat [1], Nuha Alwardat [2], Gemma Lou De Santis [3], Samantha Zomparelli [3], Paola Gualtieri [2], Giulia Bigioni [4], Lorenzo Romano [3], Laura Di Renzo [3]

Review > Eat Weight Disord. 2020 Aug;25(4):851-856. doi: 10.1007/s40519-019-00703-x. Epub 2019 May 6.

The effect of lipedema on health-related quality of life and psychological status: a narrative review of the literature

Nuha Alwardat [1 2], Laura Di Renzo [3], Mohammad Alwardat [4 5], Lorenzo Romano [6], Gemma Lou De Santis [6], Paola Gualtieri [3], Elena Carrano [6], Petronilla Nocerino [3], Antonino De Lorenzo [3]

> Int J Environ Res Public Health. 2022 Oct 21;19(20):13679. doi: 10.3390/ijerph192013679.

The Difficulties in Emotional Regulation among a Cohort of Females with Lipedema

Mohammad Al-Wardat [1], Chantelle Clarke [2], Nuha Alwardat [3], Manal Kassab [4], Chiara Salimei [5], Paola Gualtieri [6], Marco Marchetti [5 7], Talitha Best [2], Laura Di Renzo [6]

> Eur Rev Med Pharmacol Sci. 2020 Mar;24(6):3236-3244. doi: 10.26355/eurrev_202003_20690.

The role of IL-6 gene polymorphisms in the risk of lipedema

L Di Renzo [1], P Gualtieri, N Alwardat, G De Santis, S Zomparelli, L Romano, M Marchetti, S Michelin, A Capacci, A Piccioni, M Costacurta, M G Tarsitano, F Franceschi, G Merra

Comment > Eur Rev Med Pharmacol Sci. 2023 Feb;27(4):1625-1632. doi: 10.26355/eurrev_202302_31407.

The role of MTHFR polymorphisms in the risk of lipedema

P Gualtieri [1], M Al-Wadart, G L De Santis, N Alwadart, D Della Morte, C Clarke, T Best, C Salimei, G Bigioni, R Cianci, A De Lorenzo, L Di Renzo

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Characterization of Gut Microbiota Profile in Lipedema: A Pilot Study

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Abstract

Background: Lipedema is a progressive disorder of subcutaneous connective tissue, predominantly affecting women, and characterized by an increase in subcutaneous adipose tissue, particularly in the lower body. This study aims to explore the gut microbiota (GM) profile in lipedema patients to characterize the associated GM and compare it with the control group. **Methods**: A prospective randomized case–control pilot study was conducted from September 2023 to May 2024, involving 55 Caucasian women, aged 20–60. The participants were divided into two groups: 35 with lipedema (LIPPY) and 20 controls (CTRL). Body composition was assessed using Dual X-ray Absorbimetry (DXA), and GM analysis was performed through 16S rRNA gene sequencing. **Results**: LIPPY subjects showed increased Intramuscular Adipose Tissue (IMAT) and reduced Lean Mass (LM)/Fat Mass (FM) ratios. While alpha and beta diversity metrics did not differ significantly between groups, differential abundance analysis identified a significant reduction in *Eggerthellaceae* (Log Fold Change (LFC) = −0.19, $p = 0.04$) and enrichment of *Propionibacteriaceae* (LFC = +0.18, $p = 0.009$) and *Acidaminococcaceae* (LFC = +0.32, $p = 0.013$) in the LIPPY group. Genus-level analysis showed a significant reduction in *Blautia* and *Ruminiclostridium* (LFC = −0.32 and −0.02; $p = 0.02$ and 0.04) and enrichment of *Anaerostipes*, *Propionibacterium*, and *Phascolarctobacterium* (LFC = +0.07, +0.17, and +0.34; $p = 0.02$, 0.005, 0.005, respectively). In correlation analyses, within LIPPY, *Eggerthellaceae* correlated negatively with Body Mass Index (BMI) ($\rho = -0.61$, $p < 0.05$) and positively with Appenicular (Appen) LM/Weight and AppenLM/BMI ($\rho = +0.43$ and +0.41, $p < 0.05$), while *Anaerostipes* correlated positively with these lean mass indices ($\rho = +0.40$, $p < 0.05$). In CTRL, only *Anaerostipes* showed a significant negative correlation with BMI ($\rho = -0.64$, $p < 0.05$). **Conclusions**: This study provides the first evidence of a distinct GM profile in LIPPY, with notable links to adverse body composition markers such as IMAT. Trial Registration: Trial registered on 24 June 2013 with ClinicalTrial.gov (NCT01890070).

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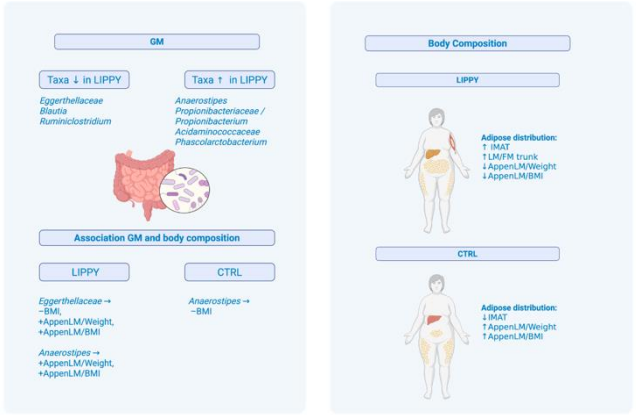


Figure 8. Summary of main results. Differences in body composition are highlighted, showing an increase in IMAT and a reduction in lean mass indices compared to fat mass in the LIPPY group. In addition, the main microbial taxa that differ between groups and their associations with BMI and appendicular lean mass indices are reported. Legend: ↑, increased; ↓, decreased; →, correlated.

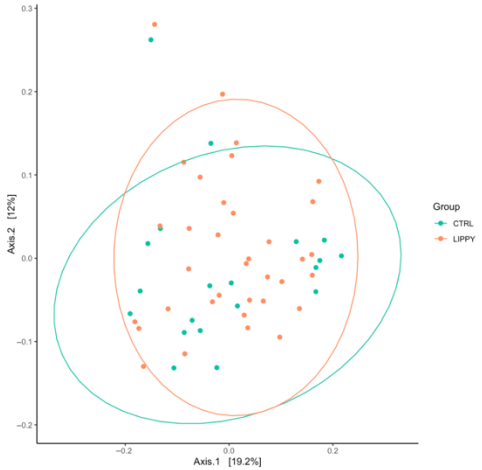


Figure 4. Beta diversity analysis using PcoA plots based on Bray–Curtis and Jaccard dissimilarity.

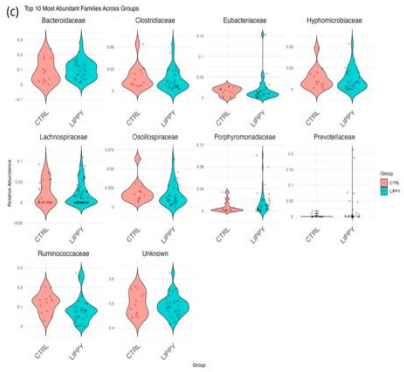


Figure 2. Microbial composition of LIPPY and CTRL groups. (**a**) Top 9 most abundant phyla, (**b**) Top 10 families, (**c**) Top 10 genera. Bar plots show mean relative abundances in each group.

Alpha diversity metrics, including Shannon and Inverse Simpson indices, did not differ significantly between groups ($p > 0.05$) (Figure 3).

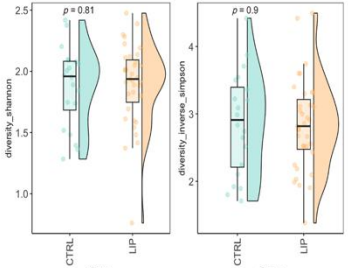


Figure 3. Alpha diversity of gut microbiota in control (CTRL) and Lipoedema (LIPPY) groups. Violin plots with overlaid boxplots and individual data points illustrate microbial alpha diversity, assessed using the Shannon index (**left**) and the Inverse Simpson index (**right**). Individual data points represent the alpha diversity values of each participant, providing a visualization of within-group variability.

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MIND Pattern Nutritional Intervention Modulates Mediterranean Diet Adherence and Gut Microbiota in Alzheimer's Disease: An Observational Case–Control Study

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Abstract

Background: Evidence on non-restrictive MIND pattern interventions in Alzheimer's (ALZ) disease remains limited. **Methods:** In an observational case–control study, 60 participants (ALZ, n = 30; cognitively healthy controls, n = 30) completed baseline (T0) and follow-up (T1) after structured MIND counseling. Adherence was assessed via the MEDAS questionnaire. Stool samples (16S rRNA profiling) were taken and anthropometry and cognitive/functional measures were recorded at T0/T1. **Results:** In the ALZ group, MEDAS improved as adherence to the Mediterranean diet increased (increasing the use of vegetables ≥ 2/day, $p < 0.01$; and lowering butter adoption ≤ 1/day, $p = 0.02$), with a shift from low to moderate/high adherence; in controls, baseline Mediterranean diet adherence was already high, and changes in MEDAS categories were modest (low adherence from 13.8% to 3.6%, high adherence from 37.9% to 50.0%), with no statistically significant overall change ($p = 0.39$). Regarding gut microbiota (GM), in the ALZ group, alpha diversity increased significantly and Bray–Curtis PCoA separated T0 from T1. Species-level analysis showed increases in SCFA-linked taxa (e.g., *Anaerobutyricum hallii*, *Blautia luti*, *Eubacterium coprostanoligenes*) and reductions in dysbiosis/mucin-degrading taxa (e.g., *Mediterraneibacter torques*, *M. gnavus*, *Agathobacter rectalis*). Between-group Δ(T1 − T0) comparisons at the genus level indicated larger positive shifts in ALZ for *Anaerobutyricum*, *Oscillibacter*, *Faecalicatena*, *Romboutsia*, *Mediterraneibacter*, and *Blautia*, and more negative Δ for *Gemmiger*, *Subdoligranulum*, *Bifidobacterium*, *Clostridium*, and *Collinsella*. sPLS-DA showed partial separation (first two components ≈ 9% variance). **Conclusions:** A structured, non-restrictive MIND intervention was feasible, improved dietary adherence, and accompanied higher diversity and compositional remodeling of the GM in ALZ's disease. Larger randomized mechanistic studies are warranted.

Check for updates

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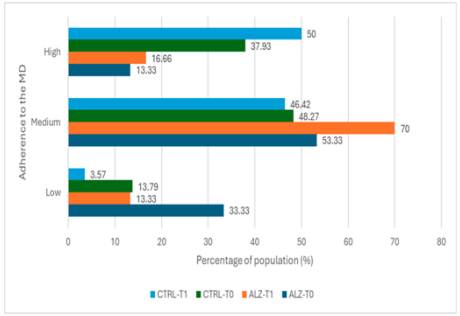


Figure 3. Distribution (%) of Mediterranean diet adherence categories (low, moderate, high; derived from MEDAS total score) in Alzheimer's and control groups at baseline and follow-up. Bars represent proportion of participants within each category.

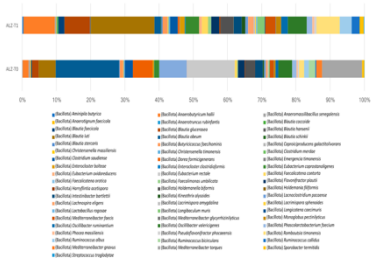


Figure 4. Species-level profiles of gut microbiota in the Alzheimer's group before (ALZ-T0) and after (ALZ-T1) the nutritional intervention. The stacked bars show the mean relative abundances (%); labeled species are those showing significant intra-group differences between T0 and T1 according to Wilcoxon signed-rank tests ($p < 0.05$).

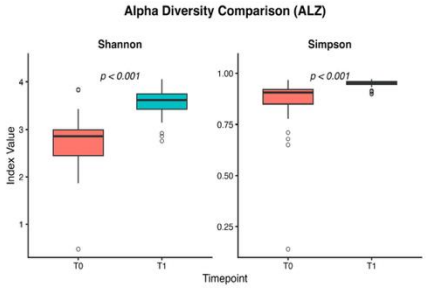


Figure 5. Boxplots of alpha diversity indices (Shannon and Simpson) in the Alzheimer's group, showing an increase in microbial richness and evenness from T0 to T1. Significant *p*-values (reported above the boxplots) were obtained with Wilcoxon signed-rank tests for paired comparisons between T0 and T1.

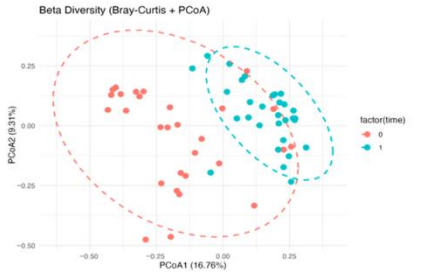


Figure 6. PCoA of gut microbiota based on Bray–Curtis distances in the Alzheimer's group, showing pre- (T0, red) and post-intervention (T1, light blue) samples with 95% confidence ellipses. Differences between T0 and T1 were tested by PERMANOVA on Bray–Curtis distances, with homogeneity of dispersion assessed by beta-dispersion.

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Effects of Italian Mediterranean Organic Diet on the Gut Microbiota: A Pilot Comparative Study with Conventional Products and Free Diet

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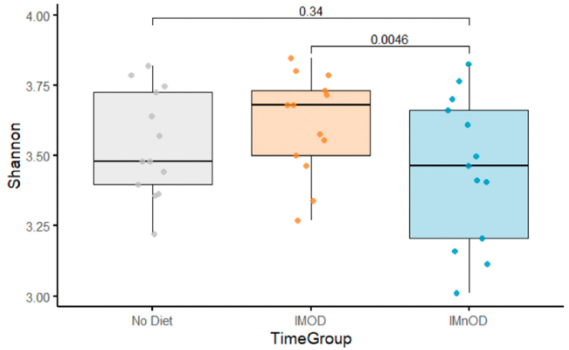
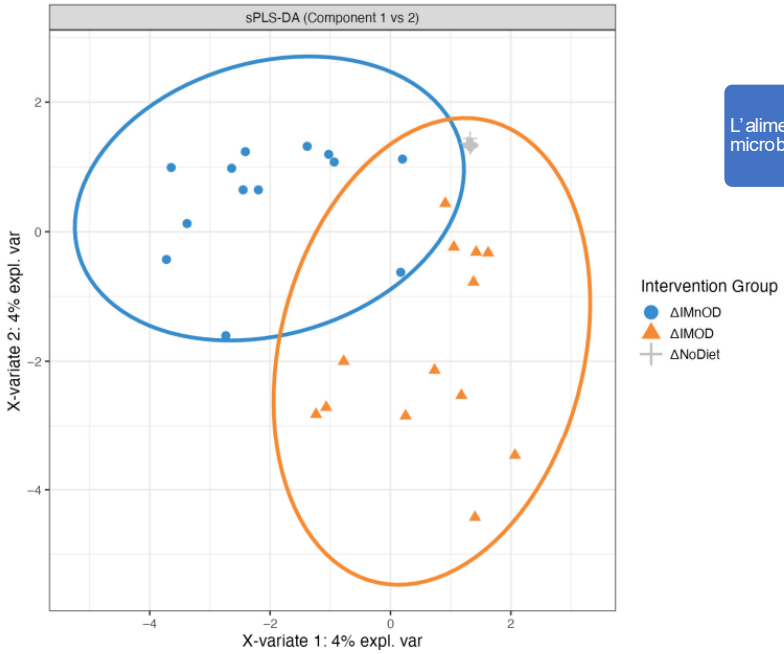


Figure 1. Boxplot of Shannon diversity values across dietary groups: No diet, IMOD, and IMnOD. Boxes represent the interquartile range, horizontal lines indicate medians, and whiskers denote minimum and maximum values. Abbreviations: IMnOD, Italian Mediterranean non-Organic Diet; IMOD, Italian Mediterranean Organic Diet; No Diet: free diet.



L'alimentazione biologica è risultata associata a una maggiore diversità alfa del microbiota intestinale rispetto a quella convenzionale.

Nei consumatori di alimenti biologici si è osservata un'abbondanza relativamente più alta di batteri produttori di SCFA (acidi grassi a corta catena), come Faecalibacterium e Roseburia.

L'alimentazione convenzionale mostrava una maggiore presenza relativa di taxa opportunisti e meno benefici rispetto al gruppo biologico.

Nei consumatori di biologico il microbiota mostrava una maggiore capacità predetta di produrre metaboliti antinfiammatori, mentre in quello convenzionale risultavano più frequenti pathway legati allo stress ossidativo.

Le differenze erano coerenti con un maggior consumo di fibre, frutta e verdura non trattate nei soggetti con dieta biologica, suggerendo un'influenza diretta delle scelte alimentari sulla struttura del microbiota.

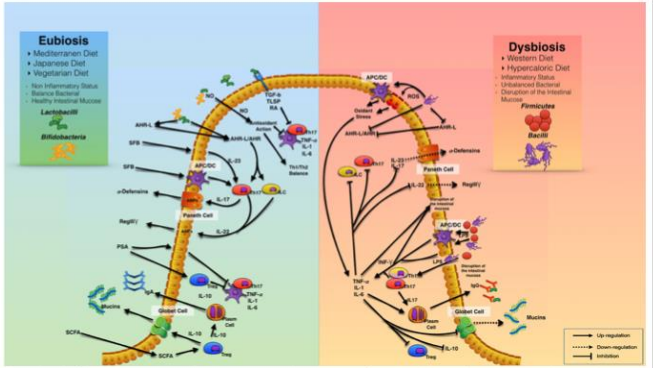


Fig. 1 The role of dietary patterns on microbiota composition and immune system function. Dysbiosis induces cytokines production and NF-kB-mediated local inflammation, promoting tumorigenesis. In the left part of the figure are shown the main mechanisms of interaction between a healthy diet, a balanced gut bacterial flora and the immune system. In the right part are shown alterations of the immune system, intestinal barrier and pathological pathways due to an unhealthy diet and unbalanced gut microbiota

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REVIEW

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The influence of diet on anti-cancer immune responsiveness

Laura Soldati[1*†], Laura Di Renzo[2†], Emilio Jirillo[3], Paolo A. Ascierto[4], Francesco M. Marincola[5] and Antonino De Lorenzo[2]

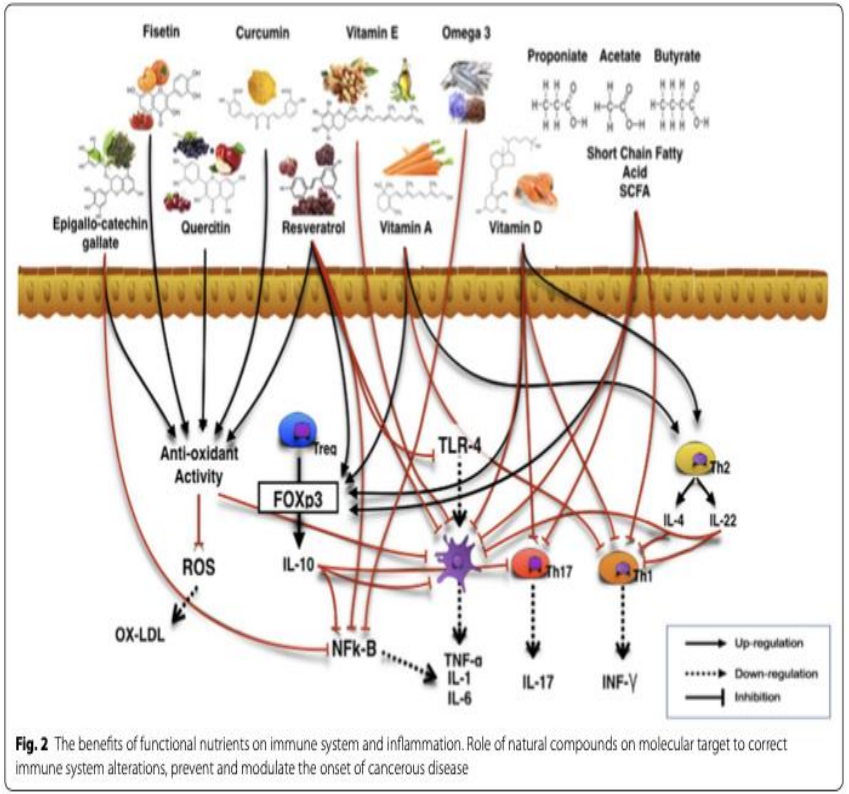


Fig. 2 The benefits of functional nutrients on immune system and inflammation. Role of natural compounds on molecular target to correct immune system alterations, prevent and modulate the onset of cancerous disease

Niente nella vita va temuto, deve essere solamente compreso
Ora è tempo di comprendere di più, così possiamo temere di meno
(Marie Curie)



Grazie per l'attenzione