IL BENESSERE CEREBRALE: AMBIENTE ED ALIMENTAZIONE

SANTA LUCIA
NEUROSCIENZE
E RIABILITAZIONE

Dr Fabrizio Piras, PhD

FABRIZIO PIRAS

Psicologo, dottore di ricerca in Neuroscienze Cognitive

- Direttore del dipartimento di *Neuroscienze Cliniche e Neuroriabilitazione*
- Responsabile dell'*Ambulatorio di Stimolazione Cognitiva*
- *Responsabile del Laboratorio di Neuropsichiatria*
- Coordinatore del Master in *Neuroriabilitazione di Alta Specialità, Metodologie Neurocognitive e Neuromotorie* (Fondazione Santa Lucia-Treccani Accademia)
- Docente di *Valutazione Neuropsicologica e Neuroriabilitazione Cognitiva*, Università Telematica Internazionale Uninettuno
- Autore di 150 pubblicazioni su riviste scientifiche internazionali, 2 libri, 6 capitoli di libro

OUTLINE

IL SISTEMA NERVOSO

L'ALIMENTAZIONE

L'AMBIENTE

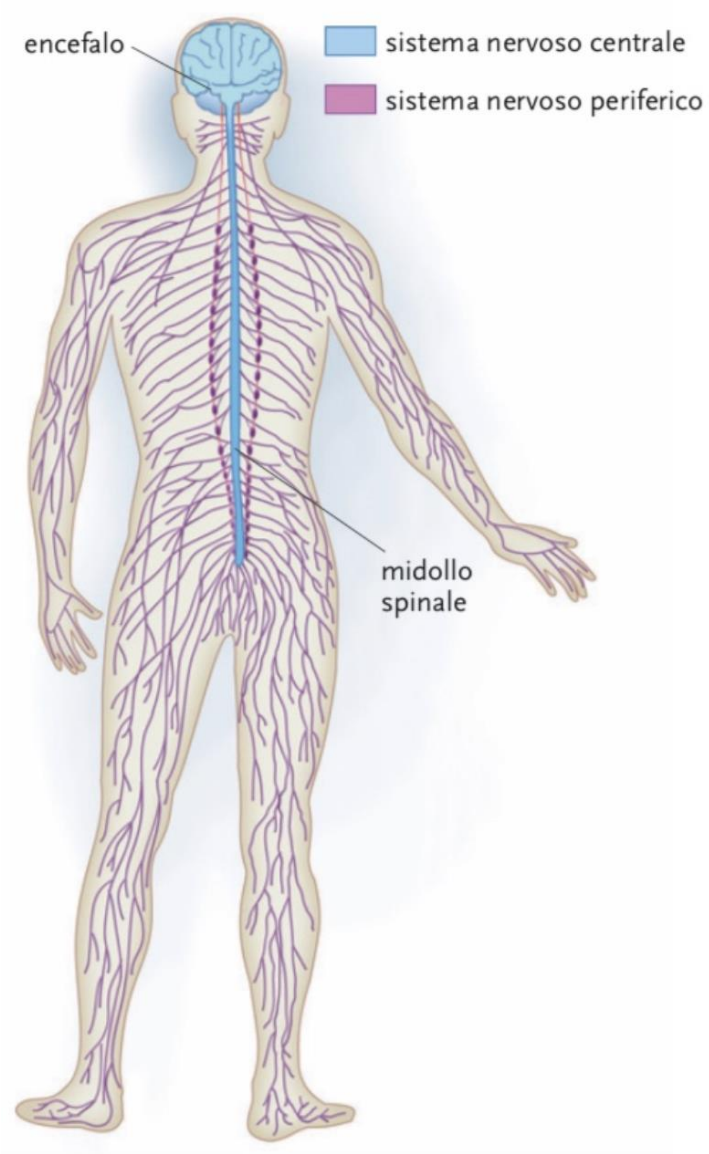
IL SISTEMA NERVOSO

Regolazione di:

- Funzioni vitali involontarie
- Attività volontarie

Formato da:

- Sistema nervoso centrale
- Sistema nervoso periferico



IL SISTEMA NERVOSO

Formato da:

- Sistema nervoso centrale
- Sistema nervoso periferico

  - Encefalo
  - Midollo spinale

IL SISTEMA NERVOSO

Formato da:

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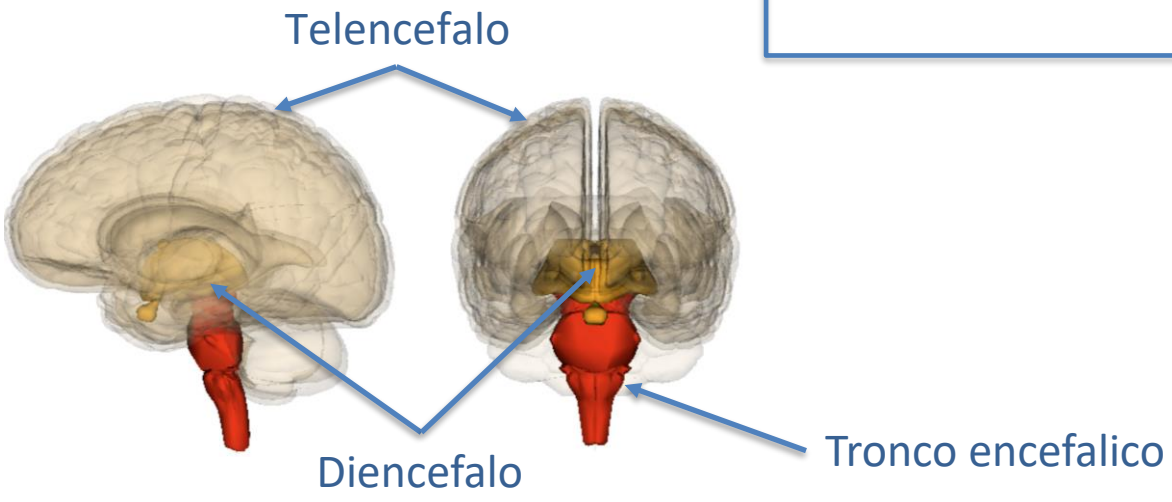
- Encefalo
- Midollo spinale

- Cervello
- Cervelletto
- Tronco encefalico

- Telencefalo
- Diencefalo



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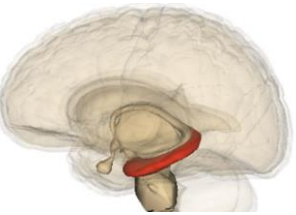
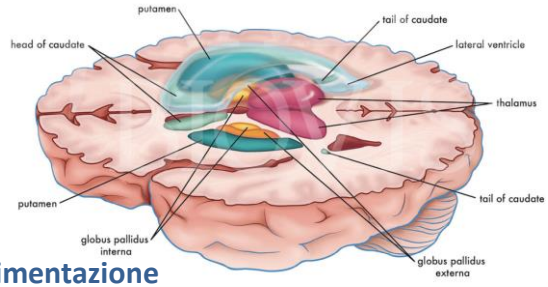
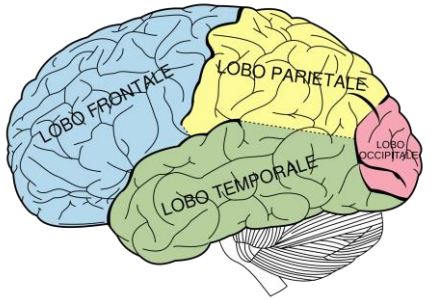
- Telencefalo
- Diencefalo

- Lobi cerebrali
- Nuclei profondi
- Ippocampo







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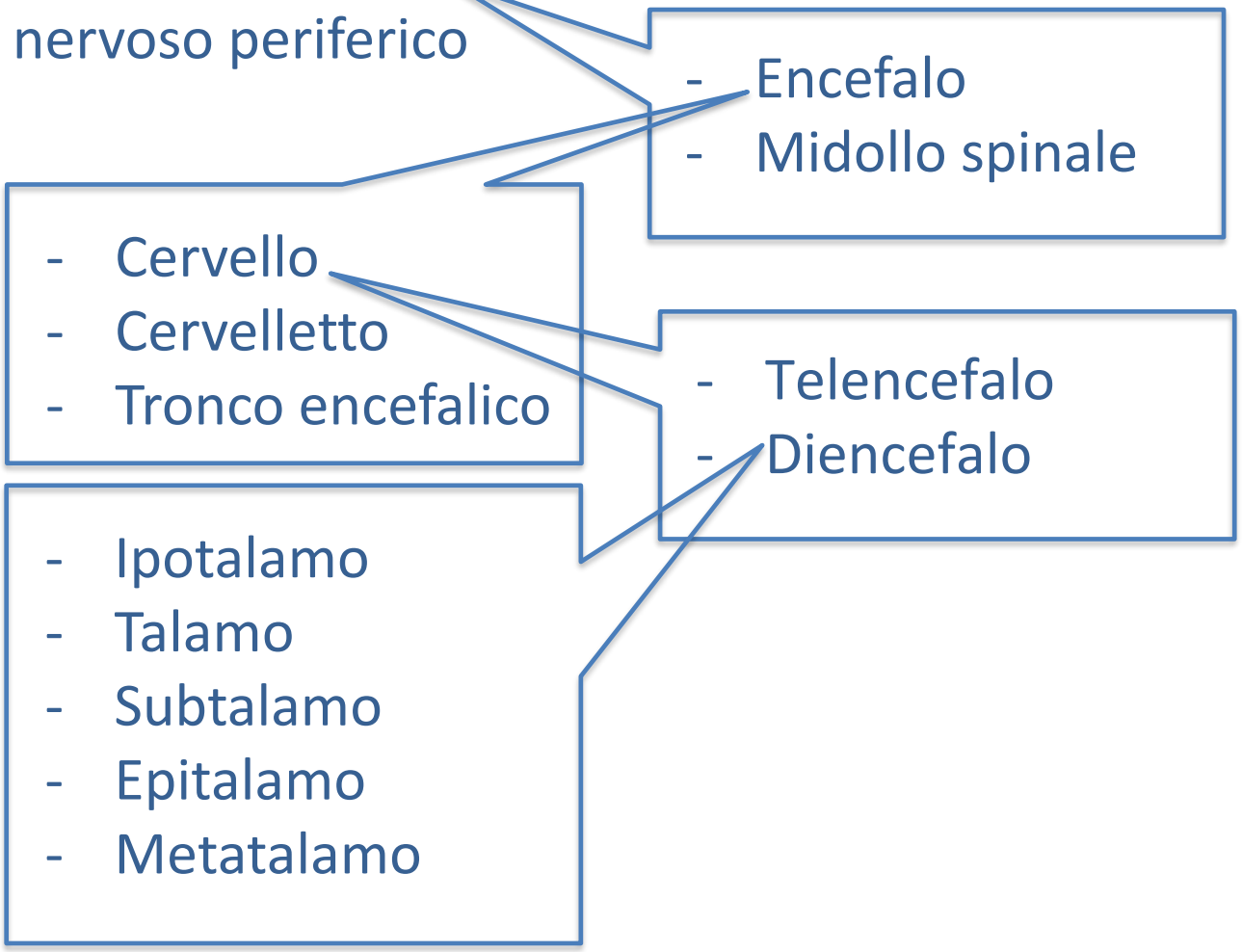
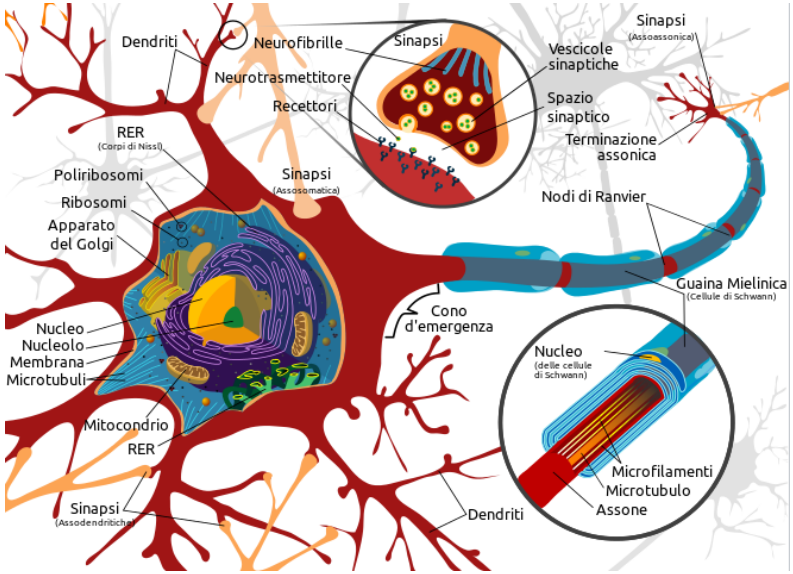
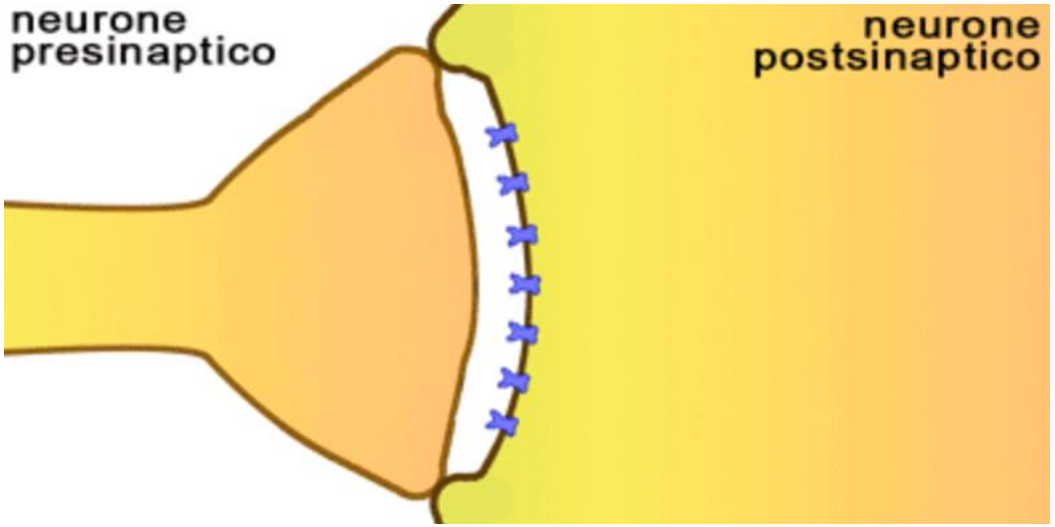


IL SISTEMA NERVOSO

Il flusso delle informazioni





- 86 miliardi di neuroni
- N sinapsi = N neuroni x 1000
- Il segnale viaggia a 432 km/h

2% del peso corporeo, 25% di energia

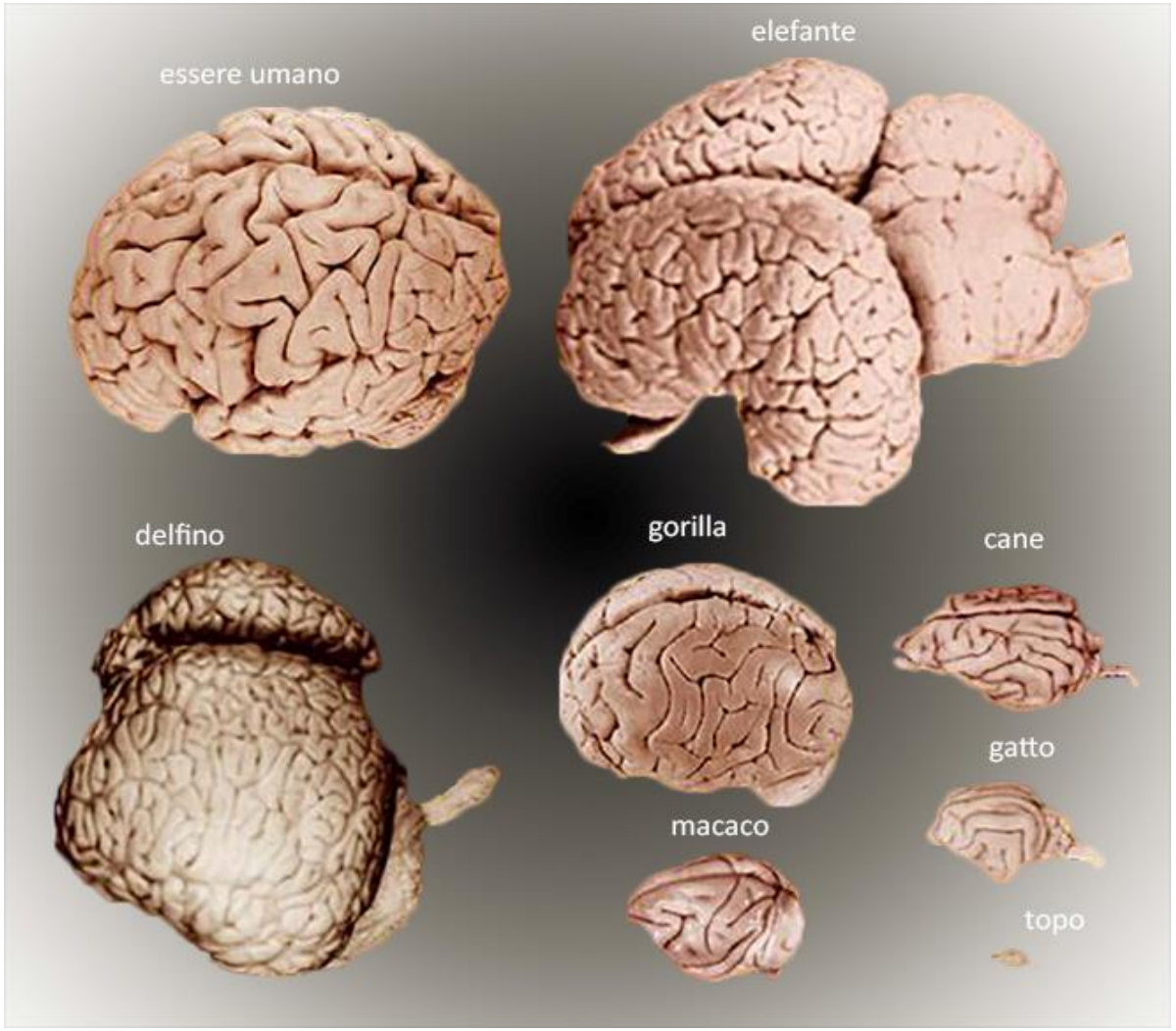
Perché il nostro cervello è speciale rispetto ad altri animali?

a) È più grande
b) Consuma meno energia
c) E’ più grande in relazione alla dimensione corporea
d) È morfologicamente più complesso

IL SISTEMA NERVOSO

Perché il nostro cervello è speciale?

E’ più grande



IL SISTEMA NERVOSO

Perché il nostro cervello è speciale?

E’ più grande

Il quoziente di encefalizzazione o QE il rapporto tra la massa del cervello e quella che ci si aspetterebbe di trovare in un tipico animale della stessa taglia.

$$C = \frac{E}{S^r}$$

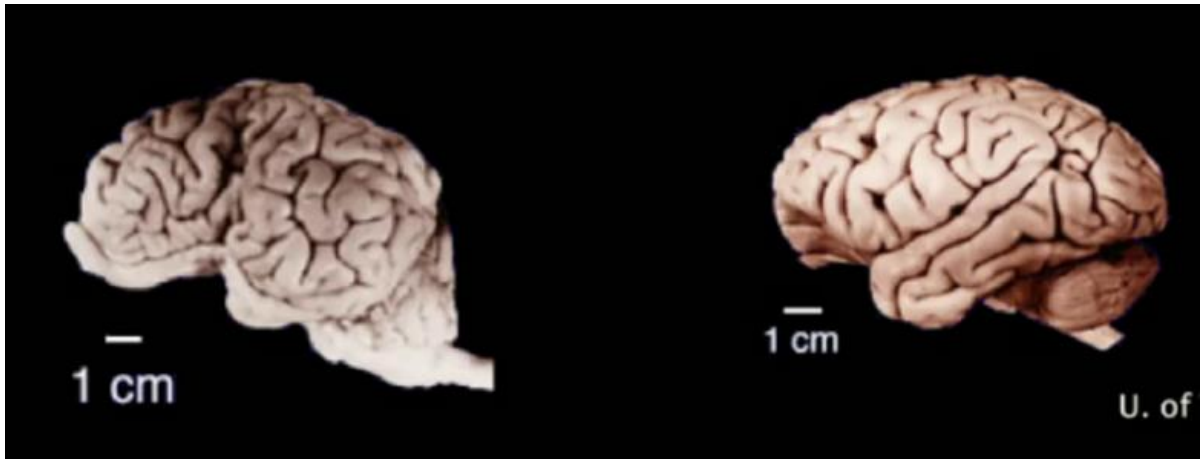
E=peso del cervello
S=peso del corpo
R= costante (per i mammiferi 0,56 e 0,66)

Uomo=6-8
Delfino=5
Scimmie= 1-3

IL SISTEMA NERVOSO

Perché il nostro cervello è speciale?

Numero di neuroni proporzionale alla grandezza



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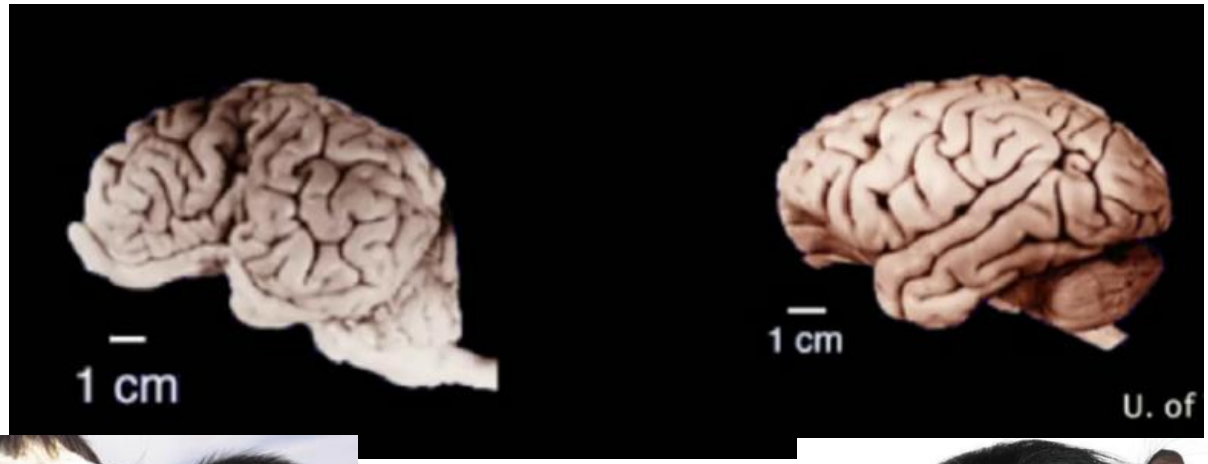

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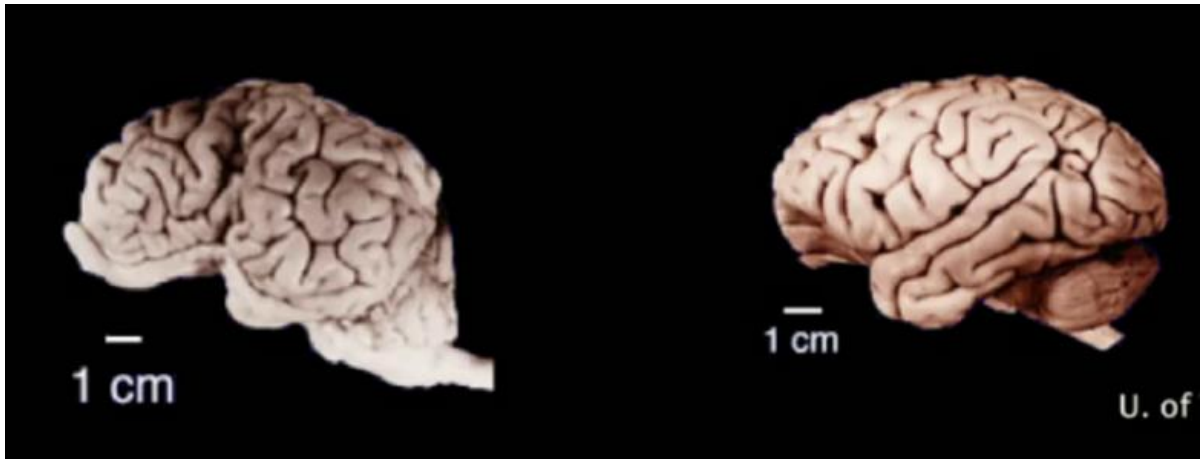


Non tutti i cervelli sono fatti allo stesso modo

IL SISTEMA NERVOSO

Perché il nostro cervello è speciale?

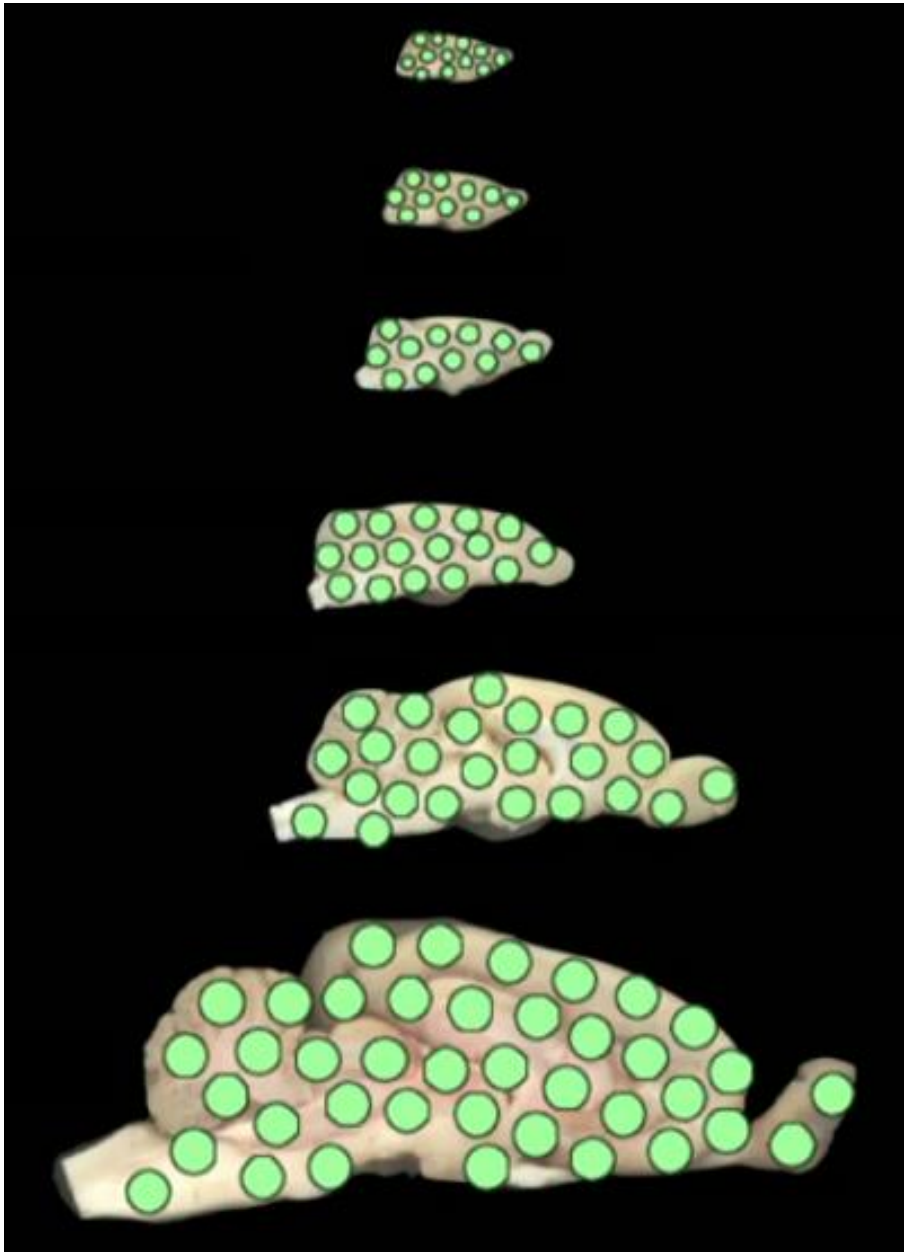
Non tutti i cervelli sono fatti allo stesso modo

Il nostro cervello è 3 volte quello di un gorilla

Pesa il 2% del nostro corpo ma consuma il 25% dell'energia

Nei grandi roditori la grandezza dei neuroni aumenta durante lo sviluppo e il cervello si gonfia

IL SISTEMA NERVOSO



IL SISTEMA NERVOSO

BRAIN SIZE AND NEURON COUNT

Cerebral cortex mass and neuron count for various mammals.

Capybara	Rhesus Macaque	Western Gorilla	Human	African Bush Elephant
non-primate	primate	primate	primate	non-primate
48.2 g	69.8 g	377 g	1232 g	2848 g
0.3 billion neurons	1.71 billion neurons	9.1 billion neurons	16.3 billion neurons	5.59 billion neurons

IL SISTEMA NERVOSO

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Non tutti i cervelli sono fatti allo stesso modo

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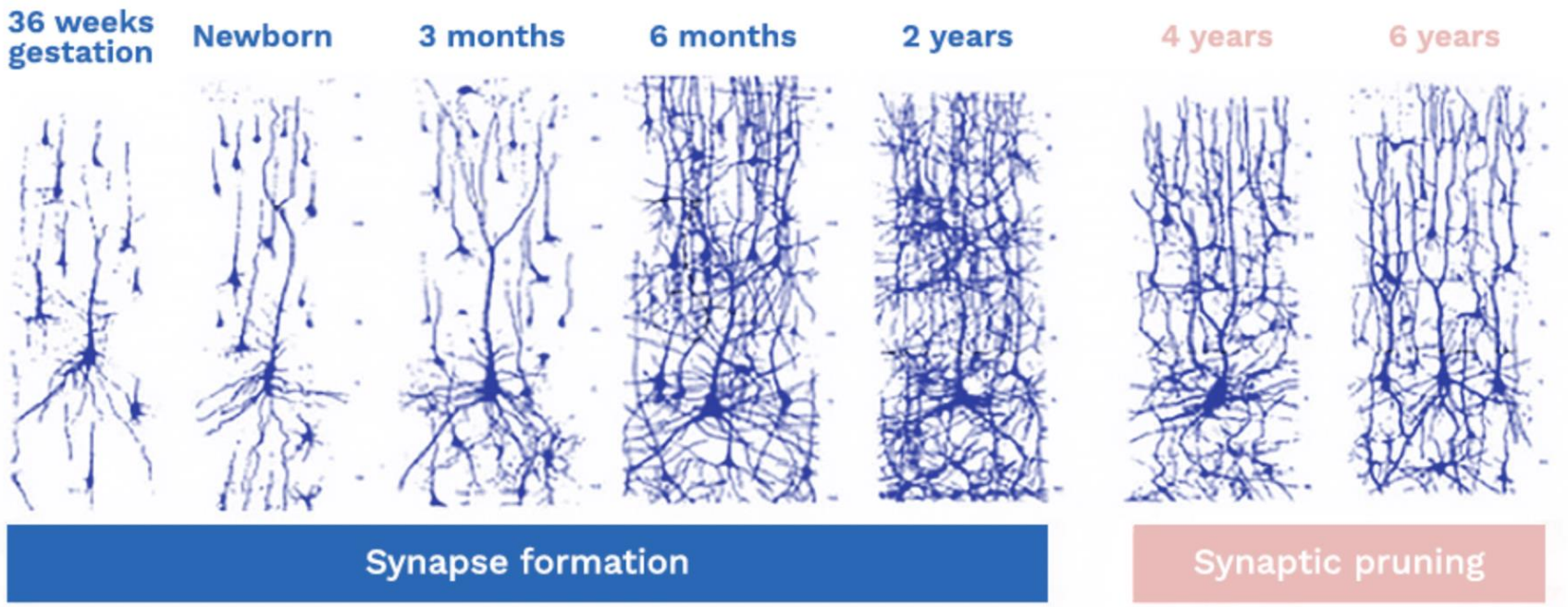
Nei primati il cervello cresce ed il numero dei neuroni rimane invariato ma crescono le fibre nervose ed il numero di connessioni

IL SISTEMA NERVOSO



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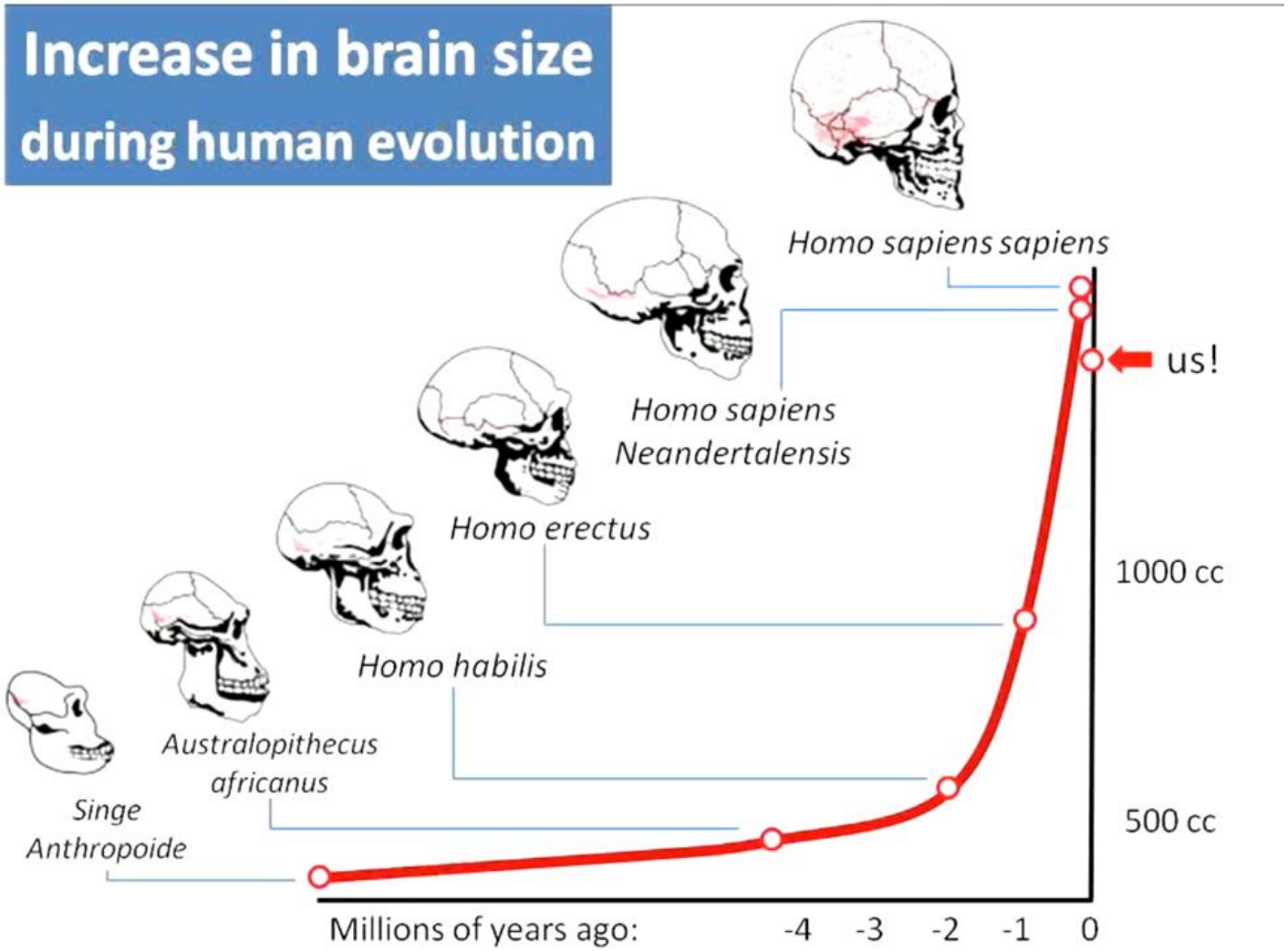

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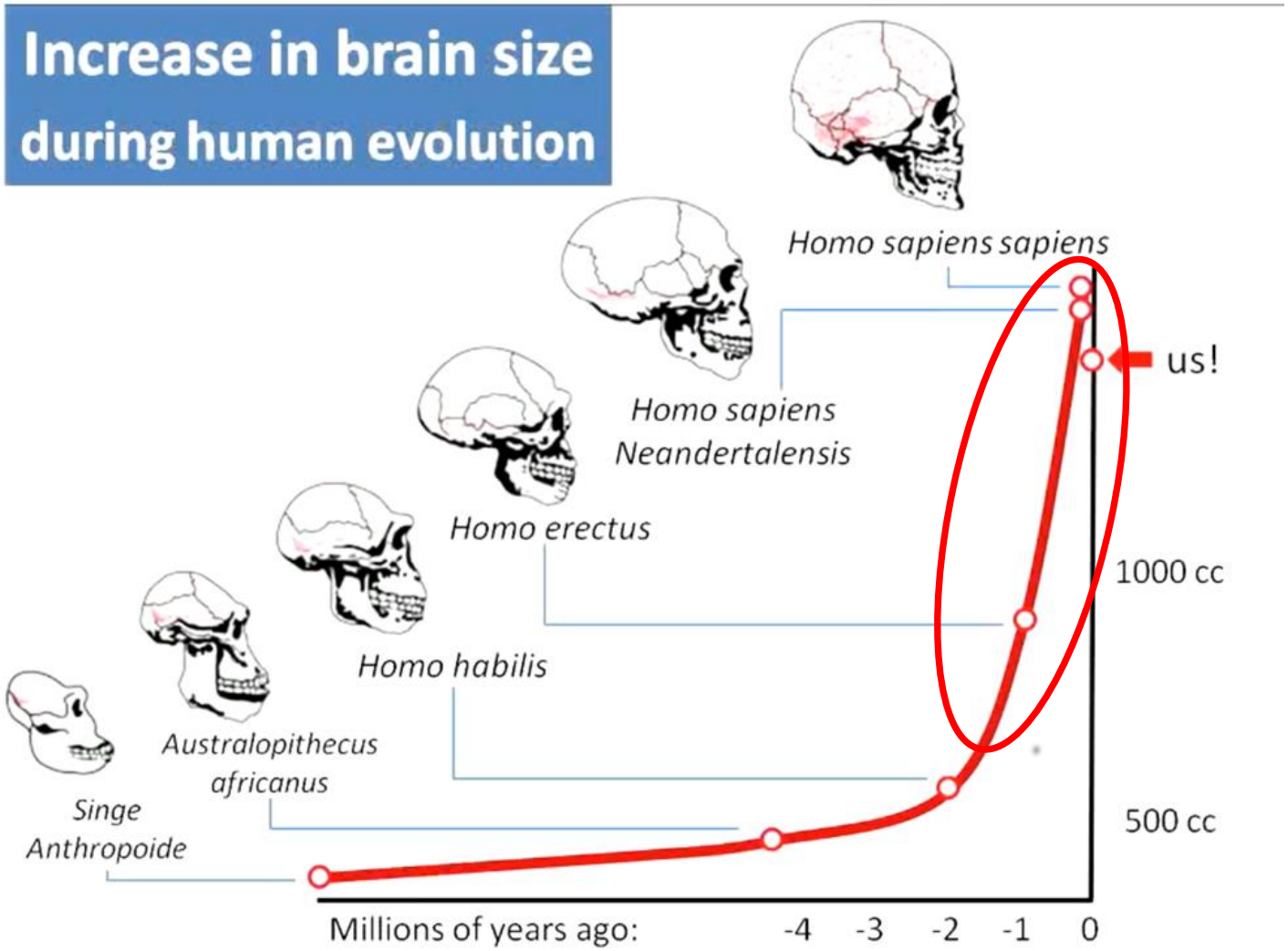

IL SISTEMA NERVOSO

Perché il nostro cervello è speciale?

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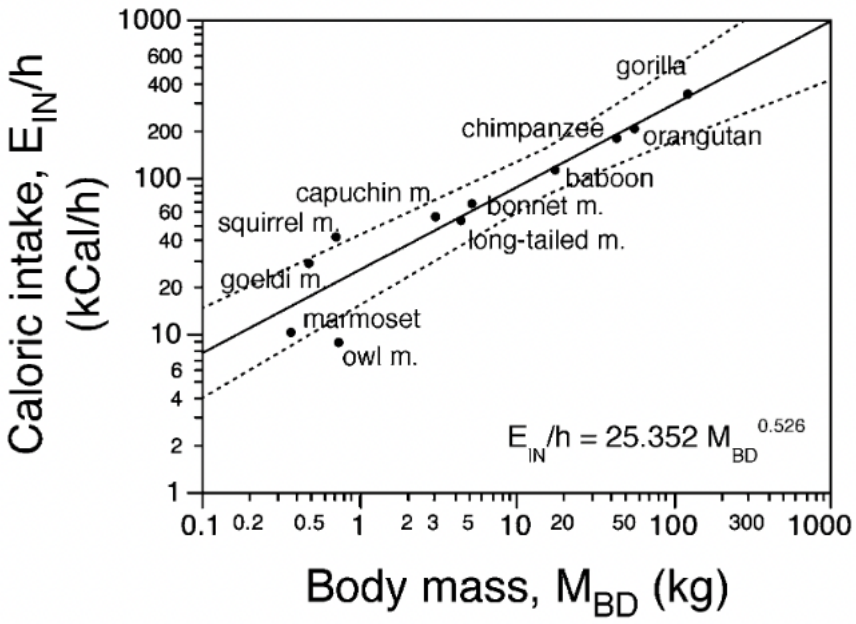
Perché il nostro cervello è speciale?

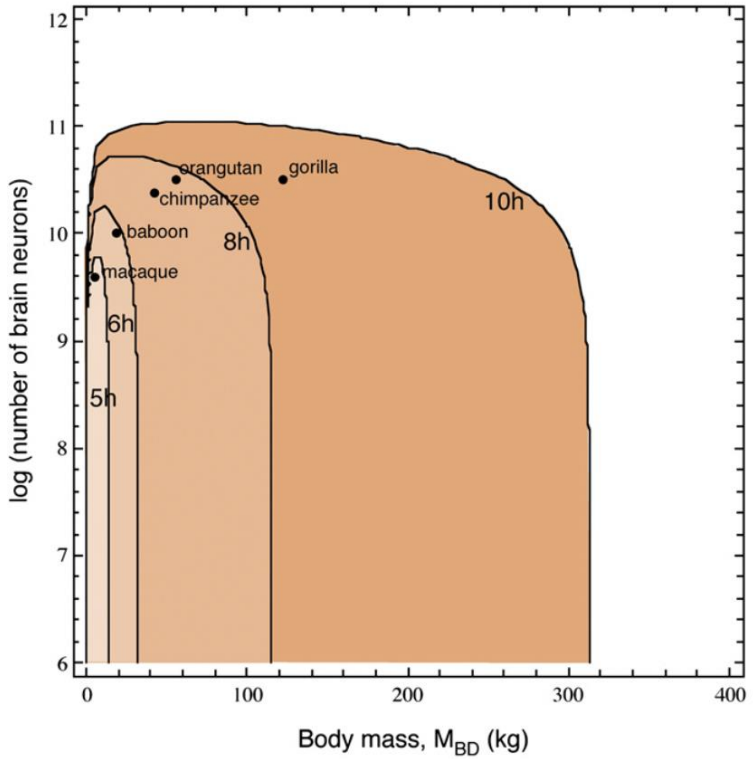
PNAS

Metabolic constraint imposes tradeoff between body size and number of brain neurons in human evolution

Karina Fonseca-Azevedo and Suzana Herculano-Houzel[1]

Instituto de Ciências Biomédicas, Universidade Federal do Rio de Janeiro, 21941-590, Rio de Janeiro, Brazil; and Instituto Nacional de Neurociência Translacional, 04023-900, São Paulo, Brazil





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Despite a general trend for larger mammals to have larger brains, humans are the primates with the largest brain and number of neurons, but not the largest body mass. Why are great apes, the largest primates, not also those endowed with the largest brains? Recently, we showed that the energetic cost of the brain is a linear function of its numbers of neurons. Here we show that metabolic limitations that result from the number of hours available for feeding and the low caloric yield of raw foods impose a tradeoff between body size and number of brain neurons, which explains the small brain size of great apes compared with their large body size. This limitation was probably overcome in *Homo erectus* with the shift to a cooked diet. Absent the requirement to spend most available hours of the day feeding, the combination of newly freed time and a large number of brain neurons affordable on a cooked diet may thus have been a major positive driving force to the rapid increased in brain size in human evolution.

CERVELLO E ALIMENTAZIONE

Neurogenesi

Plasticità sinaptica

Genes Nutr (2009) 4:271–282
DOI 10.1007/s12263-009-0134-5

REVIEW

Neurogenesi

Impact of diet on adult hippocampal neurogenesis

Doris Stangl · Sandrine Thuret

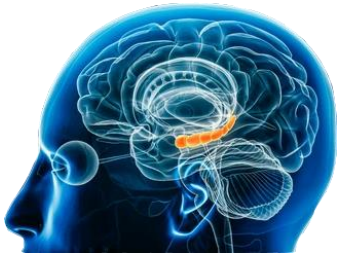


Table 1 Modulation of adult hippocampal neurogenesis (AHN) by diet

Diet	Study models	Effect on AHN	References
Caloric restriction/dietary restriction	Rat	Increased survival	[60]
	Mouse	Increased survival	[62], [61], [54], [8]
Omega 3 fatty acids	Rat	Increased (DHA)	[46]
Flavonoids	Rat, chronically stressed	Increased proliferation	[3]
Blueberry	Rat	Increased proliferation	[12]
Curcumin low concentrations	Mouse	Increased proliferation	[53]
Retinoic acid excess	Mouse	Decreased proliferation	[16]
Vitamin A deficiency	Rat	Decreased proliferation (rescued with retinoic acid)	[9]
Thiamine deficiency	Mouse	Decreased proliferation/survival	[128]
Zinc deficiency	Rat male	Decreased proliferation/survival	[14]
Folate deficiency	Mouse	Inhibited proliferation	[56]
Increased homocysteine	Mouse	Inhibited proliferation	[93]
			[57]
High fat	Male rat	Decreased proliferation	[67]
	Female rat	No change	
Soft diet	Rat	Decreased proliferation	[4]
Caffeine			
At physiologically relevant doses	Mouse	Decreased proliferation	[121]
At supraphysiological doses	Mouse	Increased proliferation/decreased survival	[121]
Low doses, chronically	Rat	Decreased proliferation	[36]
Ethanol	Rat	Decreased proliferation	[84], [37]
	Mouse	Decreased proliferation	[103]

CERVELLO E ALIMENTAZIONE

frontiers | Frontiers in Neuroscience

TYPE Mini Review
PUBLISHED 23 February 2023
DOI 10.3389/fnins.2023.1147269

Neurogenesi

Check for updates

OPEN ACCESS

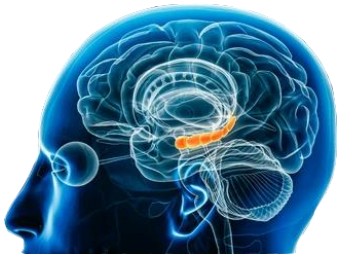
EDITED BY
Sumana Chakravarty,
Indian Institute of Chemical Technology (CSIR), India

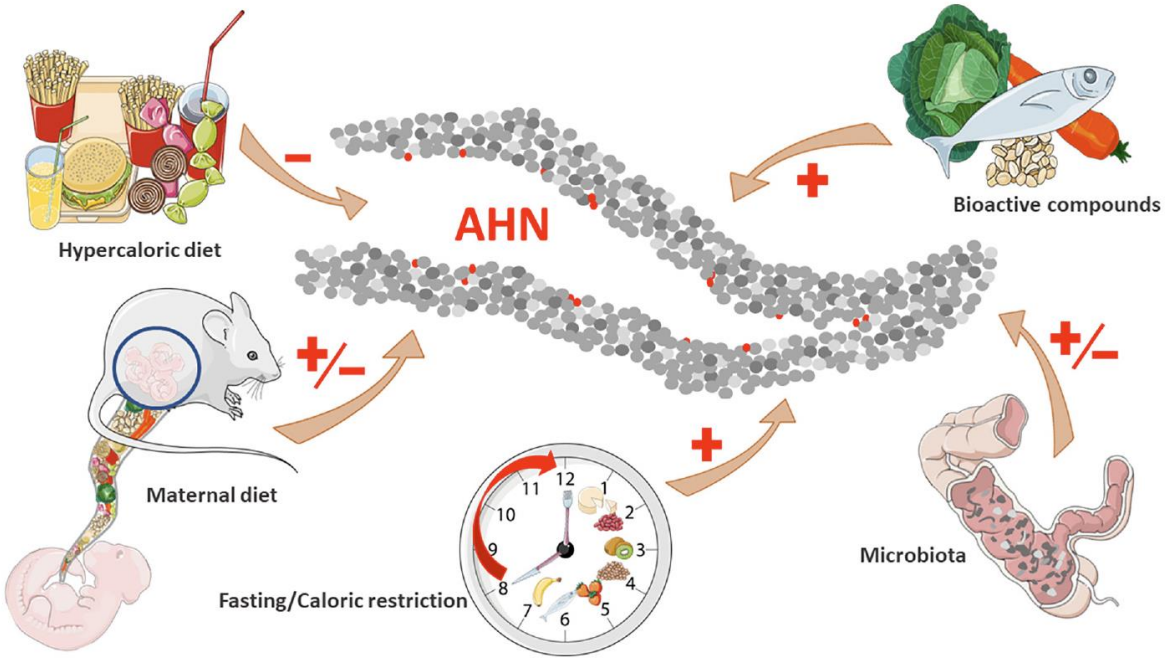
REVIEWED BY
Daniel Radford-Smith,
University of Oxford, United Kingdom

*CORRESPONDENCE
Estela Castilla-Ortega

Nutrition and adult neurogenesis in the hippocampus: Does what you eat help you remember?

Sonia Melgar-Locatelli[1,2†], Marialuisa de Ceglia[1,3†], M. Carmen Mañas-Padilla[1,2], Celia Rodriguez-Pérez[4,5,6], Estela Castilla-Ortega[1,2*], Adriana Castro-Zavala[1,2*] and Patricia Rivera[1,3*]





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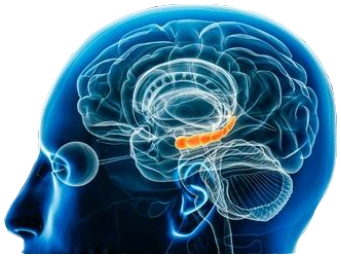

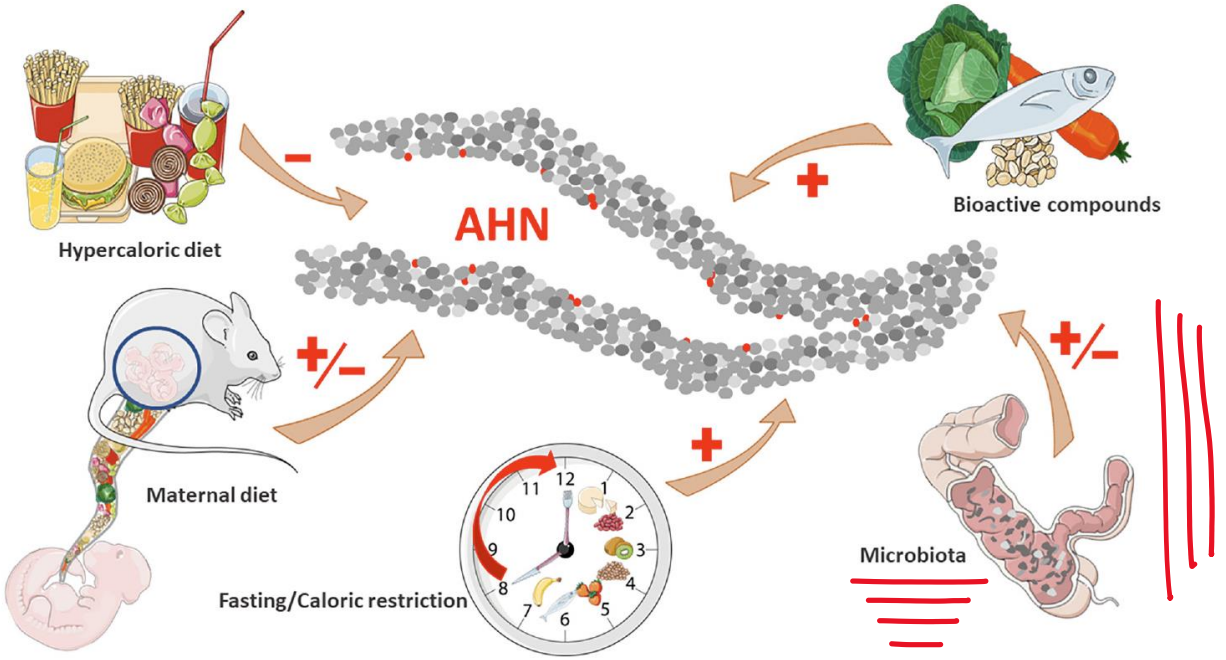

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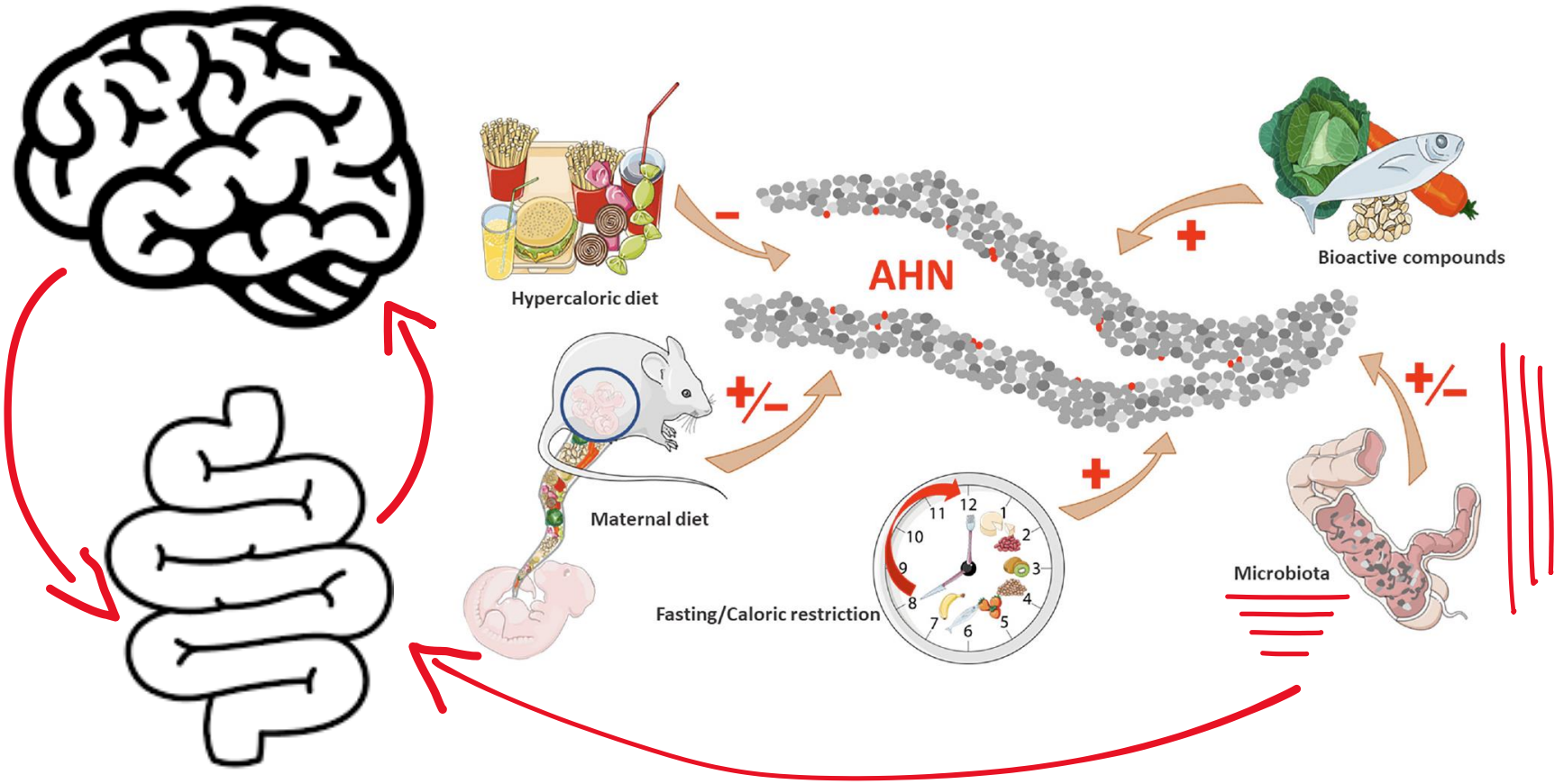


nutrients

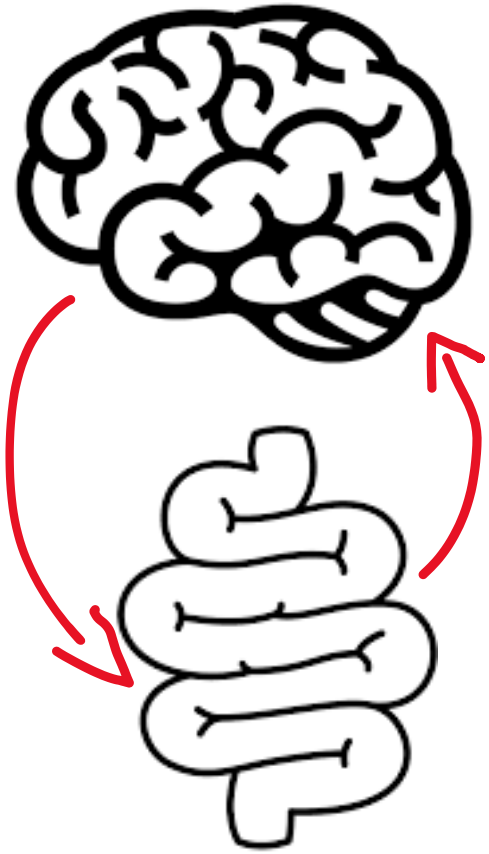
MDPI

Article

Long-Term Diet Supplementation with *Lactobacillus paracasei* K71 Prevents Age-Related Cognitive Decline in Senescence-Accelerated Mouse Prone 8

Henry M. Corpuz [1,2], Saki Ichikawa [3], Misa Arimura [3], Toshihiro Mihara [4], Takehisa Kumagai [4], Takakazu Mitani [5], Soichiro Nakamura [1] and Shigeru Katayama [1,5,*]

Neuroplasticità

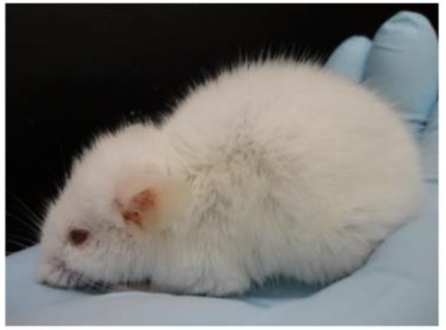


Lactobacillus paracasei K71

43 weeks feeding

Cognitive performance↑

BDNF↑ pCREB↑ Serotonin↑



SAMP8

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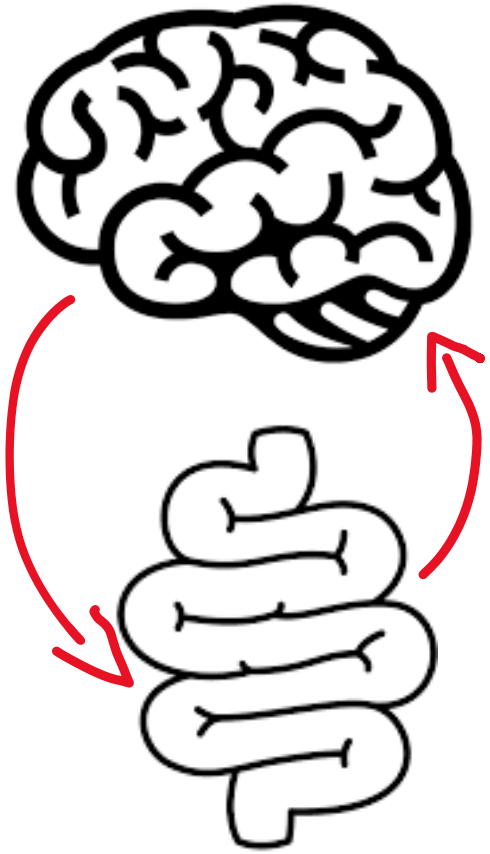
MINI REVIEW
published: 30 January 2017
doi: 10.3389/fnbeh.2017.00009

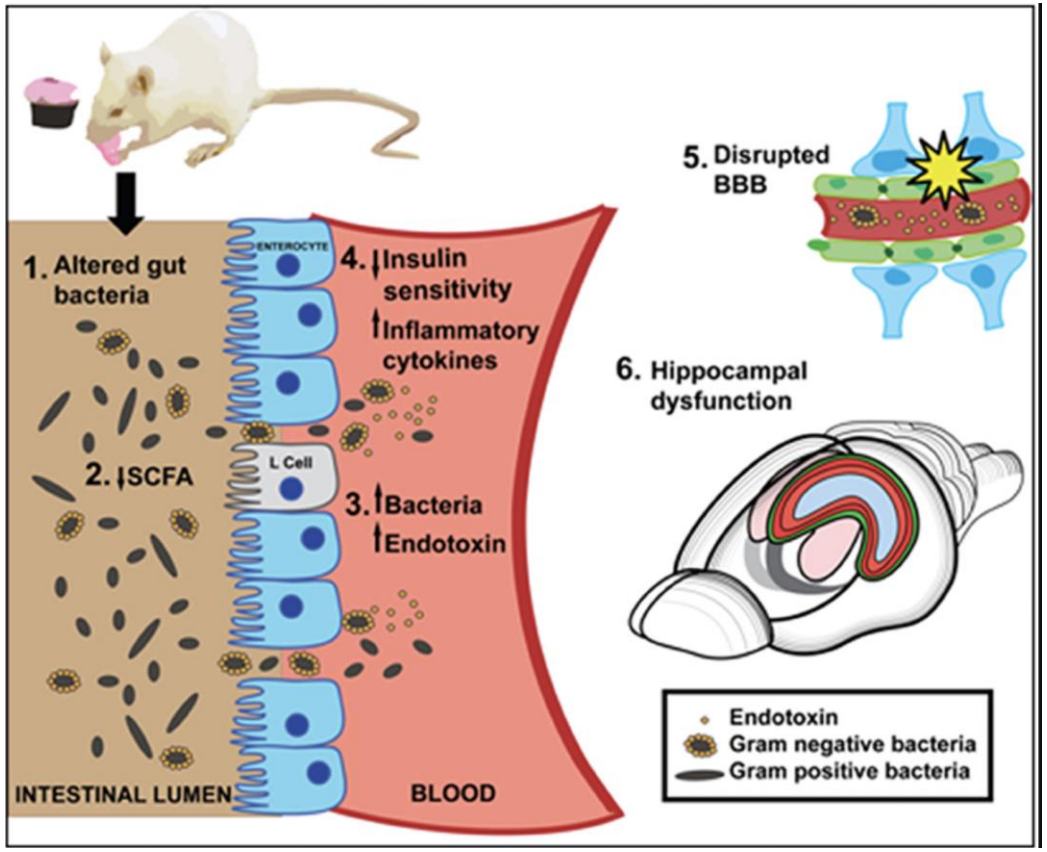
Neuroplasticità

Gut to Brain Dysbiosis: Mechanisms Linking Western Diet Consumption, the Microbiome, and Cognitive Impairment

Emily E. Noble[1], *Ted M. Hsu*[1,2] *and Scott E. Kanoski*[1,2]*

[1] Human and Evolutionary Biology Section, Department of Biological Sciences, University of Southern California, Los Angeles, CA, USA, [2] Neuroscience Program, University of Southern California, Los Angeles, CA, USA



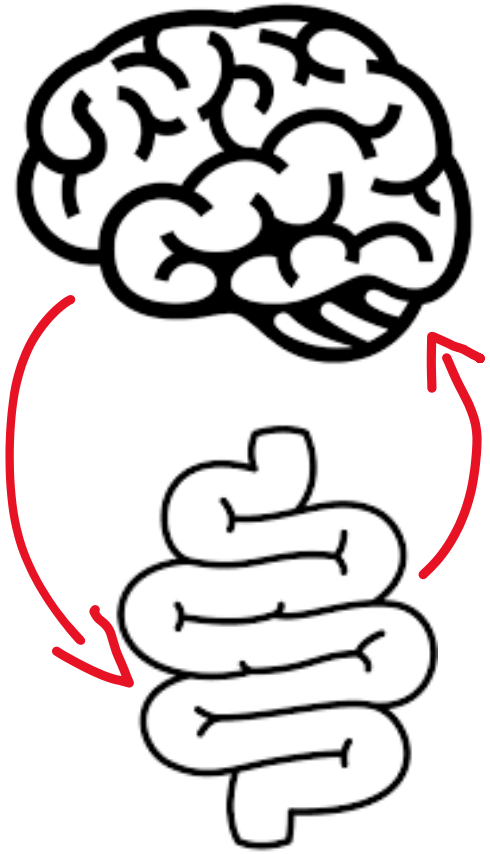


frontiers in Behavioral Neuroscience
ORIGINAL RESEARCH
published: 21 November 2016
doi: 10.3389/fnbeh.2016.00225

Neuroplasticità

Switching Adolescent High-Fat Diet to Adult Control Diet Restores Neurocognitive Alterations

Chloé Boitard[1,2†], Shauna L. Parkes[1,2,3†], Amandine Cavaroc[1,2], Frédéric Tantot[1,2], Nathalie Castanon[1,2], Sophie Layé[1,2], Sophie Tronel[2,4], Gustavo Pacheco-Lopez[5], Etienne Coutureau[2,3] and Guillaume Ferreira[1,2]*



In addition to metabolic and cardiovascular disorders, obesity is associated with adverse cognitive and emotional outcomes. Its growing prevalence in adolescents is particularly alarming since this is a period of ongoing maturation for brain structures (including the hippocampus and amygdala) and for the hypothalamic-pituitary-adrenal (HPA) stress axis, which is required for cognitive and emotional processing. We recently demonstrated that adolescent, but not adult, high-fat diet (HF) exposure leads to impaired hippocampal function and enhanced amygdala function through HPA axis alteration (Boitard et al., 2012, 2014, 2015). Here, we assessed whether the effects of adolescent HF consumption on brain function are permanent or reversible. After adolescent exposure to HF, switching to a standard control diet restored levels of hippocampal neurogenesis and normalized enhanced HPA axis reactivity, amygdala activity and avoidance memory. Therefore, while the adolescent period is highly vulnerable to the deleterious effects of diet-induced obesity, adult exposure to a standard diet appears sufficient to reverse alterations of brain function.

CERVELLO E ALIMENTAZIONE

FOOD FOR THOUGHT 2020

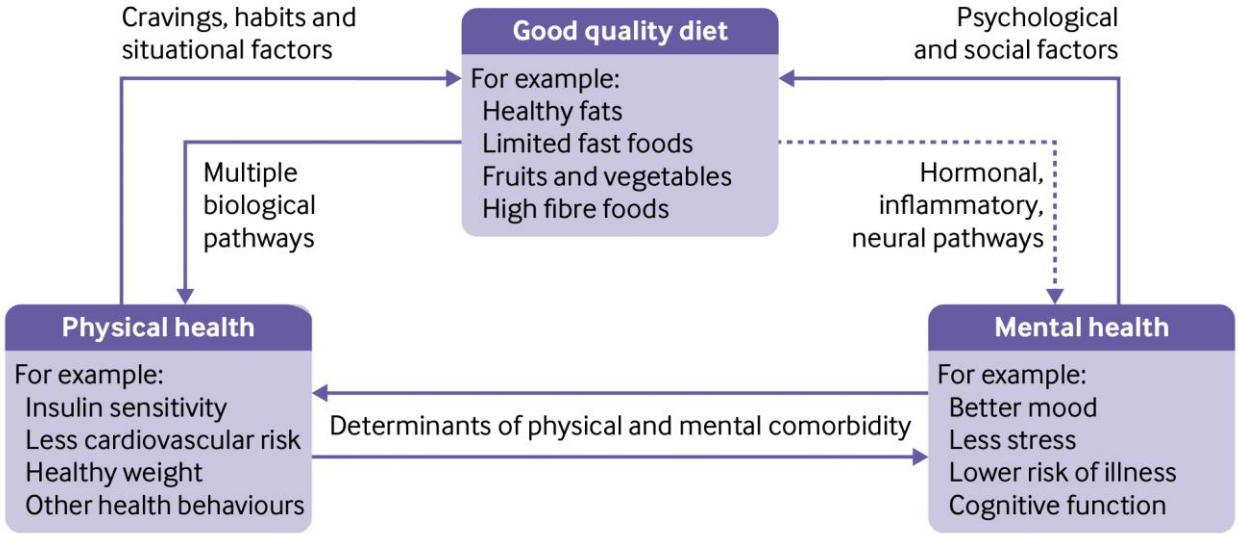
Food and mood: how do diet and nutrition affect mental wellbeing?

Poor nutrition may be a causal factor in the experience of low mood, and improving diet may help to protect not only the physical health but also the mental health of the population, say **Joseph Firth and colleagues**

BMJ: first published as 10.1136/bm

KEY MESSAGES

- Healthy eating patterns, such as the Mediterranean diet, are associated with better mental health than "unhealthy" eating patterns, such as the Western diet
- The effects of certain foods or dietary patterns on glycaemia, immune activation, and the gut microbiome may play a role in the relationships between food and mood
- More research is needed to understand the mechanisms that link food and mental wellbeing and determine how and when nutrition can be used to improve mental health



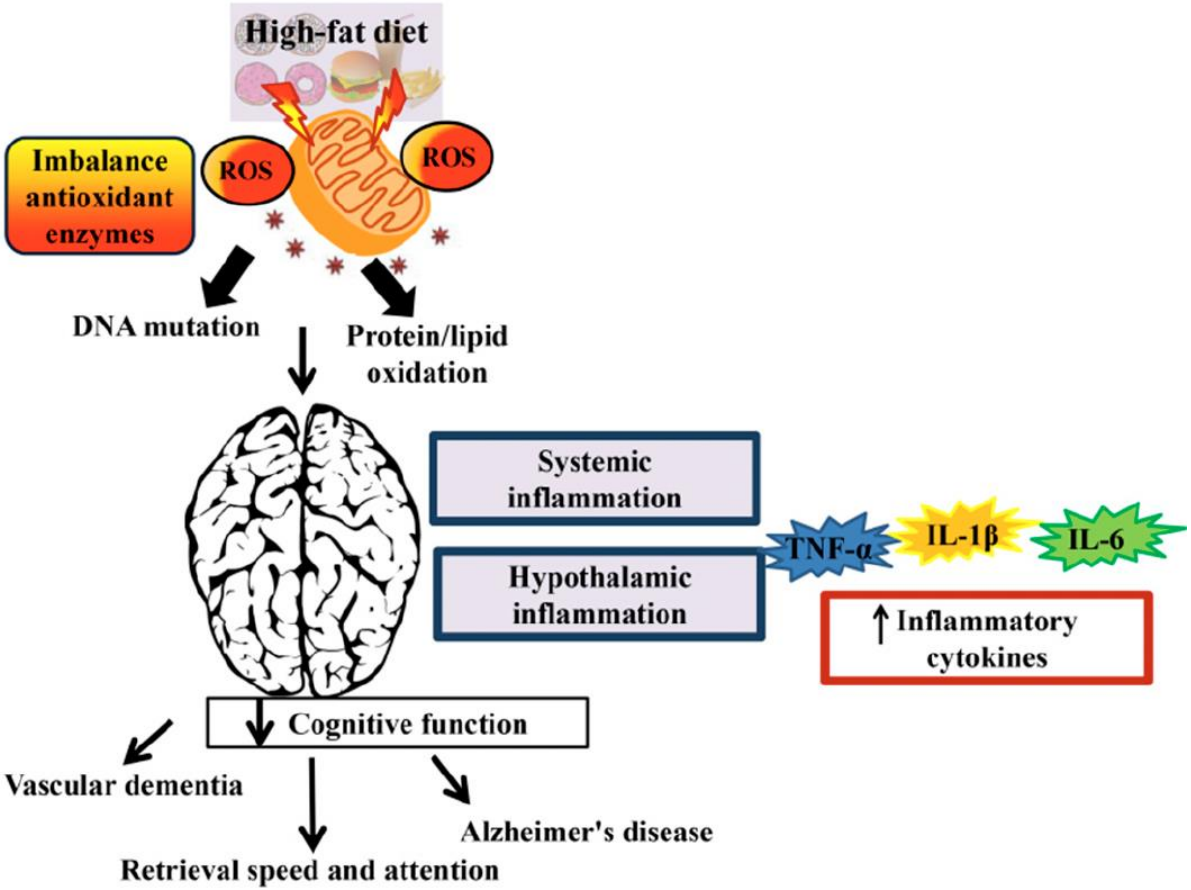
CERVELLO E ALIMENTAZIONE

nutrients Nutrients 2019, 11, 2579; doi:10.3390/nu11112579 MDPI

Review

Effect of High-Fat Diets on Oxidative Stress, Cellular Inflammatory Response and Cognitive Function

Bee Ling Tan [1] and Mohd Esa Norhaizan [1,2,3,*]



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nutrients *Nutrients* **2023**, *15*, 1436. https://doi.org/10.3390/nu15061436 MDPI

Review

The Role of Diet as a Modulator of the Inflammatory Process in the Neurological Diseases

Antonina Kurowska, Wojciech Ziemichód, Mariola Herbet and Iwona Piątkowska-Chmiel *

Abstract: Neurological diseases are recognized as major causes of disability and mortality worldwide. Due to the dynamic progress of diseases such as Alzheimer's disease (AD), Parkinson's Disease (PD), Schizophrenia, Depression, and Multiple Sclerosis (MD), scientists are mobilized to look for new and more effective methods of interventions. A growing body of evidence suggests that inflammatory processes and an imbalance in the composition and function of the gut microbiome, which play a critical role in the pathogenesis of various neurological diseases and dietary interventions, such as the Mediterranean diet the DASH diet, or the ketogenic diet can have beneficial effects on their course. The aim of this review was to take a closer look at the role of diet and its ingredients in modulating inflammation associated with the development and/or progression of central nervous system diseases. Presented data shows that consuming a diet abundant in fruits, vegetables, nuts, herbs, spices, and legumes that are sources of anti-inflammatory elements such as omega-3 fatty acids, polyphenols, vitamins, essential minerals, and probiotics while avoiding foods that promote inflammation, create a positive brain environment and is associated with a reduced risk of neurological diseases. Personalized nutritional interventions may constitute a non-invasive and effective strategy in combating neurological disorders.

Herbs and Spices	Major Bioactive Compounds	Potential Beneficial Effects	Anti-Inflammatory Mechanism	References
Garlic (*Allium sativum* L.)	allicin, quercetin, kaempferol	anti-inflammatory	suppress the production of inflammatory cytokines	[55]
Turmeric (*Curcuma longa* L.)	curcumin, demethoxycurcumin, bisdemethoxycurcumin	anti-inflammatory	inhibits microglial activation and reduces cytokine release	[54]
Ginger (*Zingiber officinale* Roscoe)	gingerols, shogaols, paradols	anti-inflammatory antioxidant analgesic	Inhibit cyclooxygenase (COX) and 5-lipoxygenase (LOX), and downregulation of the expression of pro-inflammatory genes suppress the production of	[56–58]
Rosemary (*Rosmarinus officinalis* L.)	carnosic acid, betulinic acid, carnosol, ursolic acid	anti-inflammatory antioxidant	inhibit the release of pro-inflammatory mediators like NOx, IL-1, and TNF-1α while reducing leukocyte activation	[59–61]

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Research

JAMA Neurology | Original Investigation

Association Between Consumption of Ultraprocessed Foods and Cognitive Decline

Natalia Gomes Gonçalves, PhD; Naomi Vidal Ferreira, PhD; Neha Khandpur, ScD; Euridice Martinez Steele, PhD; Renata Bertazzi Levy, PhD; Paulo Andrade Lotufo, MD, PhD; Isabela M. Bensenor, PhD; Paulo Caramelli, MD, PhD; Sheila Maria Alvim de Matos, PhD; Dirce M. Marchioni, PhD; Claudia Kimie Suemoto, MD, PhD

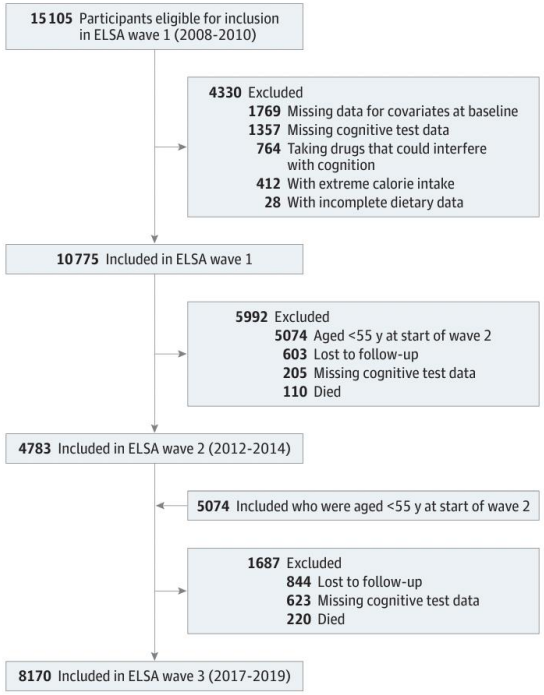
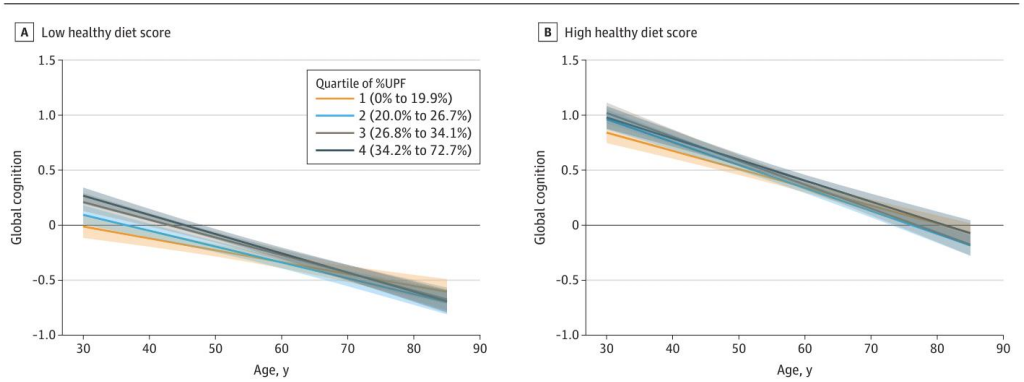


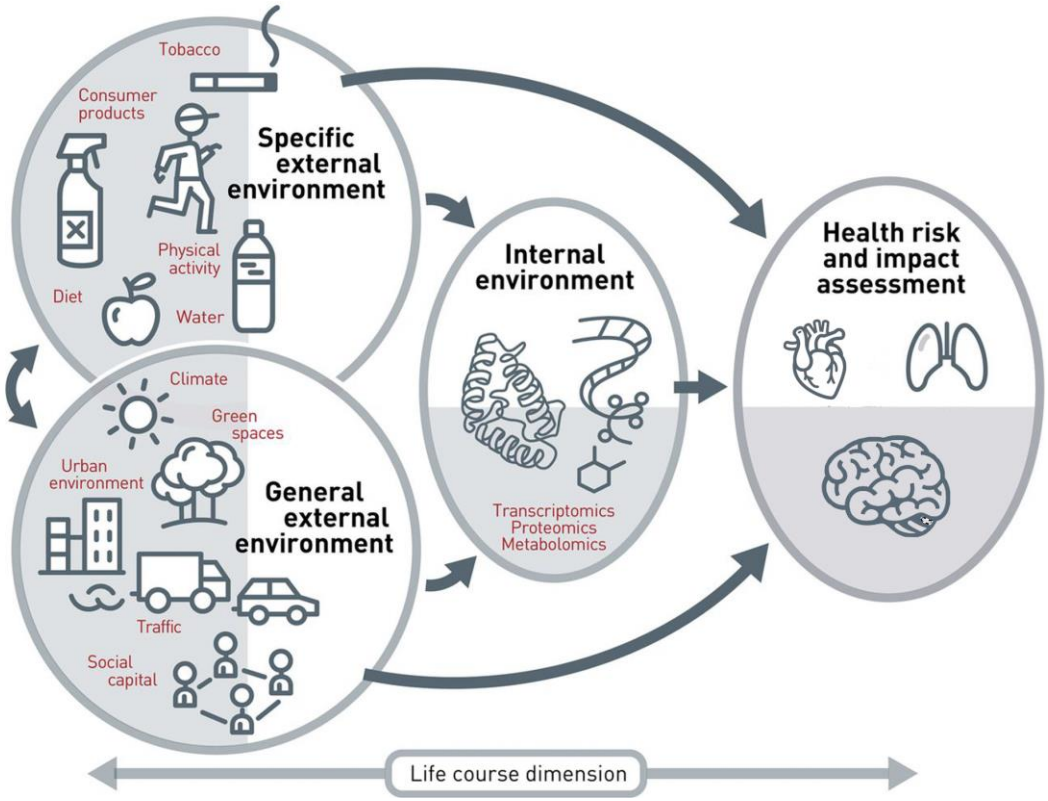
Figure 3. Trajectories of Global Cognitive Performances Over Time in Participants With Low Healthy Diet Scores and High Healthy Diet Scores



RESULTS A total of 15 105 individuals were recruited and 4330 were excluded, leaving 10 775 participants whose data were analyzed. The mean (SD) age at the baseline was 51.6 (8.9) years, 5880 participants (54.6%) were women, 5723 (53.1%) were White, and 6106 (56.6%) had at least a college degree. During a median (range) follow-up of 8 (6-10) years, individuals with ultraprocessed food consumption above the first quartile showed a 28% faster rate of global cognitive decline (β = −0.004; 95% CI, −0.006 to −0.001; *P* = .003) and a 25% faster rate of executive function decline (β = −0.003, 95% CI, −0.005 to 0.000; *P* = .01) compared with those in the first quartile.

Esposoma

la totalità dell'esposizione dal concepimento alla fine della vita, comprese le esposizioni a sostanze chimiche esogene e a prodotti naturali, nonché a sostanze chimiche generate internamente in risposta a un insulto tossico o a fattori legati allo stile di vita, come la dieta, il fumo e lo stress



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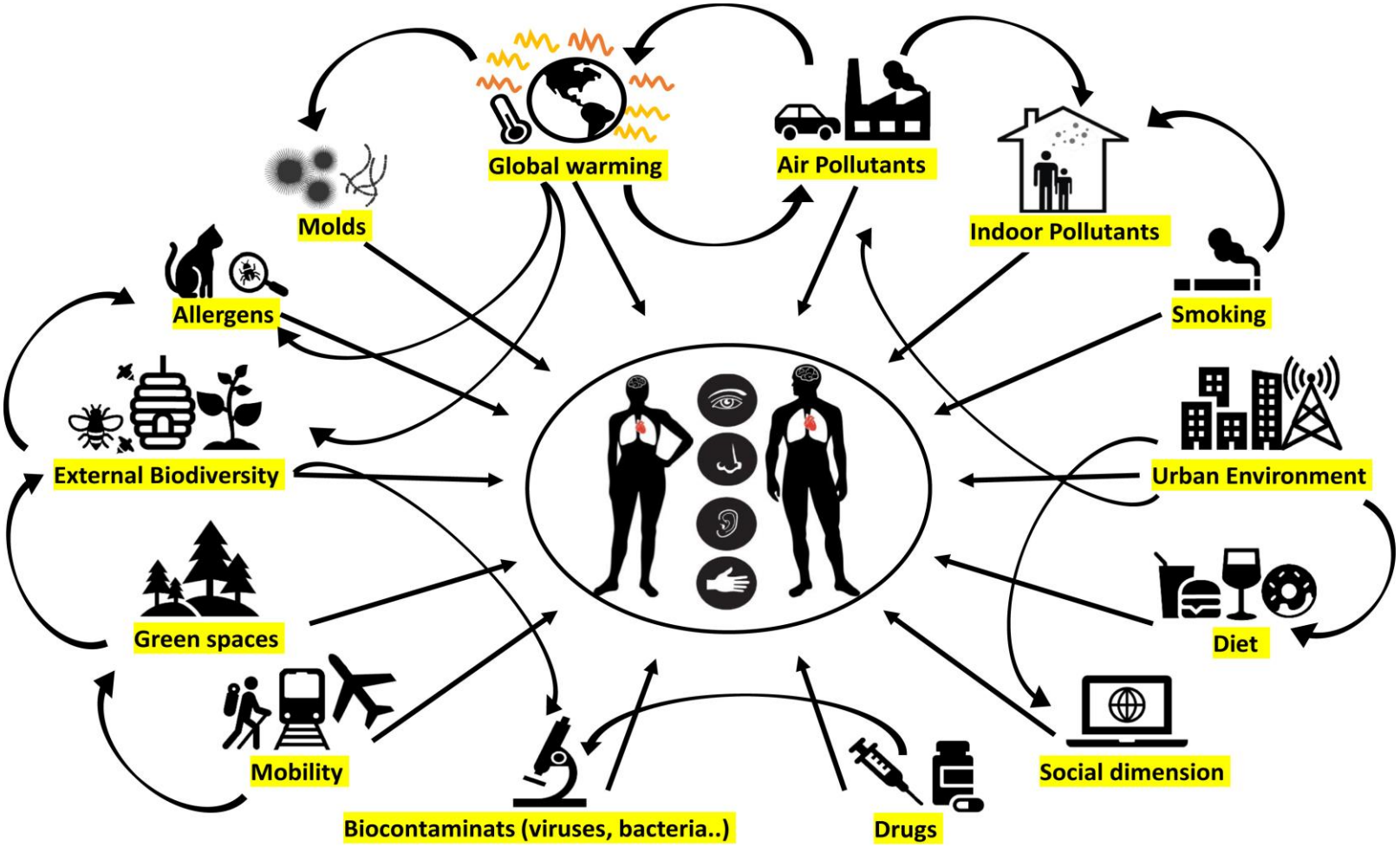
Paradigms and perspectives

Call to action: Air pollution, asthma, and allergy in the exposome era

Isabella Annesi-Maesano, MD, PhD, DSc,[a] Cara Nichole Maesano, PhD,[a] Benedetta Biagioni, MD,[b] Gennaro D'Amato, MD,[c] and Lorenzo Cecchi, MD, PhD[d] *Montpellier, France; and Bologna, Naples, and Florence, Italy*



Journal of the Endocrine Society, 2024, **8**, 1–9
https://doi.org/10.1210/jendso/bvad163
Advance access publication 11 January 2024
Research Article

Chemicals Used in Plastic Materials: An Estimate of the Attributable Disease Burden and Costs in the United States

Leonardo Trasande,[1,2,3] Roopa Krithivasan,[4] Kevin Park,[5] Vladislav Obsekov,[6] and Michael Belliveau[4]

Table 8. Estimates of plastic-attributable disease burden and costs

Exposure	Life stage of exposure	Outcome	Cost of illness across exposure routes (source [6] unless otherwise indicated)	PRF applied in base case (sensitivity analysis)	Plastic-related cases (sensitivity analysis)	Plastic-related cost of illness (sensitivity analysis)
PBDE-47	Prenatal	IQ points loss and intellectual disability	$162 billion	98% (97%-99%)	713 000 IQ points lost 23 900 cases of intellectual disability (23 700-24 100)	$158 billion ($157-160 billion)
	Prenatal	Cryptorchidism	$35.7 million		4200 cases (4200-4300)	$35.0 million ($34.6-35.3 million)
	Prenatal	Testicular cancer	$81 million		3500 cases (3500-3600)	$79.4 million ($78.6-80.2 million)
	All	All	$162 billion		N/A	**$159 billion ($157-161 billion)**
DEHP	Women	Obesity	$1.95 billion	98% (96%-99%)	5350 cases (5250-5410)	$1.92 billion ($1.88-1.94 billion)
		Type 2 diabetes	$259 million		2870 cases (2810-2900)	$253 million ($249-256 million)
	Women	Endometriosis	$39.7 billion		59 100 cases (57 900-59 700)	$38.9 billion ($38.1-39.3 billion)
	Adults	Cardiovascular Mortality	$23.8 billion [7]		50 200 cases (49 200-50 700)	$23.4 billion ($22.9-23.6 billion)
BBP and DBP	Men	Male infertility	$3.14 billion	100% (71%-100%)	121 000 cases (85 900-121 000)	$3.14 billion ($1.59-2.24 billion)
Total phthalates	All	All	$68.9 billion	N/A	N/A	**$66.7 billion ($64.7-67.3 billion)**
BPA	Prenatal	Childhood obesity	$1.04 billion	98% (96%-99%)	7130 (6850-7060)	**$1.02 billion ($1.00-1.03 billion)**
PFAS (PFOA in main estimates, PFOS in sensitivity analysis)	Prenatal	Low birth weight	$1.42 billion [8]	93% (16%-96%)	9350 cases (1610-93 000)	$1.32 billion ($227 million-13.2 billion)
	Prenatal	Childhood obesity	$2.65 billion [8]		118 000 cases (20 400-444 000)	$2.46 billion ($424 million-$9.22 billion)
	Children	Pneumonia	$1.49 million [8]		415 cases (72-6490)	$1.39 million ($238 000-$21.6 million)
	Pregnant people	Gestational diabetes	$414 million [8]		5640 cases (970-12 000)	$385 million ($66.2-$818 million)
	Adult	Obesity	$17.0 billon [8]		3 990 000 cases (687 000-4 120 000)	$15.8 billion ($2.72-$16.3 billion)
	Adult	Kidney cancer	$180 million [8]		132 cases (23-136)	$171 million ($29.4-$177 million)
	Adults	Couple infertility	$37.6 million [8]		551 cases (95-25 100)	$35.0 million ($6.06 million-$1.59 billion)
	Women	Hypothyroidism	$1.26 billion [8]		13 600 cases (2300-57 400)	$1.17 billion ($201 million-4.98 billion)
	Women	Type 2 diabetes	$140 million [8]		1600 cases (276-1660)	$130 million ($22.4-$134 million)
	Women	Endometriosis	$397 million [8]		647 cases (111-17 300)	$369 million ($63.5 million-$9.79 billion)
	Women	Polycystic ovary syndrome	$10.5 million [8]		6700 cases (1150-7200)	$9.77 million ($1.68-$10.5 million)
	Women	Breast cancer	$555 million [8]		392 cases (101-2971)	$516 million ($133 million-$3.92 billion)
	Men	Testicular cancer	$6.85 million [8]		5 cases (1-5)	$6.37 million ($1.10-$6.58 million)
	All	All	$24.1 billion [8]		N/A	**$22.4 billion ($3.85-60.1 billion)**
All	All	All		N/A	N/A	**$249 billion ($226-$289 billion)**

Abbreviation: BBP, butyl benzyl phthalate; BPA, bisphenol A; DBP, dibutyl phthalate; DEHP, bis(2-ethylhexyl)phthalate; N/A, not available; PBDE, polybrominated diphenyl ether; PFAS, perfluoroalkyl and polyfluoroalkyl substances; PFOA, perfluorooctanoic acid; PFOS, perfluorooctane sulfonate; PRF, plastic-related fraction.

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The NEW ENGLAND JOURNAL of MEDICINE

Table 1. Characteristics of the Medicare Cohort, 2000 through 2016.*

Characteristic	Full Cohort†	Black Persons		White Persons	
		Higher Income‡	Low Income§	Higher Income‡	Low Income§
Persons — no. (% of full cohort)	73,129,782 (100)	4,872,714 (6.7)	1,671,776 (2.3)	56,422,414 (77.2)	4,989,457 (6.8)
Person-yr — no. (% of total person-yr)	623,042,512 (100)	37,862,780 (6.1)	14,886,928 (2.4)	483,479,863 (77.6)	48,247,908 (7.7)
Deaths — no. (% of total deaths)	29,467,648 (100)	1,488,555 (5.1)	1,154,227 (3.9)	20,773,208 (70.5)	4,769,240 (16.2)
Median follow-up time — yr	8.0	7.0	8.0	8.0	8.0
Age at entry — %					
65–74 yr	80.6	86.2	77.4	80.4	72.7
75–84 yr	14.8	10.7	15.6	15.3	17.2
85–94 yr	4.2	2.5	6.2	4.0	9.0
≥95 yr	0.4	0.6	0.8	0.3	1.1
Female sex — %	55.4	54.9	68.1	54.3	68.0
Medicaid eligible — %	11.6	0	100	0	100
Ecologic variables¶					
Average $PM_{2.5}$ level — $\mu g/m^3$	9.8±3.2	10.2±3.1	10.5±3.0	9.8±3.2	9.8±3.2
Mean BMI	27.6±1.1	27.5±1.0	27.5±1.1	27.6±1.1	27.6±1.1
Ever smoked — %	47.3	46.7	46.4	47.3	47.3
Income below federal poverty line — %	10.5	10.6	11.3	10.4	10.4
Median household income — $ in thousands	49.0	51.1	49.7	49.0	48.9
Median house value — $ in thousands	162.7	180.5	176.1	162.6	161.4
Owner-occupied housing — %	72.0	68.9	67.1	72.1	72.5
Below high school education — %	28.5	28.0	29.3	28.4	28.6
Population density — persons/$mile^2$	1536.2	2124.7	2450.2	1524.3	1485.1
Meteorologic variables‖					
Mean summer temperature — °C	29.5±3.6	30.1±3.6	30.6±3.5	29.5±3.7	29.5±3.7
Mean summer relative humidity — %	88.0±11.7	88.4±11.7	88.7±11.6	88.0±11.7	88.1±11.6
Mean winter temperature — °C	7.6±7.2	9.5±7.1	10.4±6.9	7.5±7.2	7.6±7.2
Mean winter relative humidity — %	86.2±7.3	85.1±7.5	85.8±7.5	86.2±7.3	86.2±7.3
Region — %‖					
Northeast	19.6	18.7	15.9	19.6	19.5
South	36.6	45.5	51.9	36.6	36.9
Midwest	26.5	19.1	16.4	26.6	26.5
West	17.3	16.7	15.7	17.3	17.1

SPECIAL ARTICLE

Air Pollution and Mortality at the Intersection of Race and Social Class

Kevin P. Josey, Ph.D., Scott W. Delaney, Sc.D., J.D., Xiao Wu, Ph.D., Rachel C. Nethery, Ph.D., Priyanka DeSouza, Ph.D., Danielle Braun, Ph.D., and Francesca Dominici, Ph.D.

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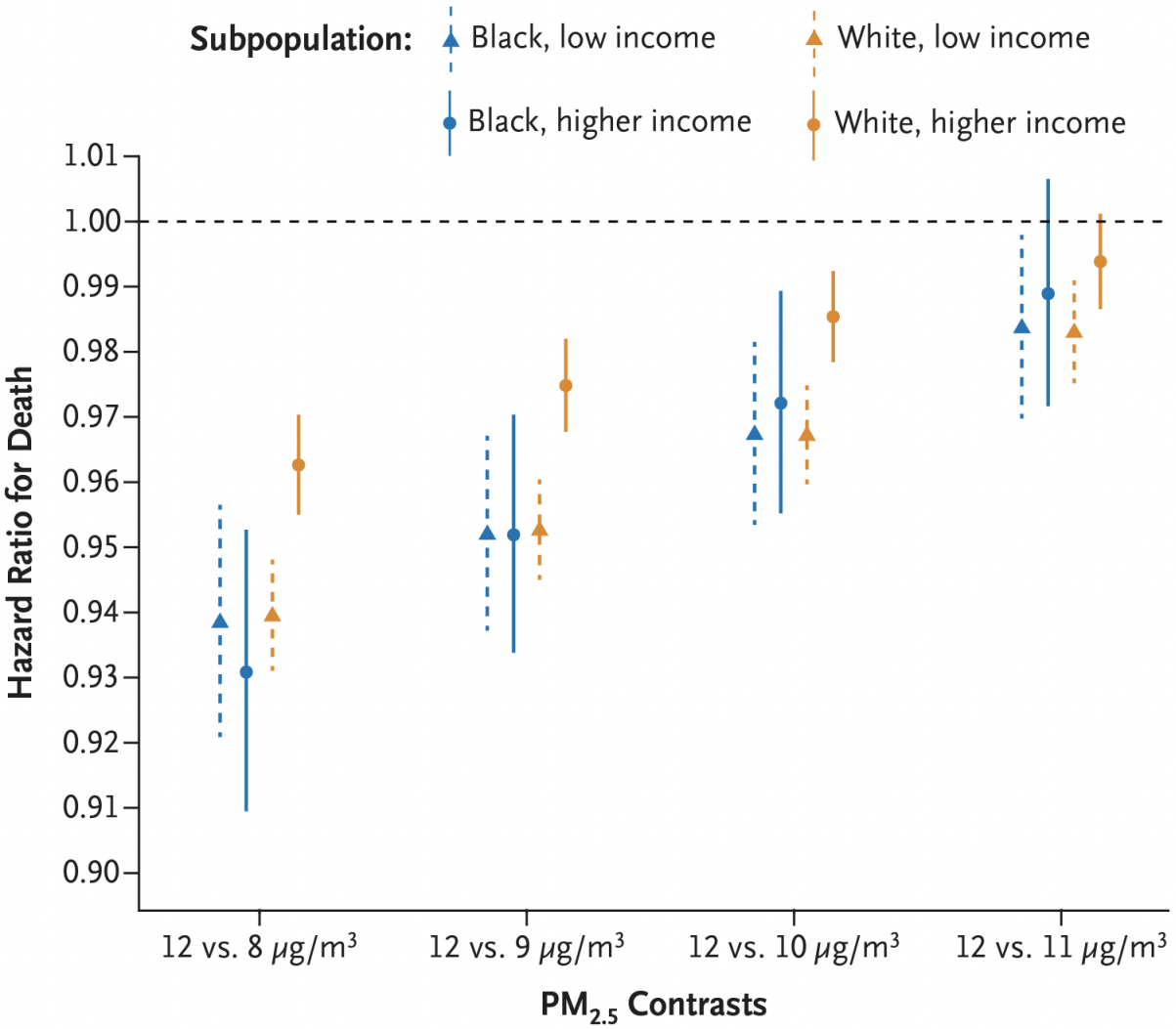

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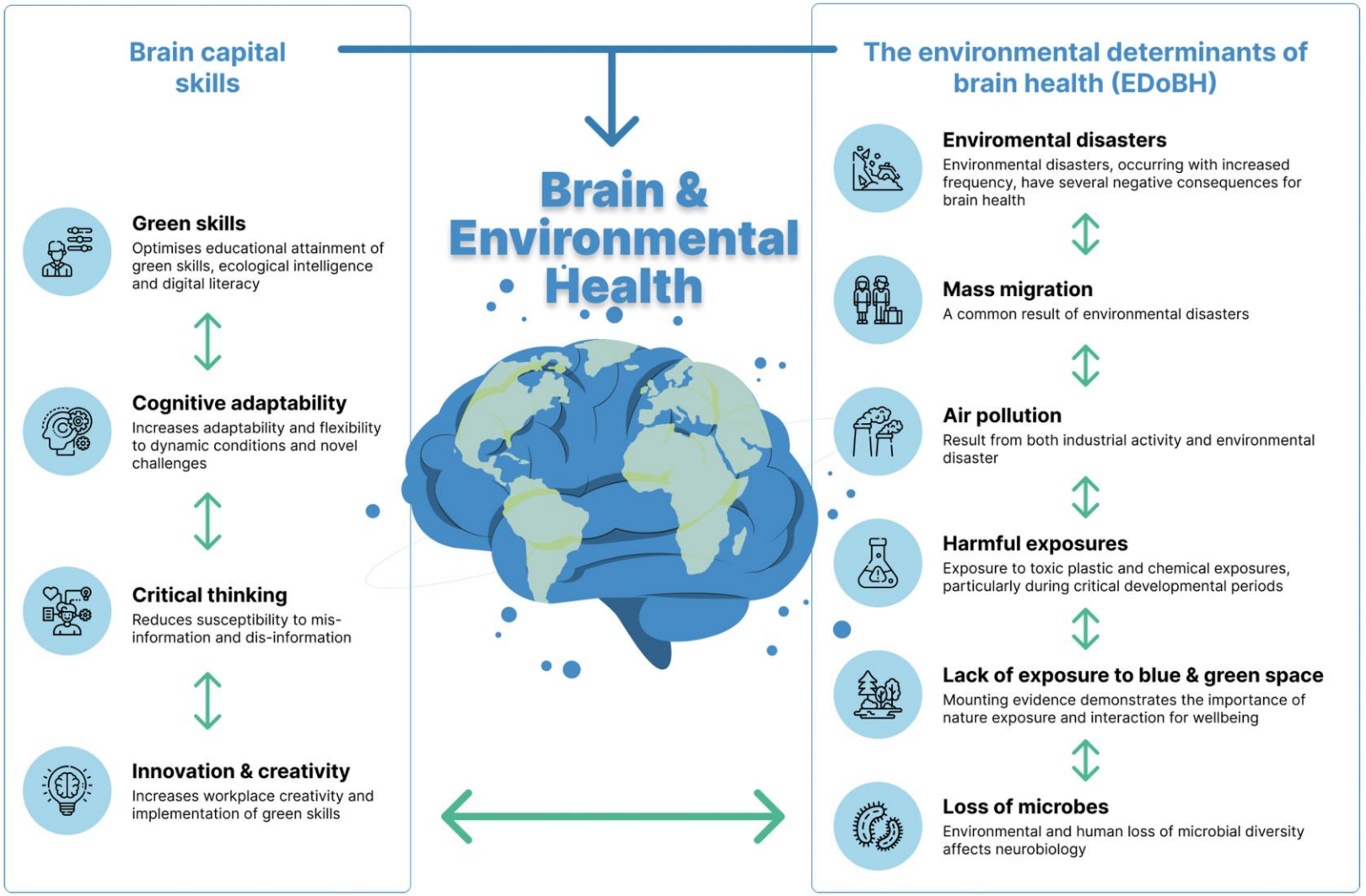
Open access Perspective

BMJ Mental Health

Brain capital, ecological development and sustainable environments

Agustin Ibanez [1,2,3] Harris Eyre [4,5,6]



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Goals

We aim to advance the environmental neurosciences through a collaborative network of neuroimaging cohorts that address key questions about how our physical environments impact the human brain. Initial research aims are to

1) Identify the impact of air pollution on neurodevelopment, brain aging and neurodegeneration
2) Determine how sex, race, and socioeconomic status modify the impact of air pollution on the brain
3) Define key interactions between environmental variables and brain structure that influence mental health
4) Determine how human behaviors (e.g., smoking, exercise, nutrition) modulate the environmental effects on the brain
5) Work with ENIGMA-Genetics group to identify gene x environment interactions that impact brain structure and function

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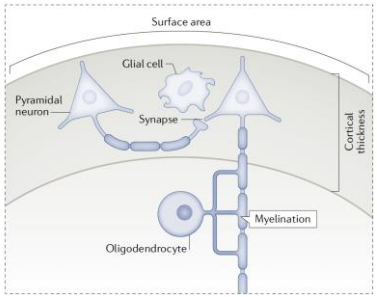
PERSPECTIVES

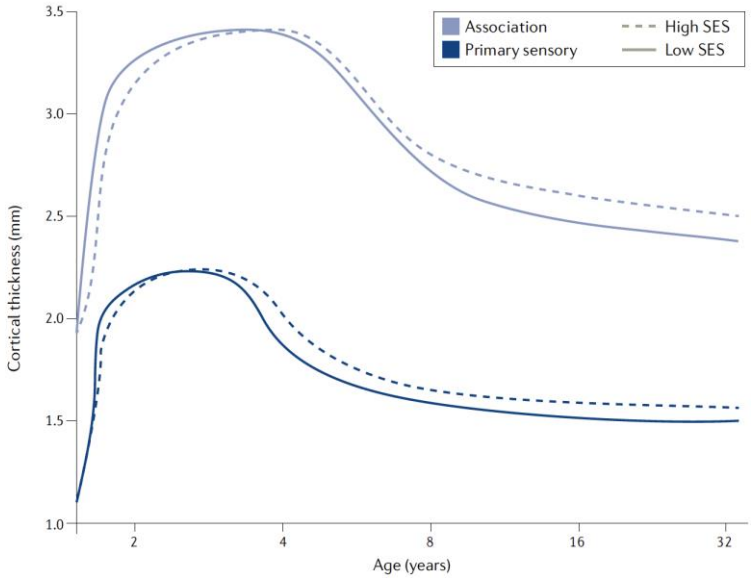
Check for updates www.nature.com/nrn

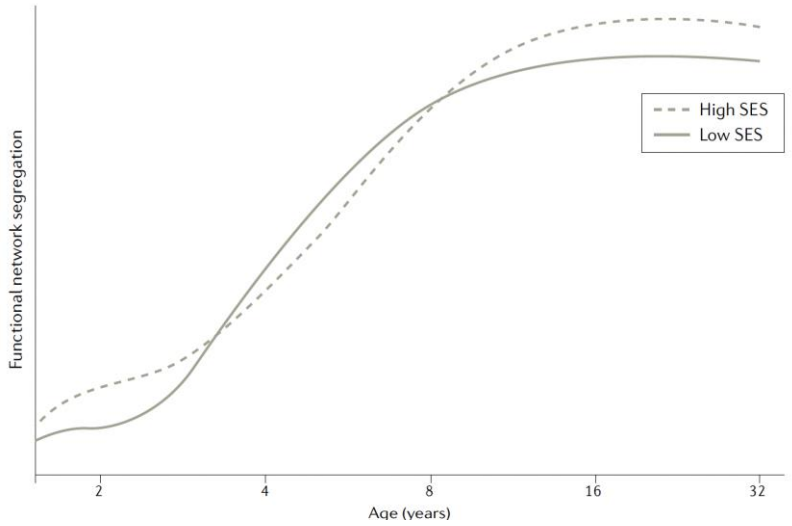
372 | JUNE 2021 | VOLUME 22

Environmental influences on the pace of brain development

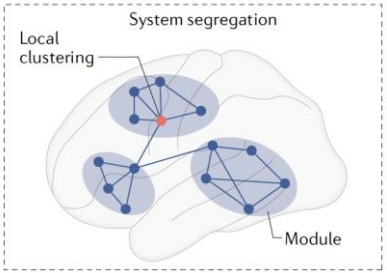
Ursula A. Tooley, Danielle S. Bassett and Allyson P. Mackey







ultimately leading to more efficient cortical networks in adulthood. We hypothesize that greater exposure to chronic stress accelerates brain maturation, whereas greater access to novel positive experiences decelerates maturation. We discuss the impact of variation in the pace of brain development on plasticity and learning. We provide a generative theoretical framework to catalyse future basic



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nature medicine

Article

https://doi.org/10.1038/s41591-023-02365-w

Effects of urban living environments on mental health in adults

Received: 28 August 2022

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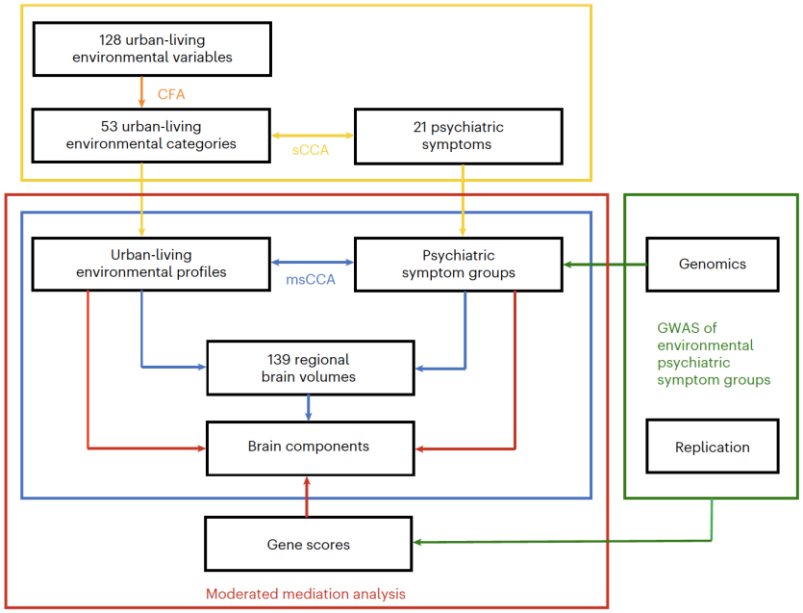
Published online: 15 June 2023

Jiayuan Xu[1,45], Nana Liu[1,45], Elli Polemiti[2,3], Liliana Garcia-Mondragon[4], Jie Tang[1], Xiaoxuan Liu[5], Tristram Lett[2,3], Le Yu[6,7], Markus M. Nöthen[8], Jianfeng Feng[9], Chunshui Yu[1,10], Andre Marquand[11,45], Gunter Schumann[2,3,45] & the environMENTAL Consortium*



Urban-living individuals are exposed to many environmental factors that may combine and interact to influence mental health. While individual factors of an urban environment have been investigated in isolation, no attempt has been made to model how complex, real-life exposure to living in the city relates to brain and mental health, and how this is moderated by genetic factors. Using the data of 156,075 participants from the UK Biobank, we carried out sparse canonical correlation analyses to investigate the relationships between urban environments and psychiatric symptoms. We found an environmental profile of social deprivation, air pollution, street network and urban land-use density that was positively correlated with an affective symptom group ($r = 0.22$, $P_{perm} < 0.001$), mediated by brain volume differences consistent with reward processing, and moderated by genes enriched for stress response, including *CRHR1*, explaining 2.01% of the variance in brain volume differences. Protective factors such as greenness and generous destination accessibility were negatively correlated with an anxiety symptom group ($r = 0.10$, $P_{perm} < 0.001$), mediated by brain regions necessary for emotion regulation and moderated by *EXD3*, explaining 1.65% of the variance. The third urban environmental profile was correlated with an emotional instability symptom group ($r = 0.03$, $P_{perm} < 0.001$). Our findings suggest that different environmental profiles of urban living may influence specific psychiatric symptom groups through distinct neurobiological pathways.

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RESEARCH ARTICLE OPEN ACCESS

Fine Particulate Matter and Parkinson Disease Risk Among Medicare Beneficiaries

Brittany Krzyzanowski, PhD, Susan Searles Nielsen, PhD, Jay R. Turner, DSc, and Brad A. Racette, MD

Neurology® 2023;101:e2058-e2067. doi:10.1212/WNL.0000000000207871

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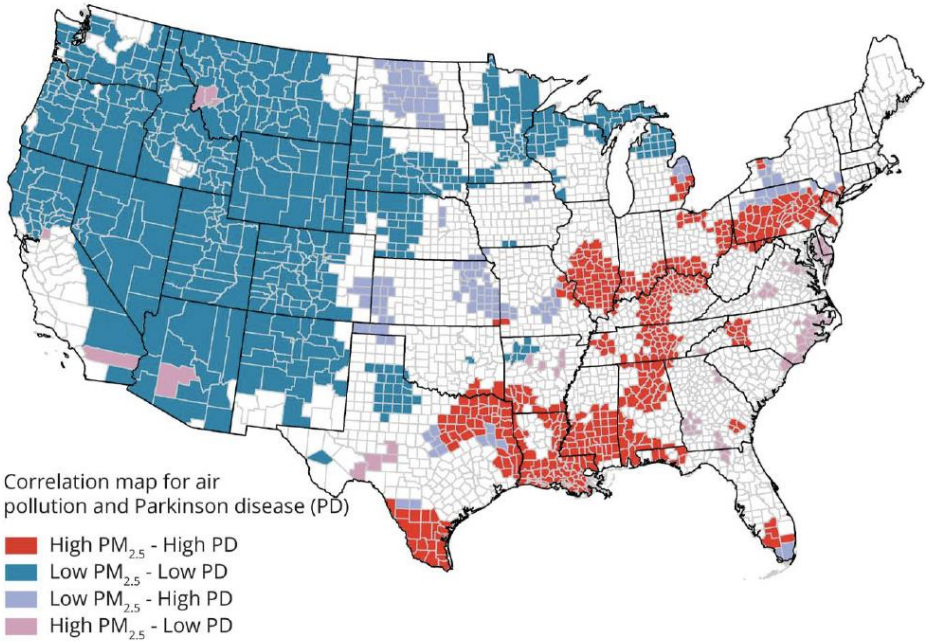

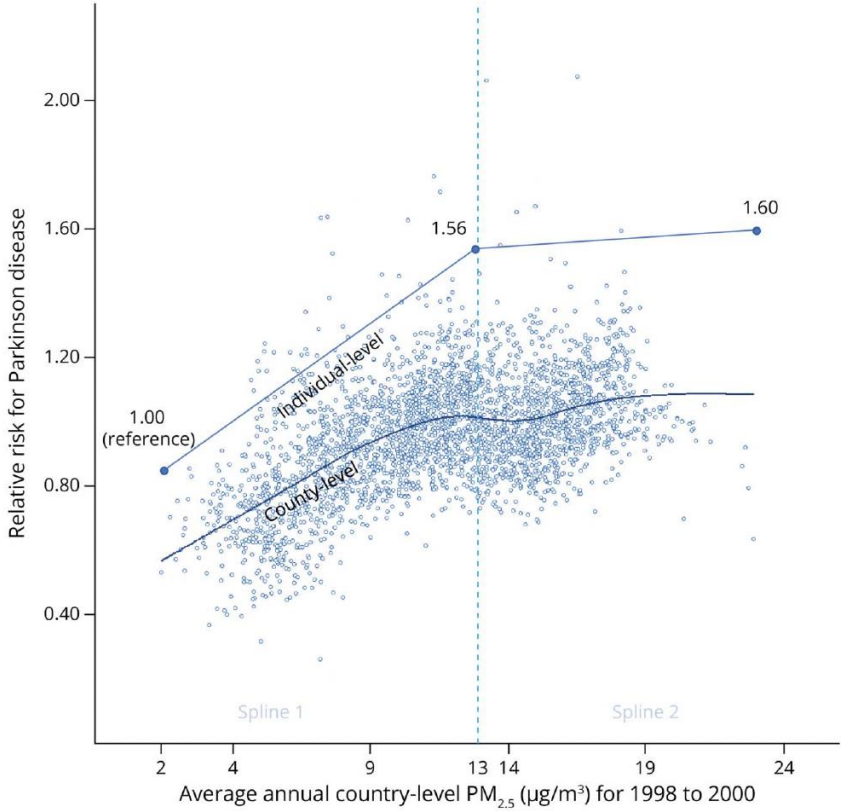

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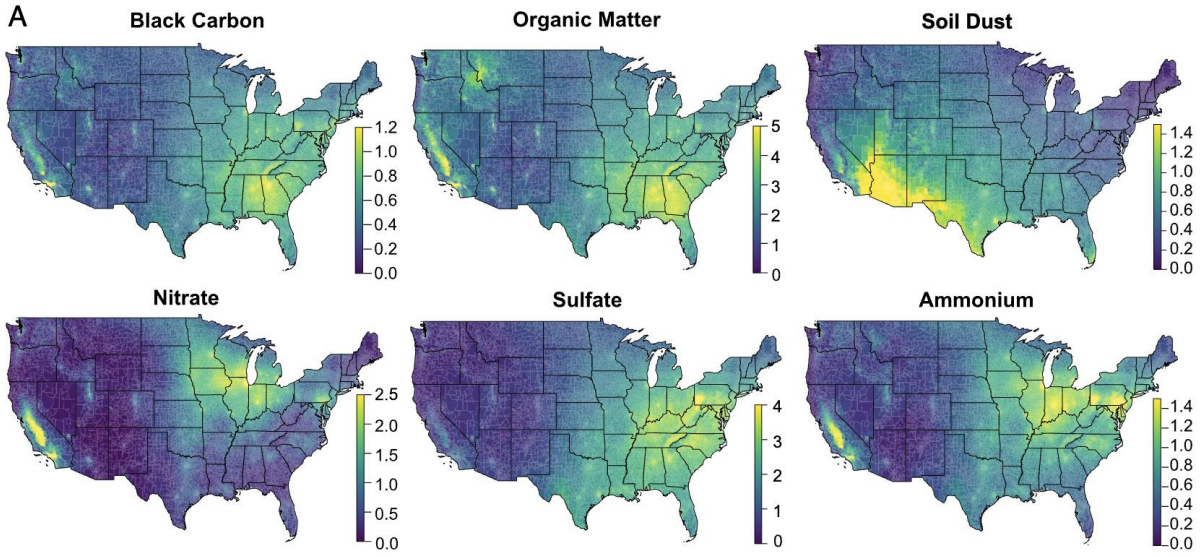
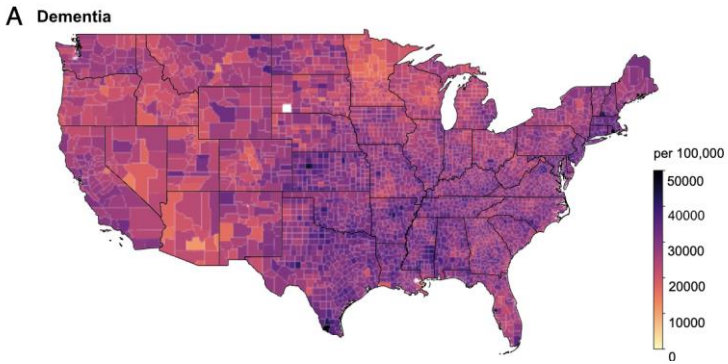
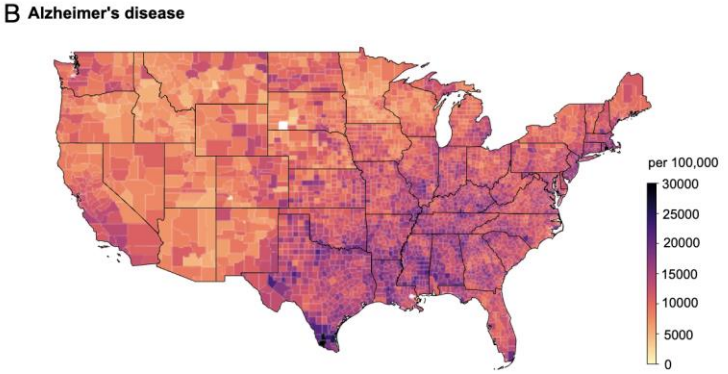
PNAS RESEARCH ARTICLE | ENVIRONMENTAL SCIENCES OPEN ACCESS

Incident dementia and long-term exposure to constituents of fine particle air pollution: A national cohort study in the United States

Liuhua Shi[a,1], Qiao Zhu[a], Yifan Wang[a], Hua Hao[a], Haisu Zhang[a], Joel Schwartz[b,c], Heresh Amini[d], Aaron van Donkelaar[e], Randall V. Martin[e], Kyle Steenland[a], Jeremy A. Sarnat[a], W. Michael Caudle[a], Tszshan Ma[a], Haomin Li[a], Howard H. Chang[f], Jeremiah Z. Liu[g], Thomas Wingo[h], Xiaobo Mao[i], Armistead G. Russell[j], Rodney J. Weber[a], and Pengfei Liu[k,1]







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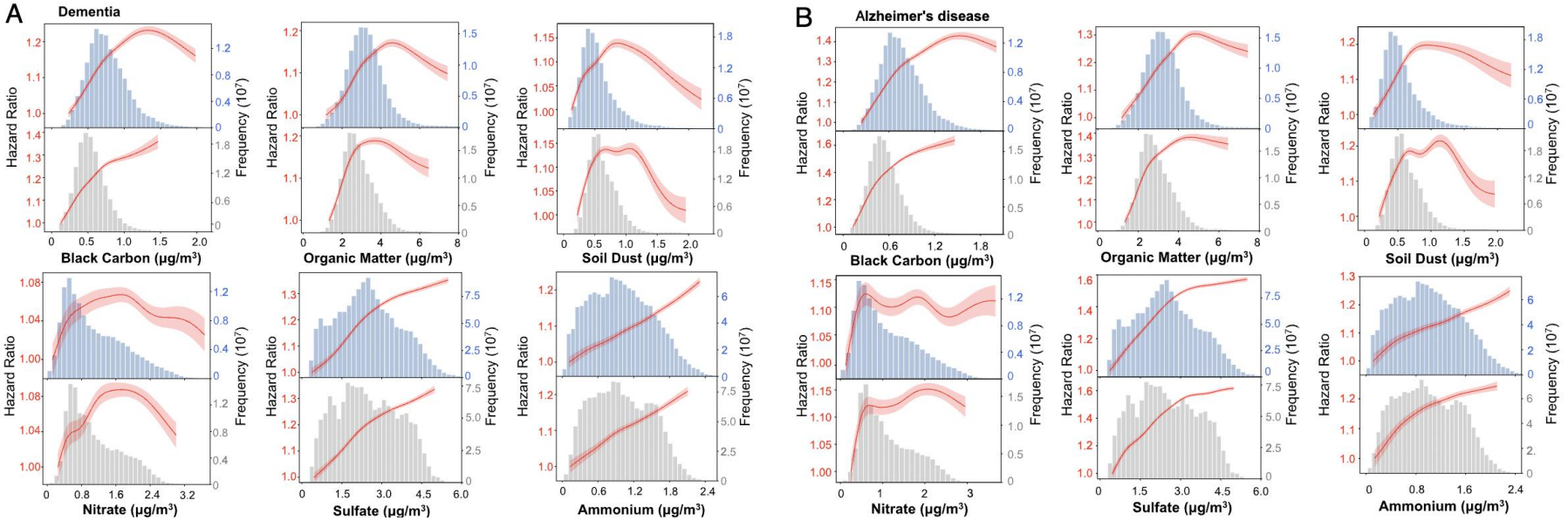

Fig. 4. The concentration–response curves for each $PM_{2.5}$ constituent and dementia (*A*) and Alzheimer's disease (*B*). The concentration–response curves, derived from the single-constituent models, are shown for the concentration ranges between 0.5th and 99.5th percentiles of the pollutants, i.e., with 1% poorly constrained extreme values excluded. For each constituent, the top panel used exposure I data (11) and the bottom panel used exposure II data (12).

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Effects of Environmental Toxins on Brain Health and Development

Maternal smoking during pregnancy negatively affects brain volumes proportional to intracranial volume in adolescents born very preterm

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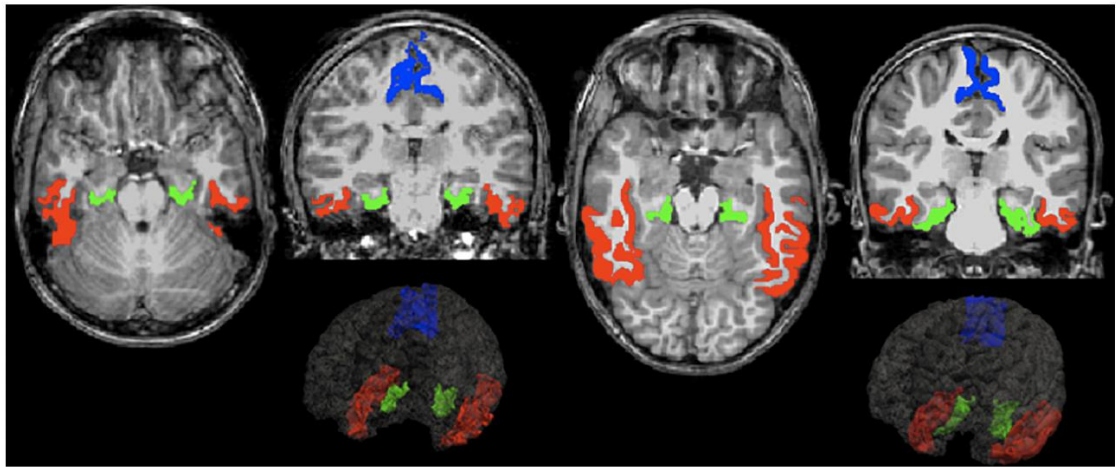

FIGURE 2
Example figures of inferior temporal (red), parahippocampal (green), and postcentral (blue) regions where differences were found between exposed **(left)** and unexposed **(right)** group of adolescents born very preterm in transaxial and coronal slices of T1-weighted magnetic resonance imaging (MRI) images.

CONCLUSIONI

- Il cervello umano è fortemente influenzato da fattori esterni
- L'alimentazione ha un impatto sullo sviluppo cerebrale a livello ontogenetico e filogenetico
- La dieta modula i processi di neurogenesi e plasticità soprattutto attraverso meccanismi legati a infiammazione e stress ossidativo
- Alimentazione ed ambiente sono fortemente legati
- La salute cerebrale/mentale è influenzata dall'ambiente
- L'esposizione a fattori ambientali negativi può aumentare il rischio di sviluppare malattie neurologiche

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