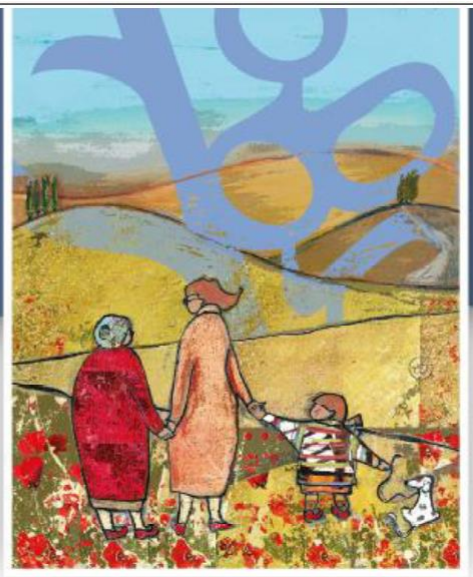
64° CONGRESSO NAZIONALE SIGG

Continuità di affetti, continuità di cure

ROMA, 27/30 NOVEMBRE 2019 - AUDITORIUM DELLA TECNICA



NUOVI RISULTATI DELLA RETE GERIATRICA ALZHEIMER (ReGAl 2.0)

Virginia Boccardi MD, PhD

AZIENDA OSPEDALIERA DI PERUGIA
S.C. DI GERIATRIA
ISTITUTO DI GERONTOLOGIA E GERIATRIA
UNIVERSITÀ DEGLI STUDI DI PERUGIA

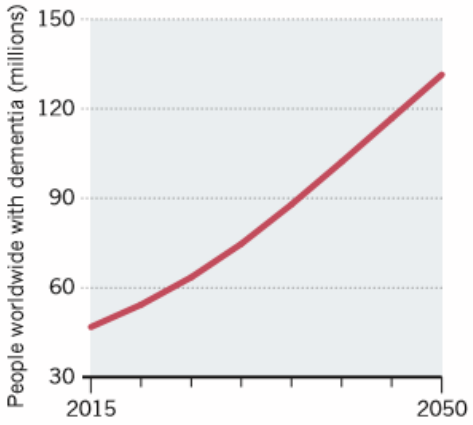
Aging remains the most powerful risk factor for developing DEMENTIA

An age-old story of dementia

The biology and epidemiology of Alzheimer's disease.

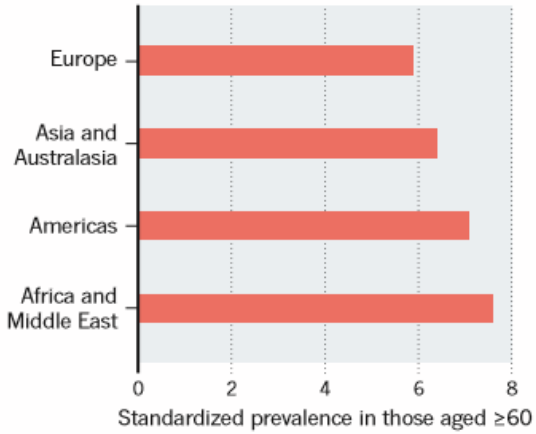
AGEING AND GROWING

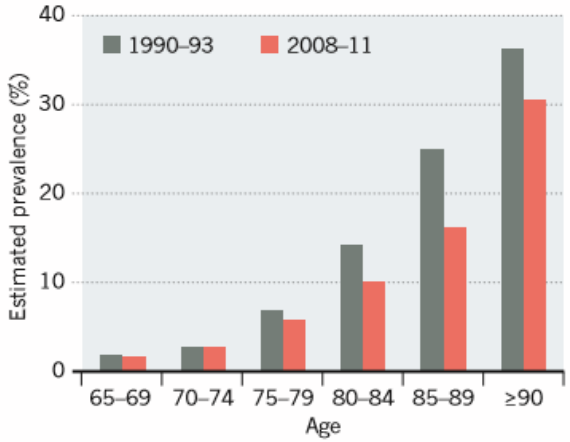
In 2015, almost 50 million people worldwide had dementia[2]. As the population grows and ages, the number affected is expected to surpass 130 million people by 2050.



LOCAL LESSONS

The prevalence of Alzheimer's disease (left) in people aged 60 or above is highest in north Africa and the Middle East, and the condition is least common in central Europe[2]. In affluent countries in the West, such as the United Kingdom, a decline in the prevalence of Alzheimer's disease in the elderly has been observed (right)[3]. This suggests that concurrent changes in lifestyle might have provided some protection from dementia (see page S18).





Liam Drew, 2018

nature
International journal of science

Ageing Research Reviews 49 (2019) 11–26

Contents lists available at ScienceDirect

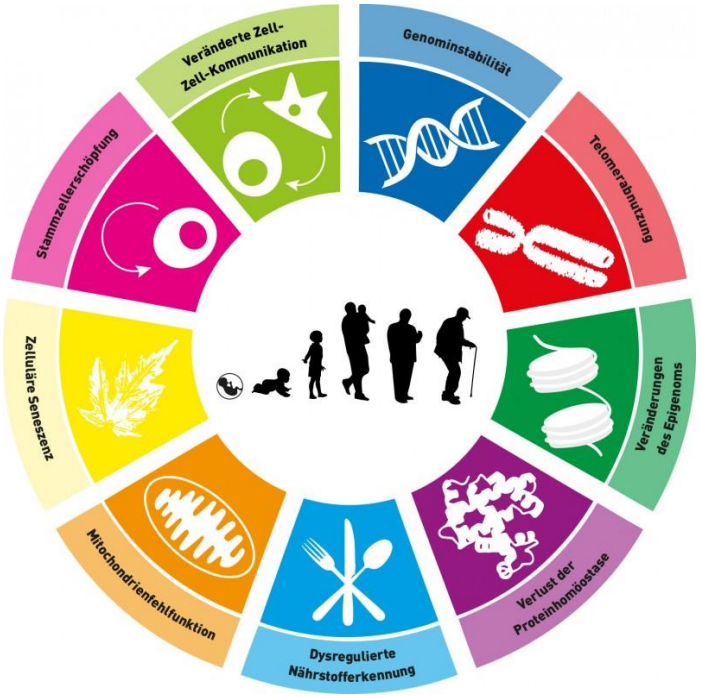
Ageing Research Reviews

journal homepage: www.elsevier.com/locate/arr

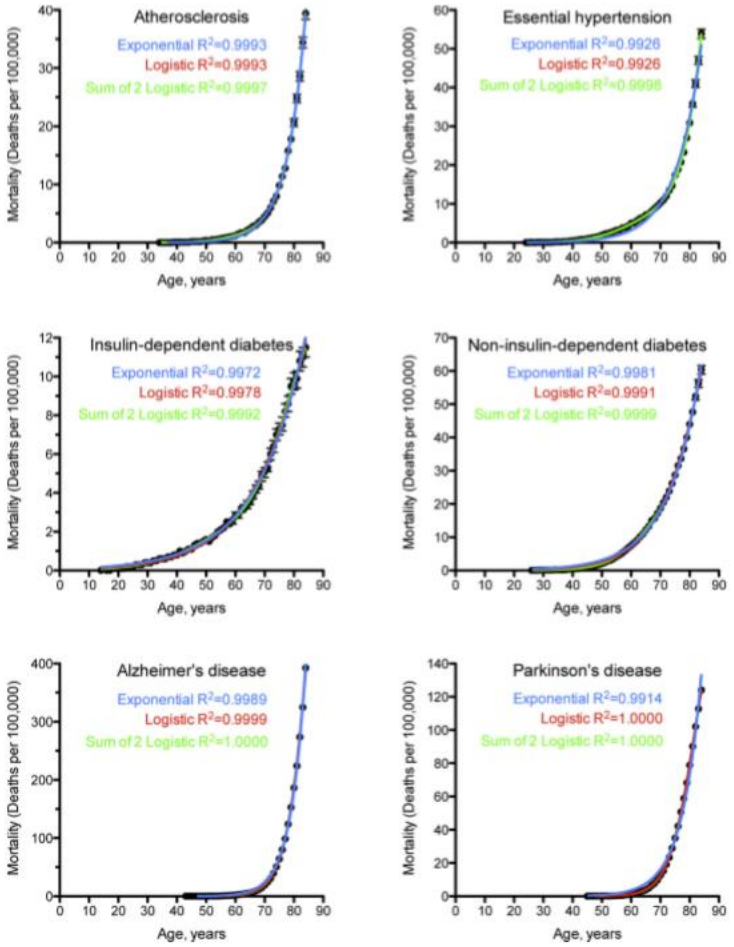
Age-related diseases as vicious cycles

Aleksey V. Belikov

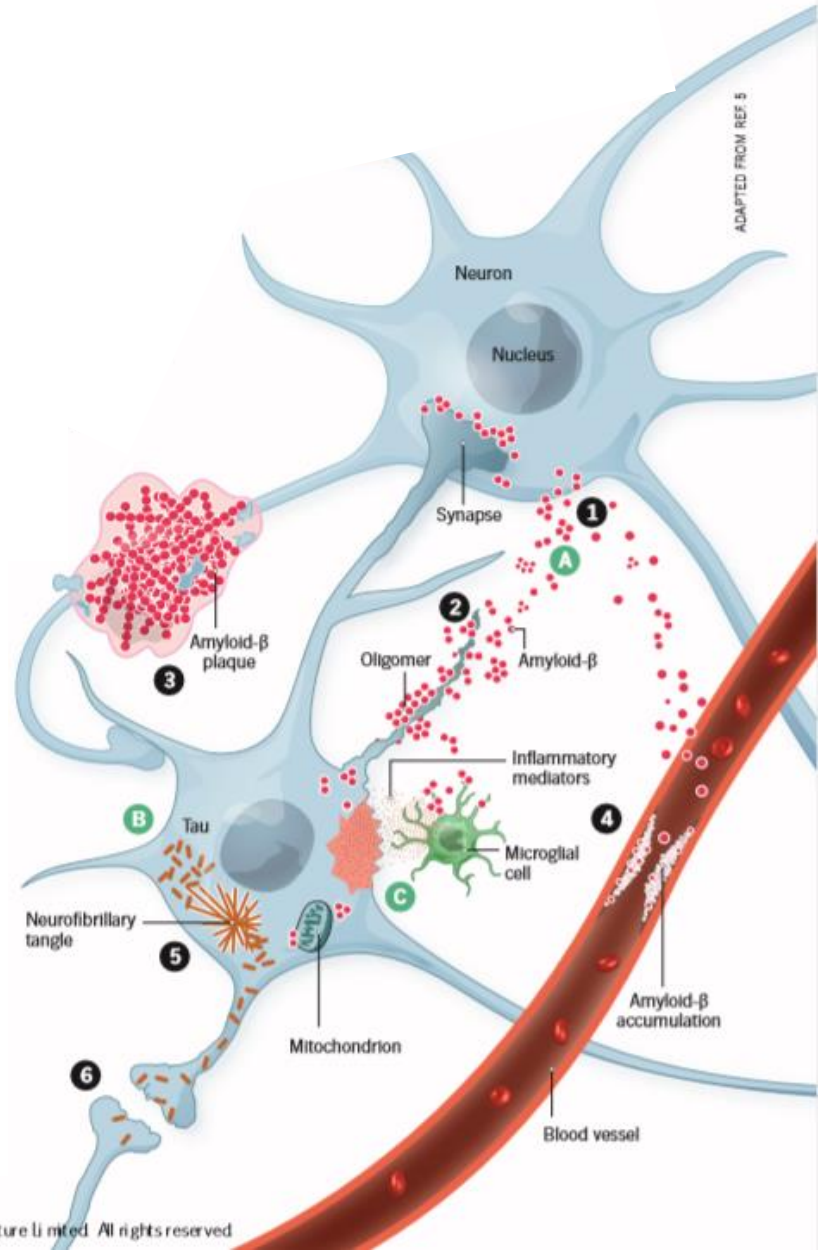
Laboratory of Innovative Medicine, School of Biological and Medical Physics, Moscow Institute of Physics and Technology, Institutsky per., 9, 141701 Dolgoprudny, Moscow Region, Russia



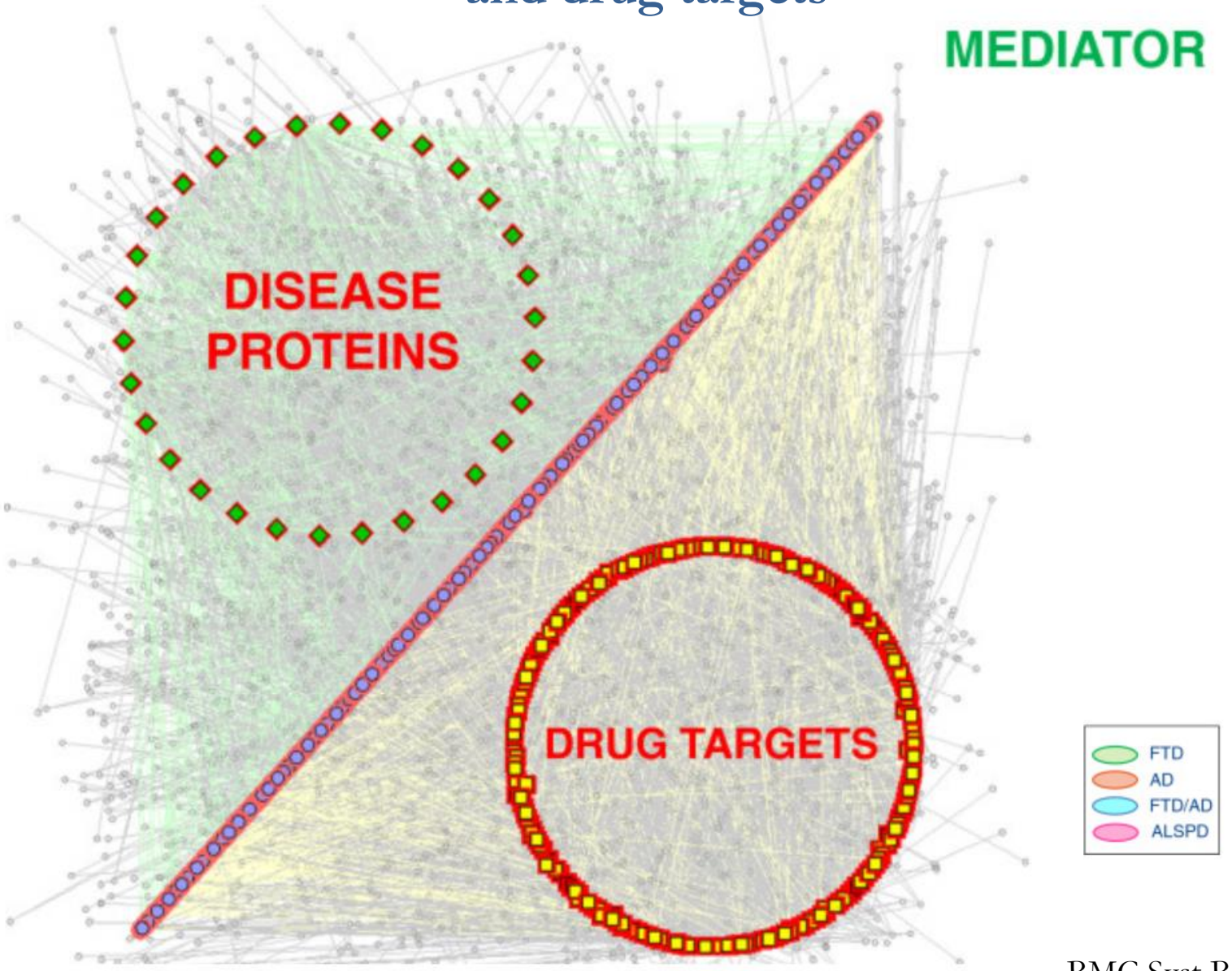
Cell 2013



…UNDERSTANDING

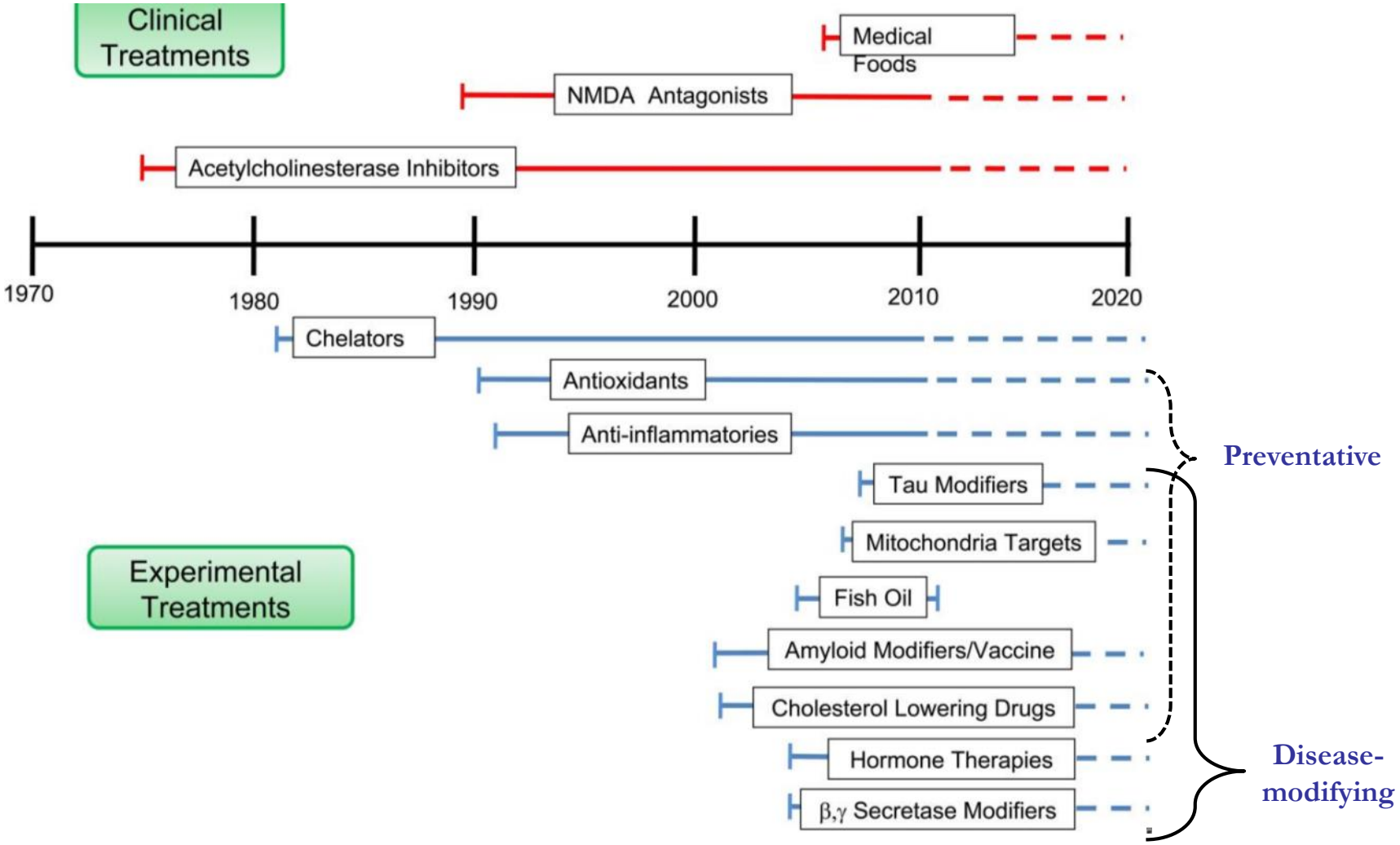


Protein-protein interactions network created using disease and drug targets



BMC Syst Biol 2014

There is currently **no "cure" for dementia**



Ther Adv Chronic Dis 2011

“Knowledge accrues in pieces, but is understood in patterns”

V. Hachinski 2007

In old age biomarkers are not a disease

Age-specific population frequencies of cerebral β-amyloidosis and neurodegeneration among people with normal cognitive function aged 50–89 years: a cross-sectional study

Clifford R Jack Jr, Heather J Wiste, Stephen D Weigand, Walter A Rocca, David S Knopman, Michelle M Mielke, Val J Lowe, Matthew L Senjem, Jeffrey L Gunter, Gregory M Preboske, Vernon S Pankratz, Prashanthi Vemuri, Ronald C Petersen

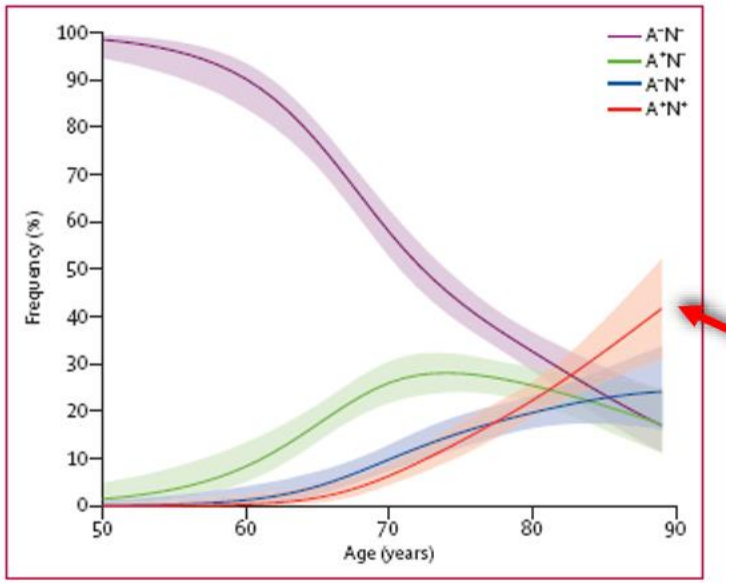


Figure 1: Estimated frequency (percentage) of participants in each biomarker group, by age
Estimates are from a multinomial model adjusted for sex. Non-linearity in age was allowed in the model by fitting age as a spline with knots at ages 60, 70, and 80 years. Shaded areas are 95% parametric bootstrap CIs.

Lancet Neurol, 2017

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Age, Neuropathology, and Dementia

George M. Savva, Ph.D., Stephen B. Wharton, F.R.C.Path., Paul G. Ince, M.D., Gillian Forster, B.Sc., Fiona E. Matthews, Ph.D., and Carol Brayne, M.D., for the Medical Research Council Cognitive Function and Ageing Study

Table 2. Odds Ratios for the Association between Neuropathological Features and Dementia at Death, Modeled at the Ages of 75 and 95 Years.*

Variable	75 Yr of Age	95 Yr of Age	P Value†
	odds ratio (95% CI)		
Tangles			
Hippocampus	8.61 (3.66–20.27)	2.11 (1.05–4.25)	0.03
Neocortex	35.16 (8.16–153.31)	7.04 (2.40–22.87)	0.14
Entorhinal cortex	4.72 (1.97–11.30)	2.94 (1.37–6.29)	0.48
Neuritic plaques			
Hippocampus	10.19 (4.28–24.25)	1.42 (0.71–2.82)	0.002
Neocortex	8.63 (3.81–19.60)	2.48 (0.92–4.14)	0.04
Entorhinal cortex	7.18 (2.99–17.25)	2.28 (1.11–4.67)	0.08
Diffuse plaques			
Hippocampus	2.36 (1.10–5.10)	2.12 (1.08–4.16)	0.86
Neocortex	2.67 (1.24–5.74)	1.83 (0.95–3.48)	0.42
Entorhinal cortex	2.91 (1.14–7.45)	1.19 (0.56–2.53)	0.20
Cortical atrophy			
Hippocampus	7.96 (2.67–23.68)	4.22 (1.80–9.91)	0.43
Neocortex	5.11 (1.94–13.46)	6.10 (2.80–13.28)	0.81
Vascular pathology			
More than one vascular pathological change	2.36 (1.09–5.11)	1.56 (0.80–3.04)	0.48
Infarcts	2.87 (1.29–6.40)	1.09 (0.53–2.26)	0.12
Hemorrhage	0.87 (0.15–4.93)	0.73 (0.15–3.57)	0.90
Lacunes	1.41 (0.58–3.46)	1.99 (0.88–4.51)	0.62
Small-vessel disease	2.69 (1.15–6.31)	1.79 (0.89–3.61)	0.52

* All pathological measures were recorded in accordance with the protocol of the Consortium to Establish a Registry for Alzheimer's Disease. The model is based on the presence of moderate or severe neurofibrillary tangles, neuritic plaques, diffuse plaques, and cortical atrophy. Vascular lesions are classified as present or absent rather than graded according to severity. The odds ratios were generated from a series of logistic-regression models; the dependent (outcome) variable for each model was a neuropathological variable, and the independent variables were dementia, age, and the interaction between age and dementia.

† P values represent the significance of the effect of age at death on the association between neuropathological features and dementia in the model.

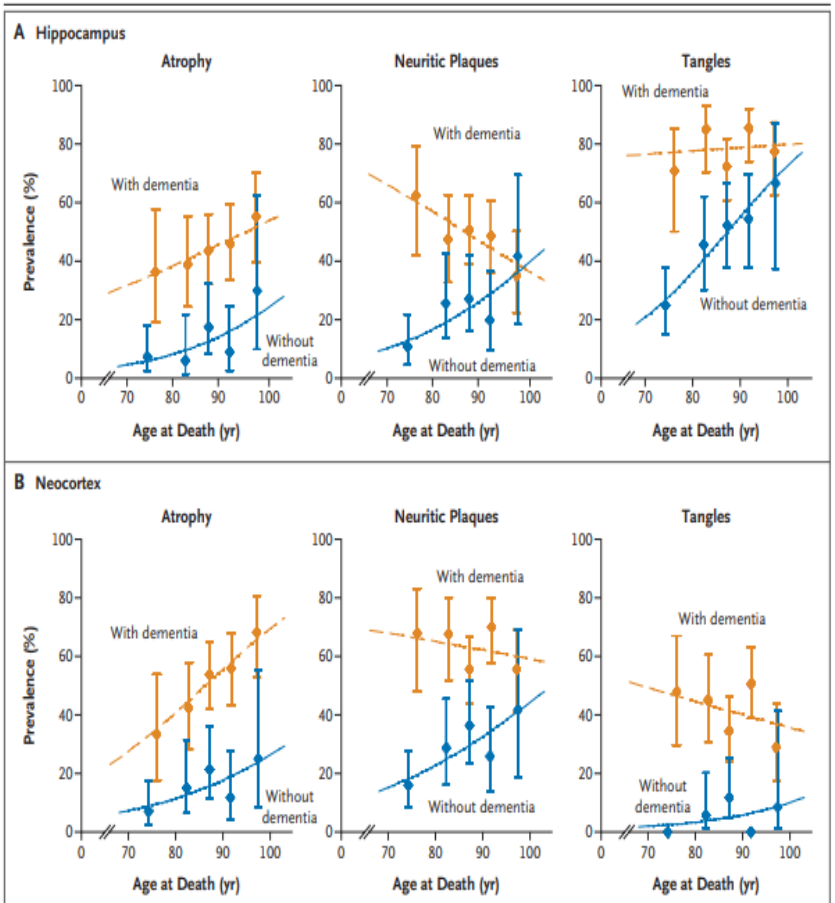


Figure 1. Modeled and Observed Prevalence of Moderate or Severe Pathological Lesions According to Age.

Persons who died with dementia (yellow) are compared with those who died without dementia (blue). Filled symbols represent the observed prevalence of moderate or severe pathological lesions, and I bars show the 95% confidence intervals. The solid and broken lines represent modeled prevalence values.

2009

Published in final edited form as:
Curr Alzheimer Res. 2012 July 1; 9(6): 709–717.

A Population-Based Clinicopathological Study in the Oldest-Old: The 90+ Study

María M. Corrada[1,2], **Daniel J. Berlau**[1,2], and **Claudia H. Kawas**[1,2,3]

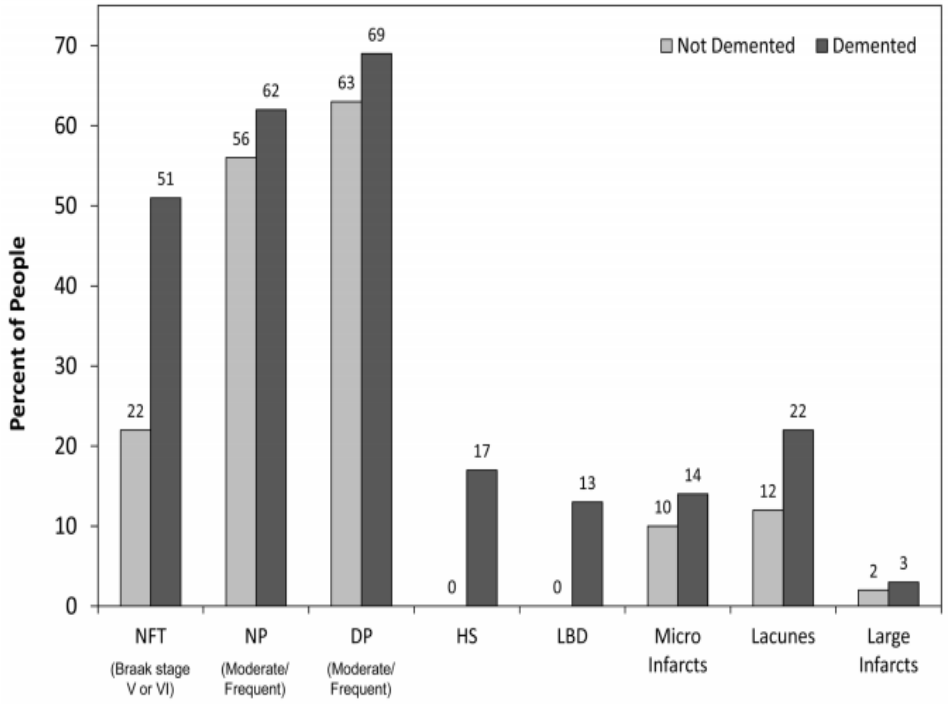


Figure 1.
Types of Pathology in Demented and Non-Demented Participants in ***The 90+ Autopsy Study*** 2003 Cohort (N=104)
NFT=neurofibrillary tangles; NP=neuritic plaques; DP=diffuse plaques; HS=hippocampal sclerosis; LBD=Lewy body disease; infarcts do not include terminal events

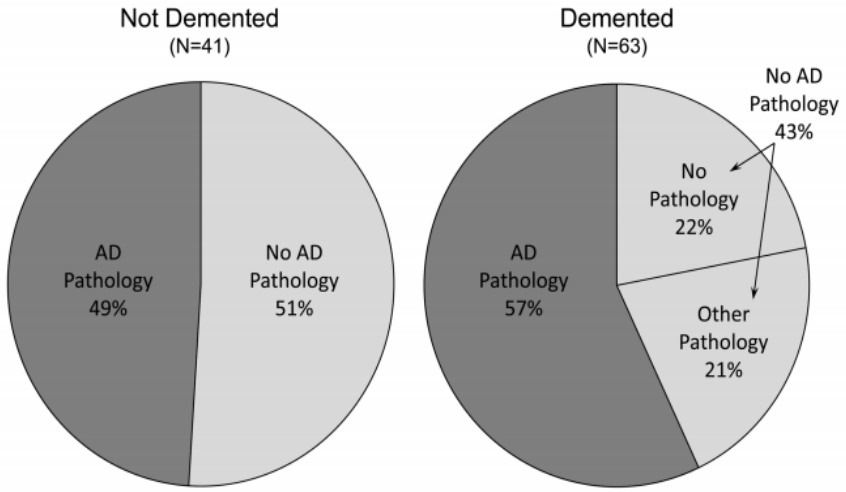
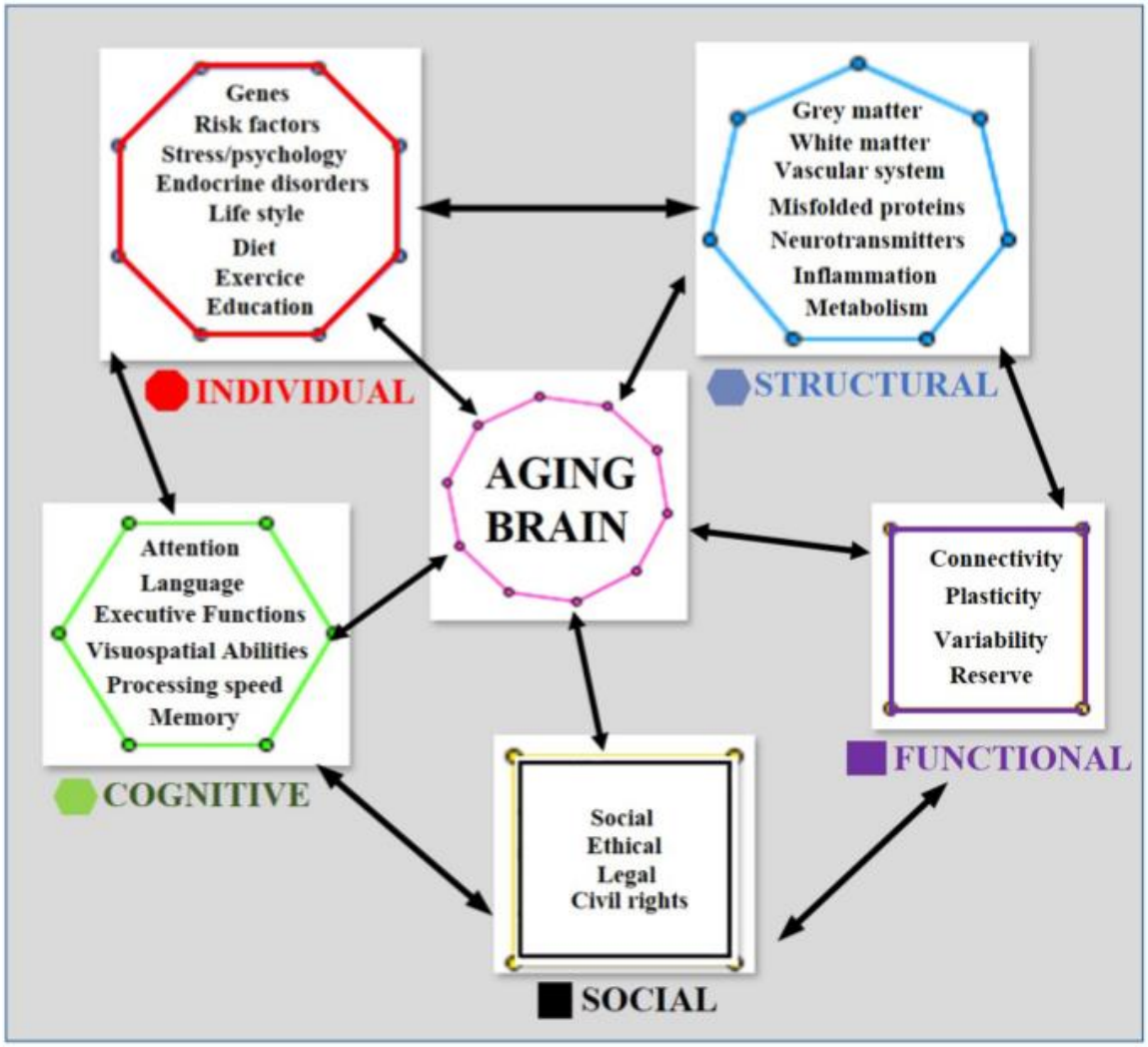


Figure 2.
NIA-Reagan Criteria for Neuropathological Alzheimer's Disease in Demented and Non-Demented Autopsy Participants in *The 90+ Study* 2003 Cohort (N=104)
AD Pathology defined as intermediate or high likelihood of AD based on NIA-Reagan Criteria
Other types of pathology include hippocampal sclerosis, diffuse Lewy body disease, Corticobasal degeneration, Braak tangle stage≥5, and vascular dementia pathology

Old age dementia:

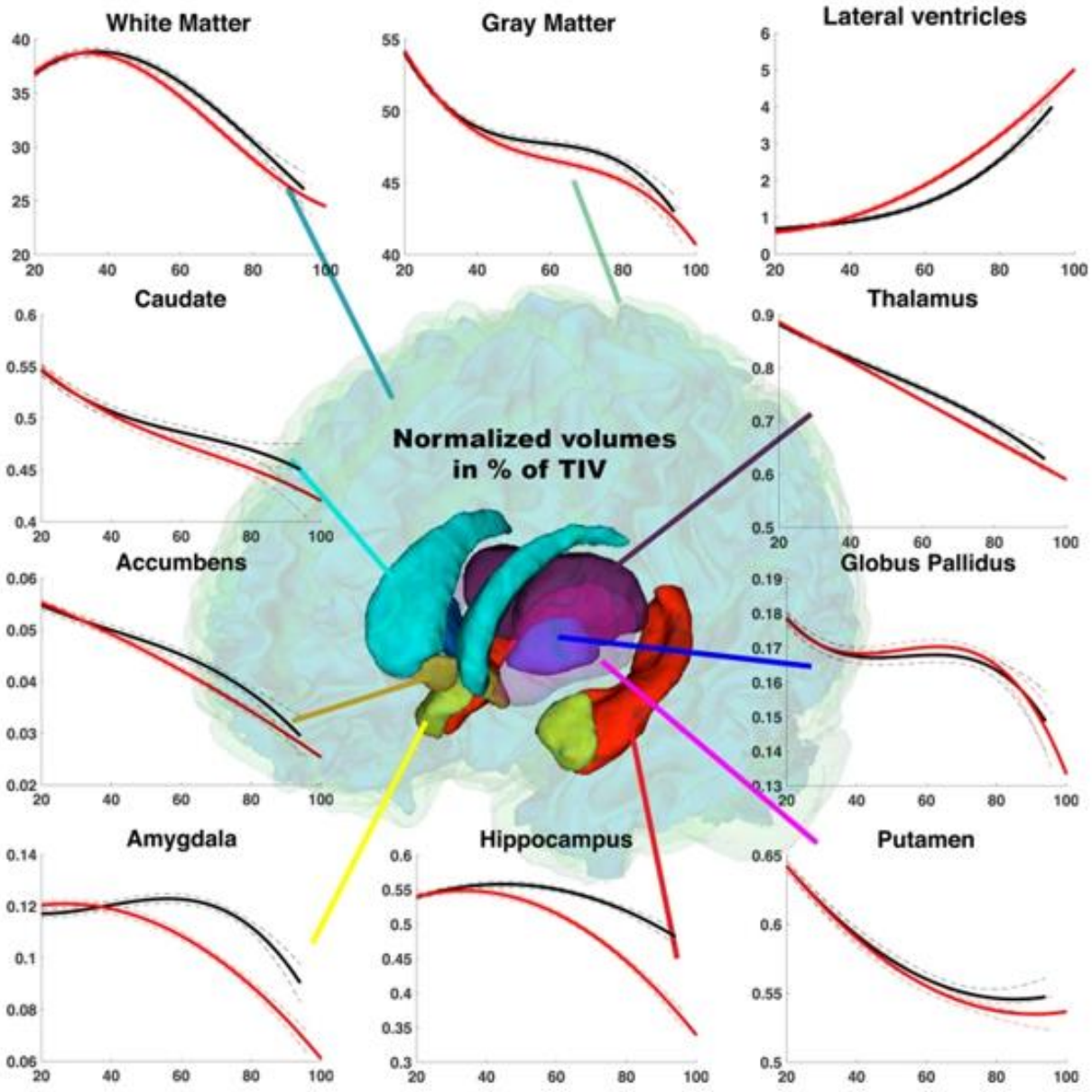
Alzheimer's disease, diseases or syndrome?

Human brain-aging is a complex, multidimensional phenomenon



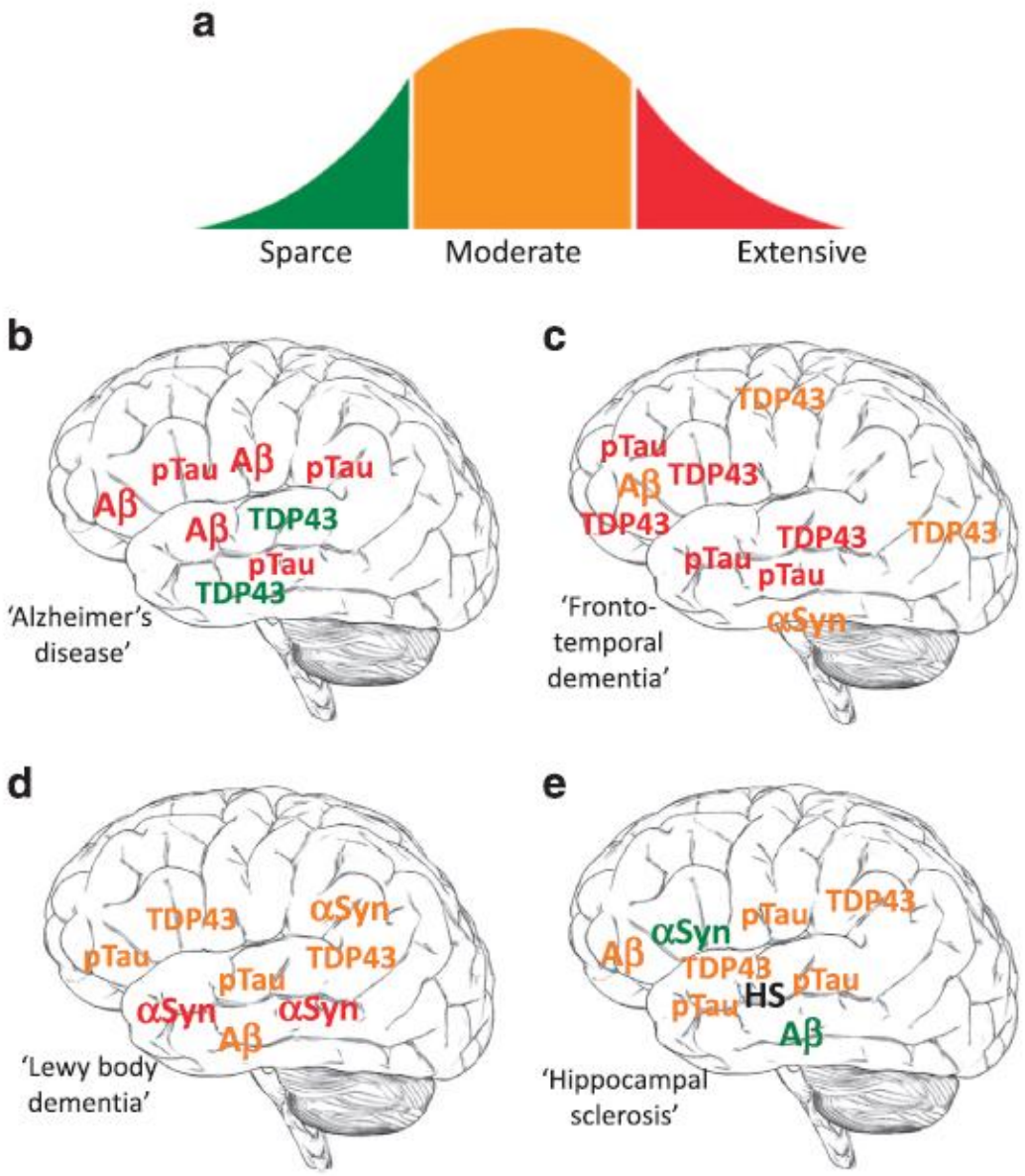
Front. Endocrinol 2019

Lifespan Changes of the Human Brain



Scientific Reports 2019

Each case of late-onset **dementia** is a unique mosaic of prototypical neuropathological landscapes



Mattson, Aging and Mechanisms of Disease, 2015

doi:10.1093/brain/awt273 Brain 2013: 136; 3738–3752 | 3738

BRAIN
A JOURNAL OF NEUROLOGY

Cell number changes in Alzheimer's disease relate to dementia, not to plaques and tangles

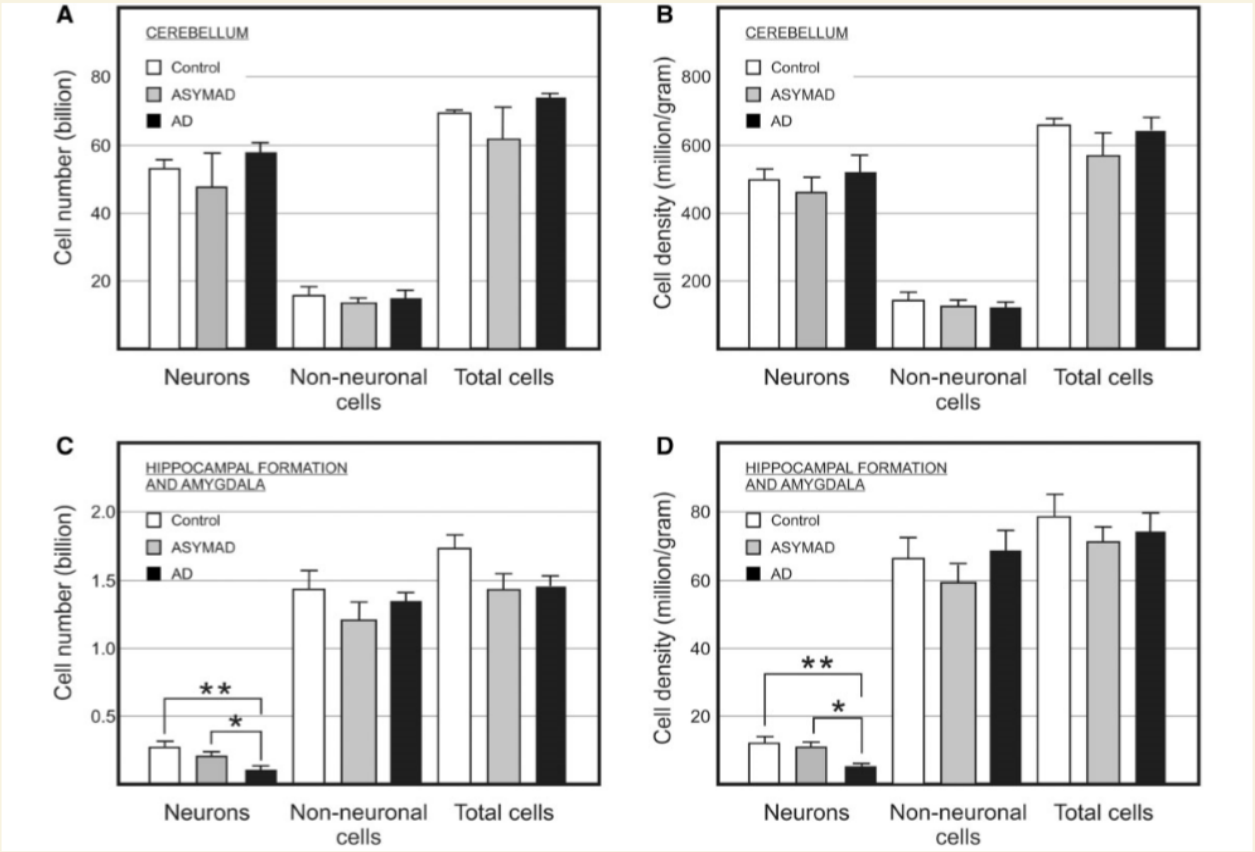
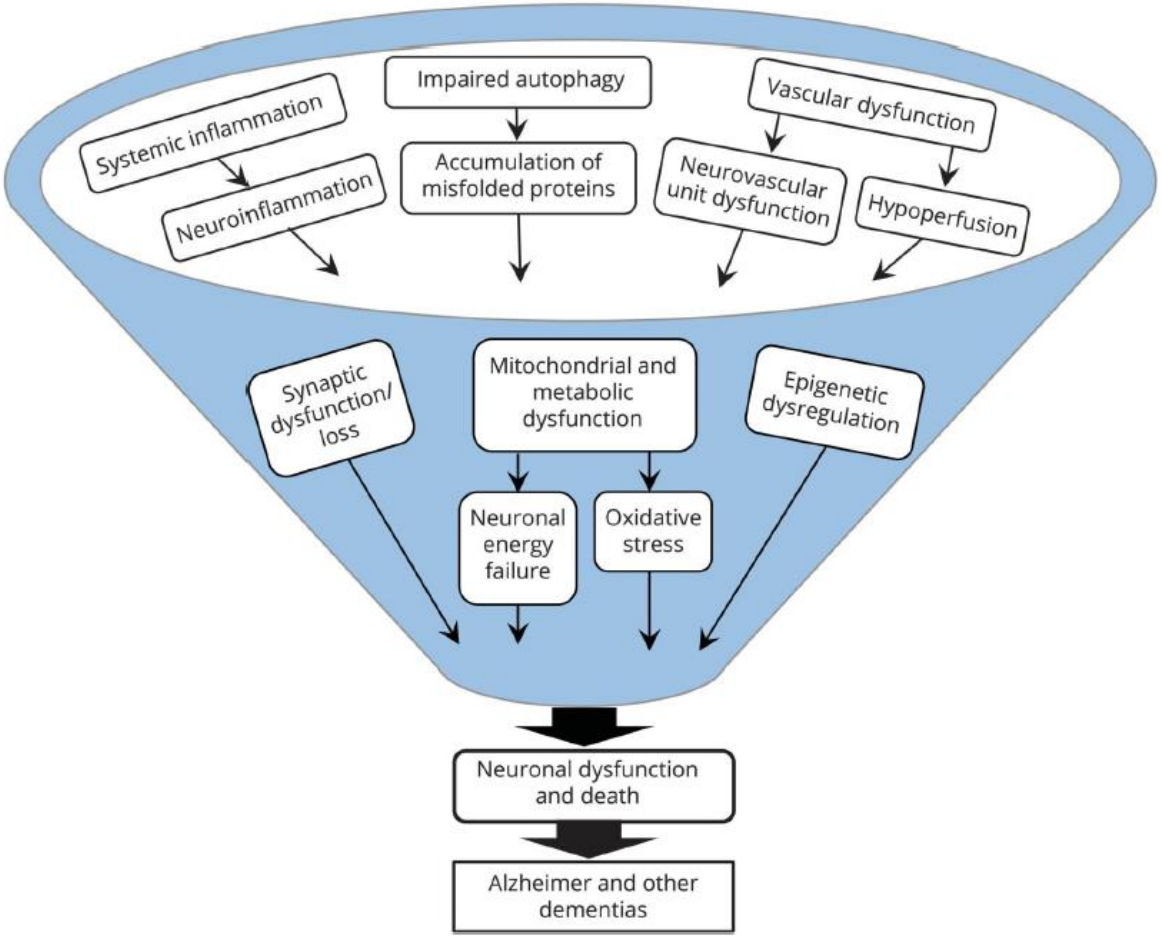


Figure 3 Absolute bilateral cell number (**A** and **C**) and density (**B** and **D**) of cerebellar (**A** and **B**) and hippocampal formation (**C** and **D**) neuronal, non-neuronal, and total cells in control, asymptomatic Alzheimer's disease (ASYMAD) and demented Alzheimer's disease (AD) groups. Each bar represents mean and standard deviation. Significant differences are indicated by $*P < 0.05$, $**P < 0.01$.

Andrade-Moraes et al., 2013

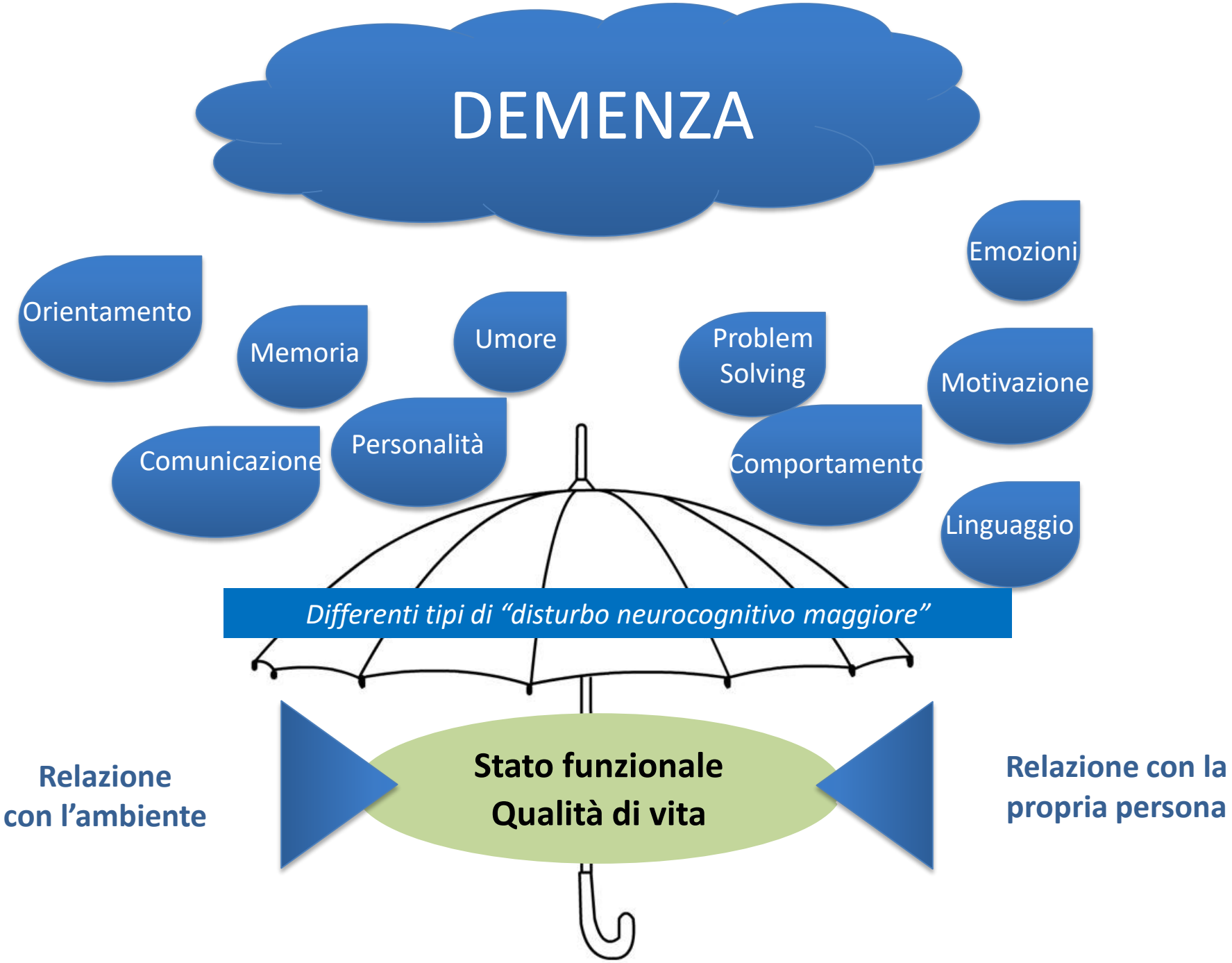
Aging, Alzheimer and other dementias

Age-related changes in biological processes contribute to neurodegeneration in Alzheimer disease (AD) and other dementias



Processes that are altered with aging that precede neurodegeneration include inflammation, impaired autophagy, vascular dysfunction, synaptic loss, mitochondrial and metabolic dysfunctions, and epigenetic changes. These processes provide numerous novel targets for new drug development for AD.

Hara et al., Neurology 2019


DEMENZA
Emozioni
Orientamento
Memoria
Umore
Problem Solving
Motivazione
Personalità
Comunicazione
Comportamento
Linguaggio
Differenti tipi di “disturbo neurocognitivo maggiore”
Relazione con l’ambiente
Stato funzionale
Qualità di vita
Relazione con la propria persona

geriatrics

MDPI

Editorial

Population Ageing: The Need for a Care Revolution in a World 2.0

Virginia Boccardi

Section of Gerontology and Geriatrics, Santa Maria della Misericordia Hospital, 60132 Perugia, Italy; virginia.boccardi@unipg.it; Tel.: +39-0755783524

Received: 7 August 2019; Accepted: 13 August 2019; Published: 14 August 2019

check for updates

Keywords: ageing; challenge; disease; frailty; care

Marjory Warren(1897-1960)

- to prevent disease whenever possible;
- to reduce medical disability to a minimum;
- to obtain and maintain maximum independence;
- to teach the patient to adjust himself intelligently to his residual ability

“Let’s put together the pieces”

Statistics on the burden of dementia: need for stronger data

THE LANCET
Neurology

Published Online
November 26, 2018
http://dx.doi.org/10.1016/

Dementia primarily affects an individual's cognitive function and many aspects of life are negatively affected by cognitive decline. There are no approved disease-modifying drugs and no approved prevention strategies for dementia; a heavy burden is placed on the individual who has dementia, their family, and society. In *The Lancet Neurology*, a report[1] from the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2016 Dementia Collaborators presents estimates of dementia-related deaths, prevalence, quality of life measures, and risk factors, with the aim of documenting global patterns and providing data for research, and to guide a wide range of public health investments.

Calculations were based on the GBD models that have been used to estimate the burden of more than 300 diseases and injuries in 195 countries and territories.[2] Because of the marked inconsistencies in the location-specific data for prevalence and incidence of dementia and mortality, and the marked heterogeneity in the studies included in this report, several of the assumptions that are usually used in the GBD methods could not be met. Therefore, the type of source data used and the modelling approaches were modified so that the data fitted the assumptions of the core GBD models. For example, for locations that did not have data available it appeared that the ratio of prevalence to cause-specific mortality from the USA, Puerto Rico, Finland, and Sweden were incorporated to estimate cause of death, prevalence, quality of life, and risk factors for dementia.

The report[1] makes an important point about the huge burden of dementia: in 2016, the global number of individuals who lived with dementia was 43·8 million (95% uncertainty interval [UI] 37·8–51·0), increased from 20·2 million (17·4–23·5) in 1990. The report also provides an opportunity to examine further the way in which dementia statistics are generated, particularly because obtaining reliable and valid dementia case counts presents challenges that do not apply to obtaining such data for many other common diseases.

Dementia is a multisystem condition. The definition is still evolving in research and clinical communities, and this affects how data are entered into administrative databases. Case ascertainment depends upon several aspects, such as the health-care infrastructure of a community, socioeconomic conditions, cultural norms, access to health care, age structure, modes of caring for elderly people, and recognition of pathological changes in function. Despite efforts to standardise dementia and Alzheimer's disease assessments since the early 1980s, dementia is still assessed in many different ways. Mild cases are not reliably identified, the terminology and categorisation of dementia have shifted over time, and there are differences in regional medical society guidelines, local context, and resources. The diagnosis can be operationalised by clinical judgment, algorithms, or questionnaire scales. There has also been a proposal[3] to define Alzheimer's disease, typically the most common clinically defined subtype of dementia, by biomarker criteria derived from MRI scans and from PET scans or CSF, regardless of cognitive function. If this proposal moves beyond a narrow research framework and into broader research efforts and clinics, further complexities will be created in data interpretation and use. For example, such criteria might result in an increased number of people diagnosed with dementia who have no functional impairment, or in fewer cases being identified in regions with no means to gather data on the proposed markers.

Going forward, there might also be challenges to our underlying models of prevalence and life lived with dementia. The currently accepted model is an exponential age-related increase in prevalence and incidence of dementia, with few cases occurring before age 70 years and many, but a poorly estimated number of, cases after age 90 years.[4,5] However, emerging data suggest that this model might be changing. For example, a study of data based on a large medical records database suggested there might be an increase in an alcohol-related earlier onset dementia;[6] several epidemiological studies have suggested there is a decline in incident cases of dementia;[7] people with dementia who are aged 90 years or older have different presentations compared with those who develop dementia at an earlier age as their presentation is complicated by multiple-morbidity;[8] and improvements in treating chronic diseases extend life, and therefore an increase in the number of people living with dementia is expected. At the same time, the increase in the occurrence of risk factors, such as diabetes,[5] at younger ages means that individuals might be exposed to risk sooner and possibly have an earlier onset of signs of dementia.

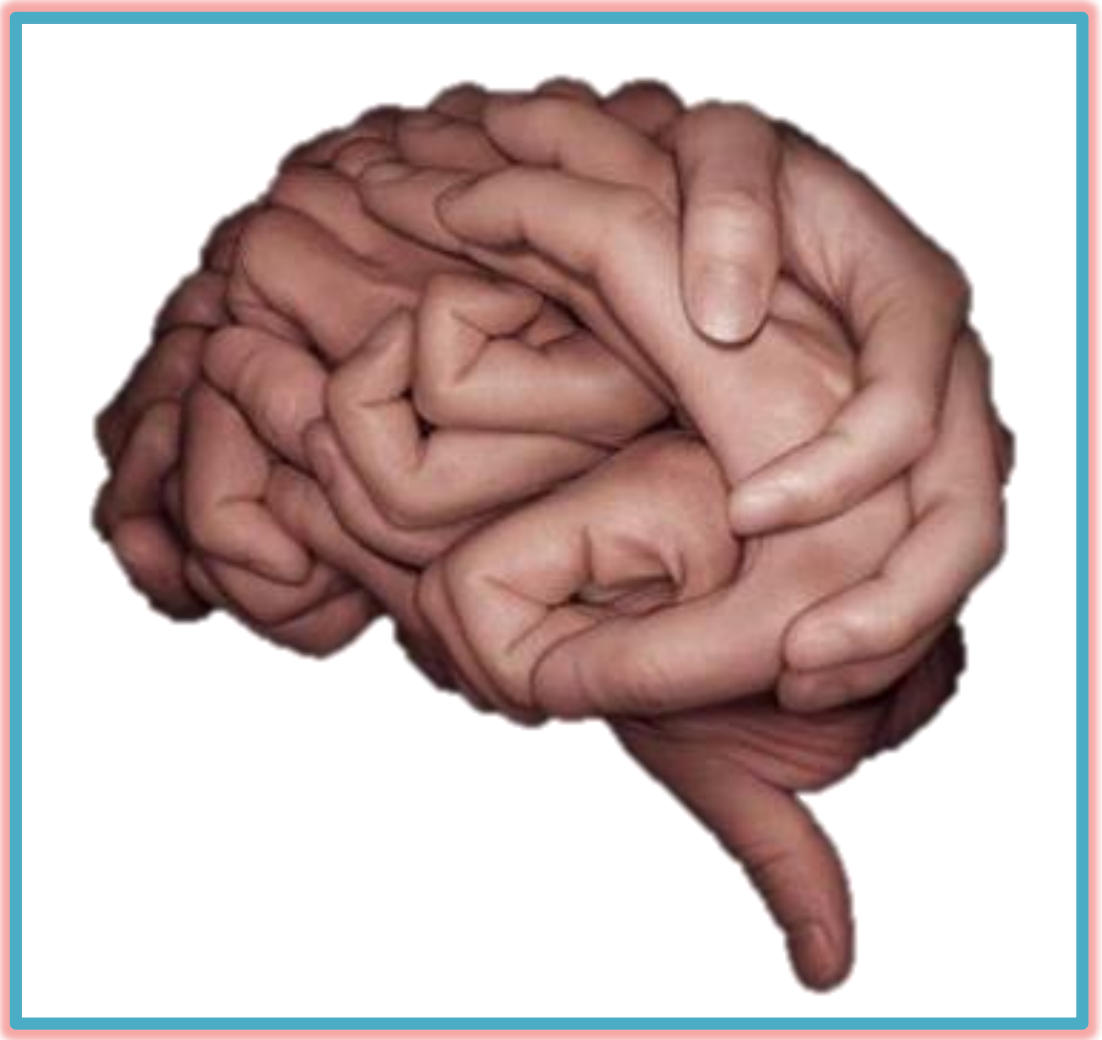
Although collecting data for public health purposes has different aims from those of research into the causes of dementia,[9] both are needed to reduce the burden of disease. The GBD 2016 Dementia Collaborators[1] report that 6·4 million (95% UI 3·4–10·5; 22·3%) of the total DALYs caused by dementia could be attributed to four modifiable risk factors that met GBD criteria for analysis (high body-mass index, high fasting plasma glucose, smoking, and a diet high in sugar-sweetened beverages). To put these findings into context, several reviews of risk factors[5] and methods to study dementia[9] are helpful. These sources have pointed out the complexities of understanding the trajectories of cognitive decline and risk factors, and the difficulties in interpreting studies that do not take into account the limitations of the study design and issues such as the selective loss over time of sicker individuals from the study, the quality and appropriateness of exposure and outcome measures, and the choice of statistical models.

From a public health and disease-prevention perspective, too few quality data are available for dementia that fit the complex reality of this devastating public health problem. Additionally, it is questionable whether the extant data are strong enough to help achieve the goals of this GBD study—to inform policy makers, researchers, and clinicians about global differences in dementia trends, clusters of dementia, and causal risk factors. To reach these goals, several areas of data collection and interpretation require strengthening: improvement of research methods used in data collection and interpretation; development of a consensus about valid coding of dementia for administrative databases; and development of flexible approaches that take into account the variation in place and over time of health and social conditions that might lead to severe cognitive impairment.

- Epidemiological model might be changing
- Risk factors might be changing
- Few quality data are available for disease-prevention

2019

Rete Geriatrica Alzheimer 2.0



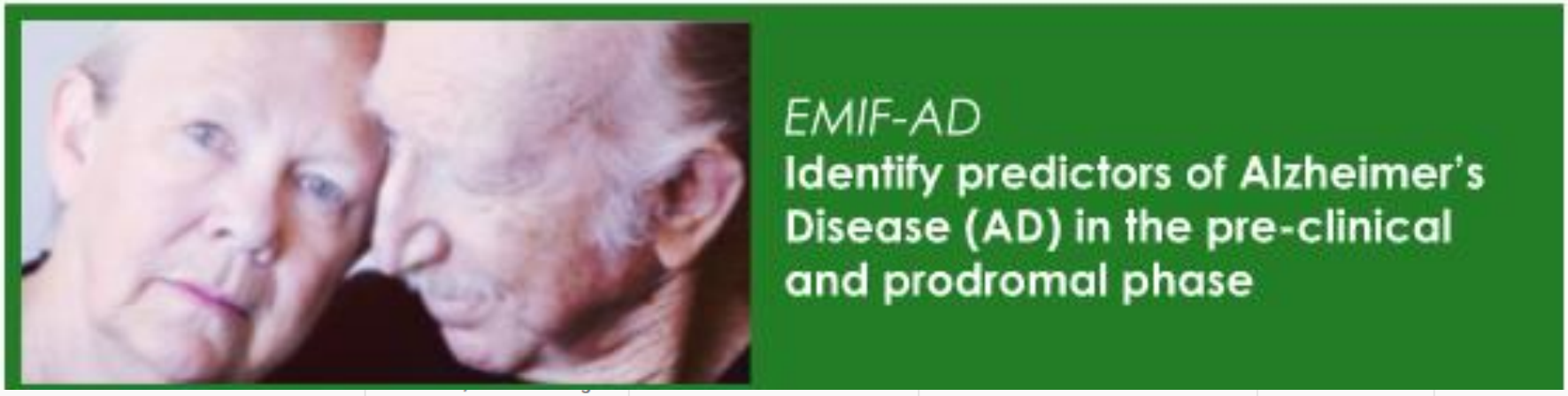
IL PROGETTO REGAL 2.0
LA CARTELLA

ReGAI 2.0

- Più vicina ai modelli informatici attuali
- Accesso tramite web
- In costante aggiornamento
- Disponibilità di un unico strumento per l'approccio al paziente

- Coinvolgimento di più figure professionali dedicate alla cura del soggetto con demenza
- Creazione di un unico "profilo informatizzato" del paziente
- Creazione di un grande dataset
- Condivisione delle conoscenze e delle metodiche di ricerca

PROGETTO REGAL
NEL NETWORK EUROPEO



Pre-AI study	Prediction of Alzheimer's disease	INSERM U610 and AP-HP Fédération de Neurologie, Hôpital de la Salpétrière	Paris, Paris, Paris, Île-de-France, France	58	251
PSI	Parelsnoer Institute	Maastricht University	Gemeente Maastricht, Provincie Limburg, Netherlands		664
RECALL - HNR	Heinz Nixdorf Recall Study	University of Duisburg Essen	Essen, Kreisfreie Stadt Essen, Regierungsbezirk Düsseldorf, Nordrhein-Westfalen, Germany	45,75	2631
ReGAI Project	Rete Geriatrica Alzheimer (Italian Geriatric Network)	Istituto di Gerontologia e Geriatria	Perugia, Provincia di Perugia, Umbria, Italy	50, 95	3592
SNAC-K	The Swedish National Study on Aging and Care in Kungsholmen	Karolinska Institutet	Botkyrka Kommun, Stockholm, Sweden	60,104	3261

PROGETTO REGAL
NEL NETWORK EUROPEO

JPND research
EU Joint Programme – Neurodegenerative Disease Research

JPND is the largest global research initiative
aimed at tackling the challenge of neurodegenerative diseases

COHORT DATA

Cohort Acronym
ReGAl

Cohort type
General population-based cohort

Disease
Alzheimer's disease, Frontotemporal dementia, Lewy body disease, Mild cognitive impairment (MCI), Parkinson's disease, Subjective memory complaints (SMC) or subjective cognitive decline (SCD), Vascular dementia

Participant type
No diagnosis

Profile

Recruitment Period	Jan 2000 - April 2017
Sample size at start or planned sample size if still recruiting	3897
Estimated Current Sample Size	0 to 4,999
Age at Recruitment	50-100
Gender	Male and Female
Abstract	In this epidemiological study we examined the prevalence of medical comorbidity in elderly subjects with cognitive deficits and dementia. The ReGAl Project (Rete Geriatrica Alzheimer- Geriatric Network on Alzheimer's disease) collected data in 33 Italian Geriatric memory clinics from January 2001 to December 2005. A total of 4,075 patient were recruited.

Last Update 21/09/2017

ıvestiator (PI)	Professor Patrizia Mecocci and Dr. Virginia Boccardi
Principal Investiator (PI)	Professor Patrizia Mecocci and Dr. Virginia Boccardi
Contact email	Email: info@regalproject.it
Contact phone number	
Address	Prof. Patrizia Mecocci, Section of Gerontology and Geriatrics, Department of Medicine, University of Perugia Piazza Menghini, 1 IT-06100 Perugia
Funders (Core support)	

VARIABLES COLLECTED

Brain related measures:	Mental health, Cognitive function, Behaviour, Neurological
Funtional rating:	Caregiver
Anthropometric:	Blood pressure, Height, Hip circumference, Waist circumference, Weight
Physical:	Cardiovascular, Hearing and Vision, Musculoskeletal, Respiratory
Biological samples:	N/A
Genotyping:	Gene screening
Brain imaging:	Magnetic resonance imaging (MRI), Magnetic resonance spectroscopy (MRS), Positron emission tomography (PET) fluorine18 flurodeoxyglucose (FDG)
Brain banking:	N/A
Lifestyle:	Smoking, Alcohol
Socio-economic:	Education, Ethnic group, Housing and accommodation, Income and finances, Marital status, Occupation and employment, Unpaid care
Health service utilisation:	Formal health and social care service utilisation including private care

PROGETTO REGAL
NEL NETWORK EUROPEO

Perugia

Patrizia Mecocci MD PhD
Professor of Gerontology and Geriatrics, University of Perugia
Director Unit of Geriatrics, Ospedale S. Maria della Misericordia Perugia
Blocco C Piano 4
S.Andrea delle Fratte
06156 Perugia (Italy)
Tel: +39 075 578 3270
Fax: +39 075 573 3878
mecocci@unipg.it

Virginia Boccardi
virginia.boccardi@unipg.it

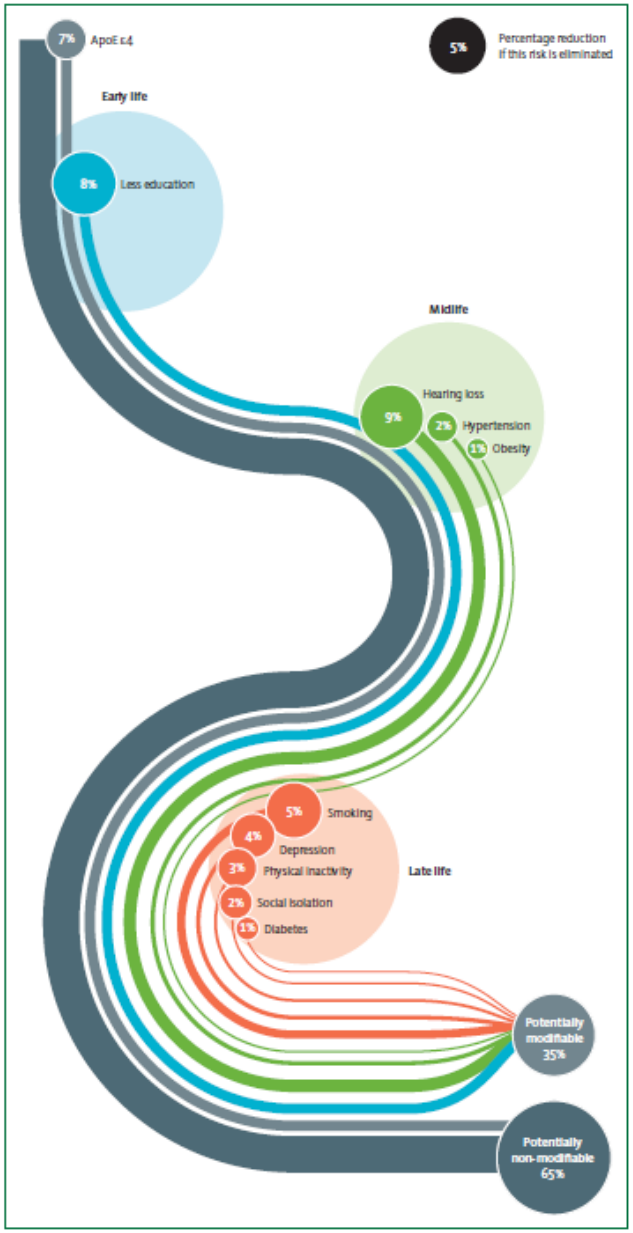
L'inquadramento dei deficit cognitivi nell'anziano

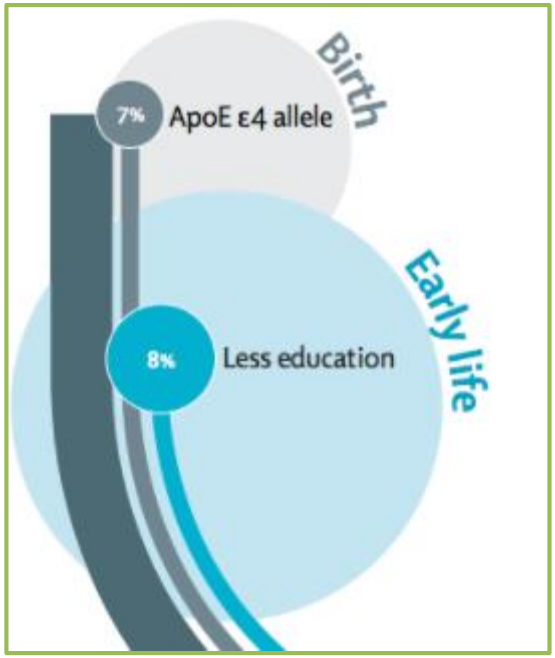
La valutazione multidimensionale

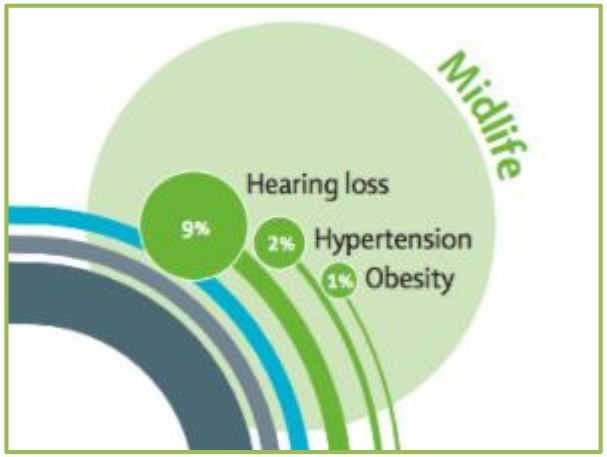
ReGAI 2.0

- Stato cognitivo
- Stato funzionale
- Stato psico-affettivo
- Comorbilità e farmacoterapia
- Stress del caregiver
- Stato socio-ambientale
- Stato nutrizionale

Dementia: a life course-approach







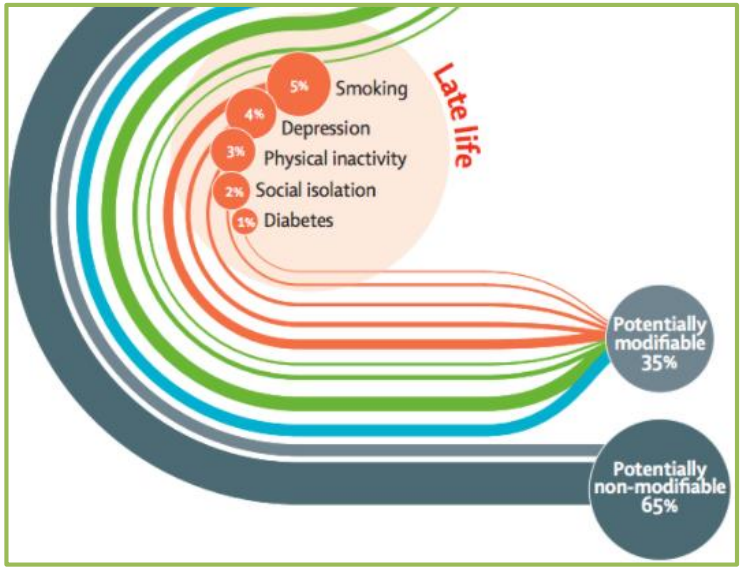


Figure 4: **Life-course model of contribution of modifiable risk factors to dementia**
Numbers are rounded to nearest integer. Figure shows potentially modifiable or non-modifiable risk factors.

Livingston et al., Lancet 2017

Pattern of changes in subjects with cognitive impairment long 10 years: results from the ReGAl

Journal of Systems and Integrative Neuroscience

oat open access text

Editorial ISSN: 2059-9781

The aging caregiver in the aged world of dementia

Virginia Boccardi and Patrizia Mecocci*

Institute of Gerontology and Geriatrics, Department of Medicine, University of Perugia, Italy

Old age population is growing worldwide at an impressive rate. Today, 8.5 percent of people (around 617 million) are aged 65 and over. This percentage is projected to increase to nearly 17 percent of the world's population by 2050 (1.6 billion) [1]. Population ageing is undoubtedly a demographic success, driven by changes in fertility and mortality due to economic and social development and to scientific progresses able to guarantee a better health for a larger number of people. However, as people live longer, chronic diseases become

arrangements are often critical and situations become unmanageable. Looking into data from the ReGAl project (Rete Geriatrica Alzheimer-Geriatric Network on Alzheimer's disease) -a large longitudinal Italian multicentric clinical-based study, promoted by the Italian Society of Gerontology and Geriatrics- from 2001 to 2005 we collected data from 544 caregivers of elderly subjects with dementia and found that their mean age was 56 years. Of them, 12% aged over 75 years. What happened just after ten years? Looking into data of the 2017 cohort, the mean age of 187 caregivers raised to 58 years and 15% were over 75 (unpublished data) and these percentages are projected to increase.

on their lives [3-5]. They have to face, understand and manage the changes they see, day by day, in their relative, provide emotional and practical help, and make difficult decisions about treatment options, use of services, finances, and long-term care. Thus, they also become "patient" (the person who suffers, in Latin), who needs support and assistance. Caregiving of dementia carries high financial, social, and emotional price. What is a new and unknown experience, in the aged world of dementia, is that caregivers aged too. Thus, it is not unusual to get into social contexts where a disabled old daughter must take care of a centenarian mother with dementia. For this reason, home care

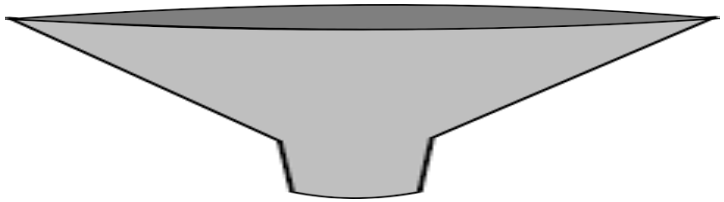
References

1. World Health Organization. An Aging World (2011). [Crossref]

Correspondence to: Patrizia Mecocci, MD PhD, Institute of Gerontology and Geriatrics, Department of Medicine, University of Perugia, Piazzale Gambuli 1, 06132 Perugia, Italy, Tel: +39 0755783270, E-mail: patrizia.mecocci@unipg.it

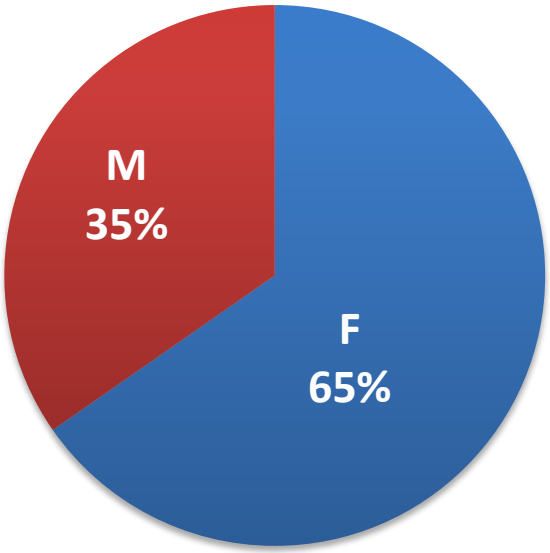
Received: August 25, 2017; **Accepted:** September 13, 2017; **Published:** September 15, 2017

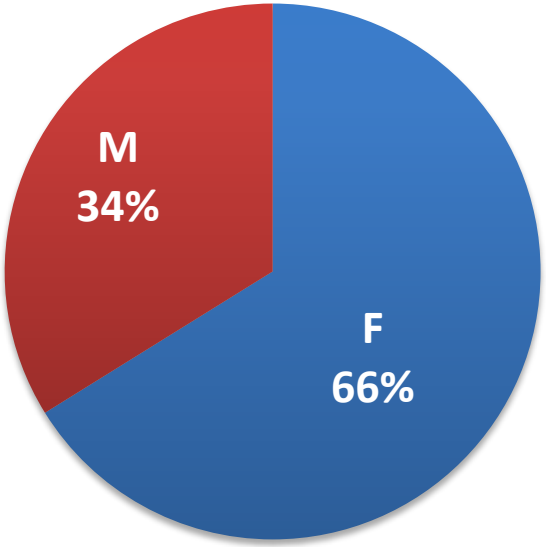
4500 subjects with cognitive impairment

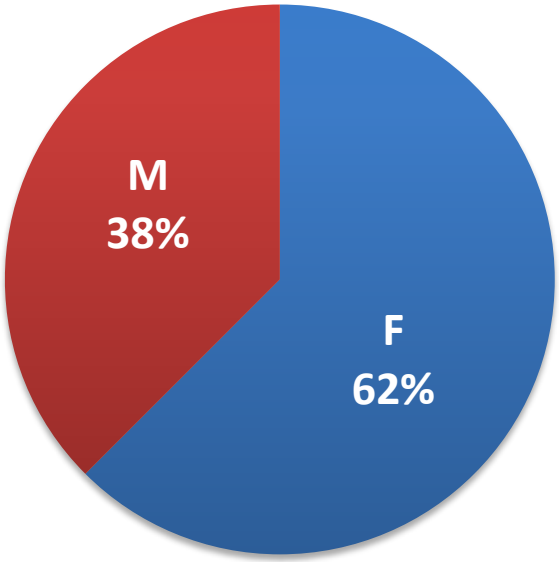


1178

2000-2001 | **2005-2006** | **2010-2011**







594 (F:388 M:206)

328 (F:217 M:111)

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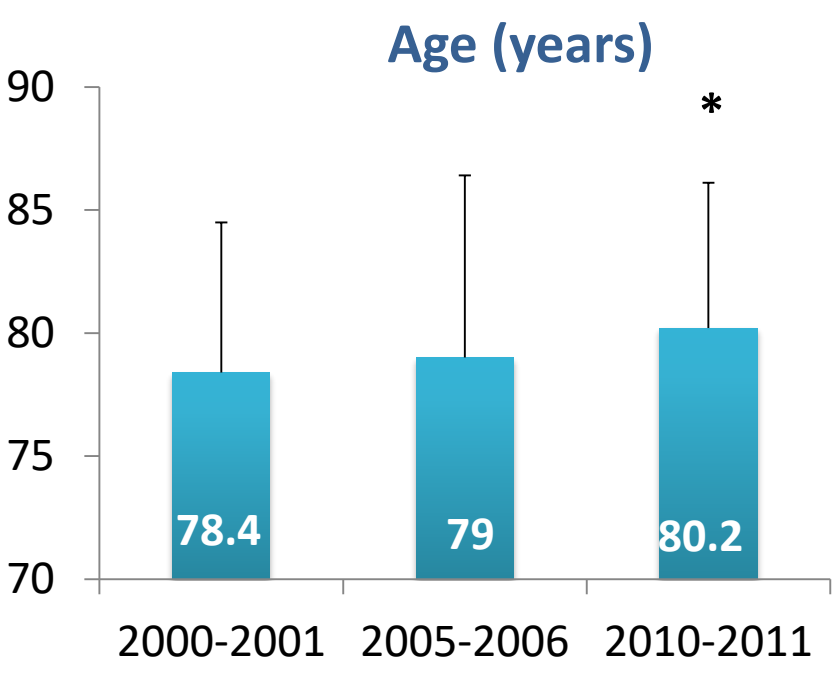

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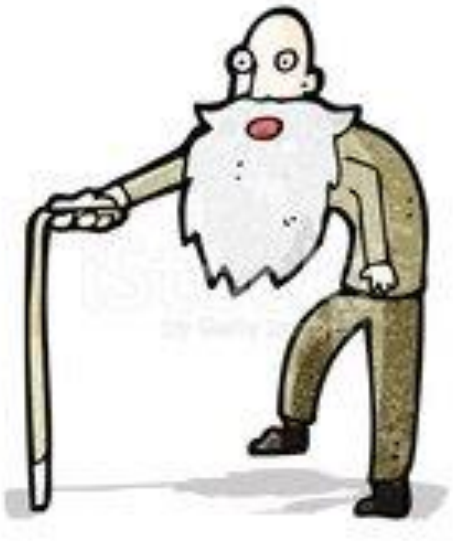

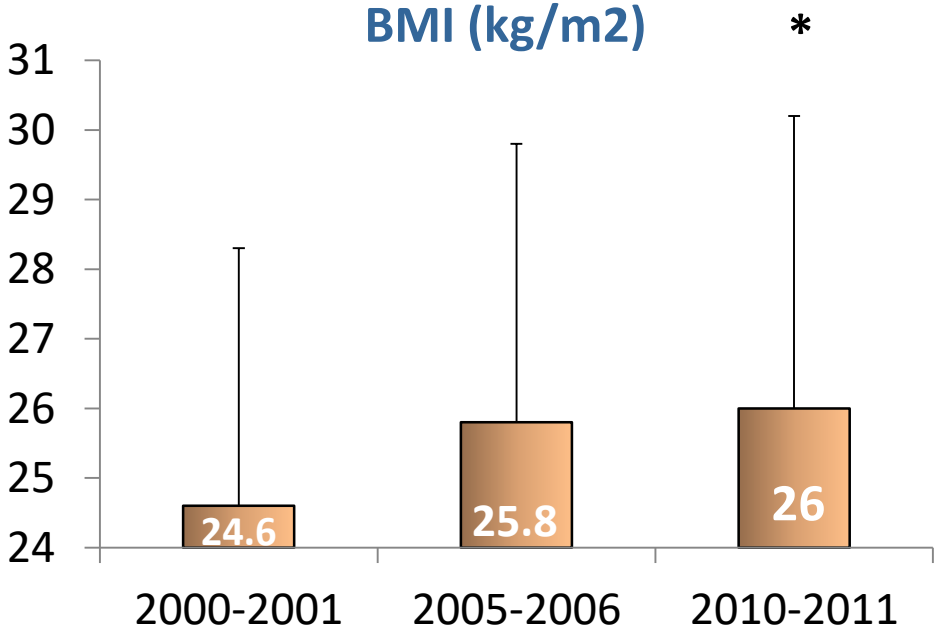

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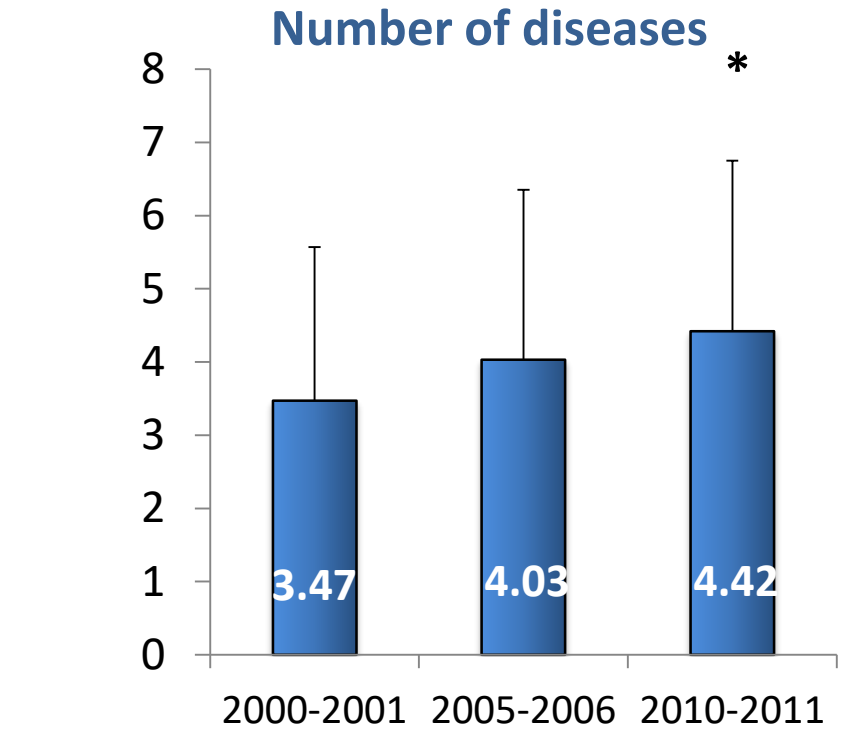

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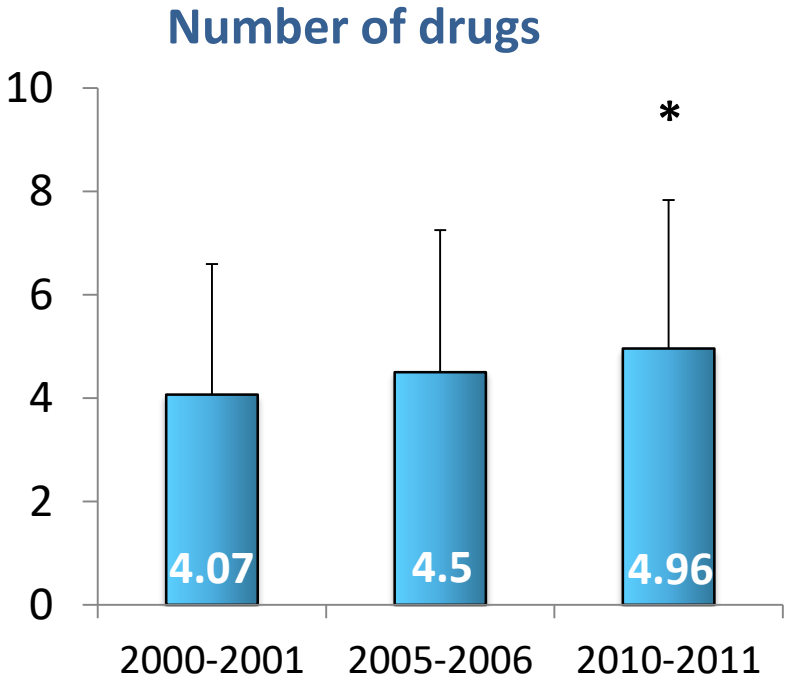

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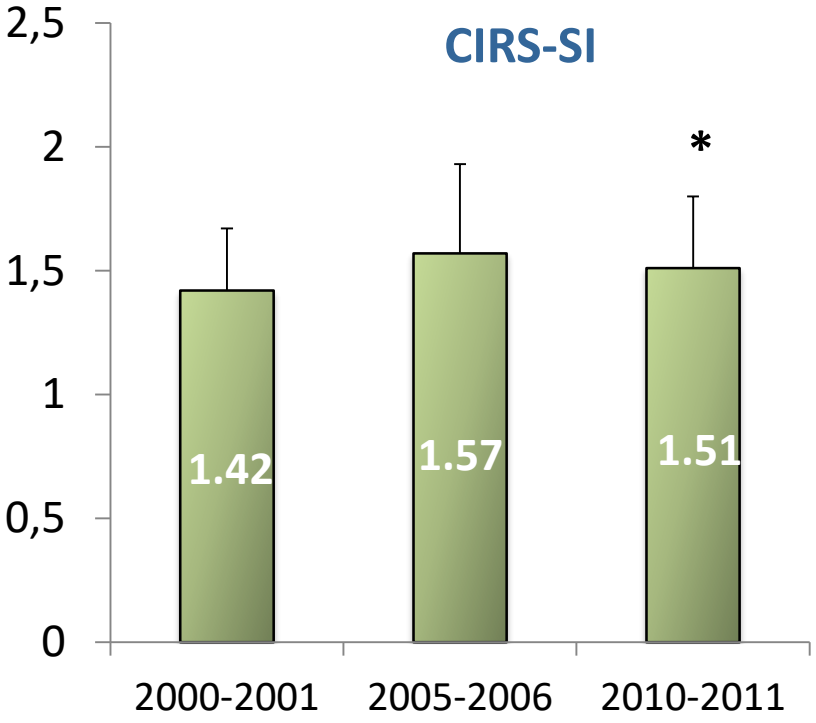

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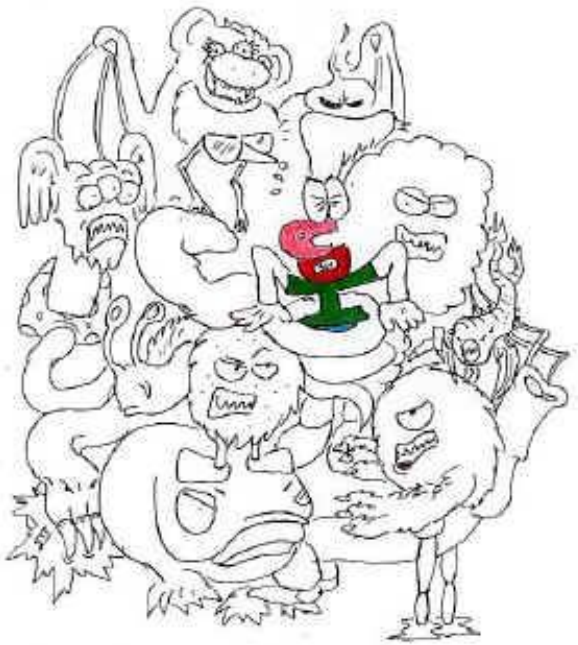

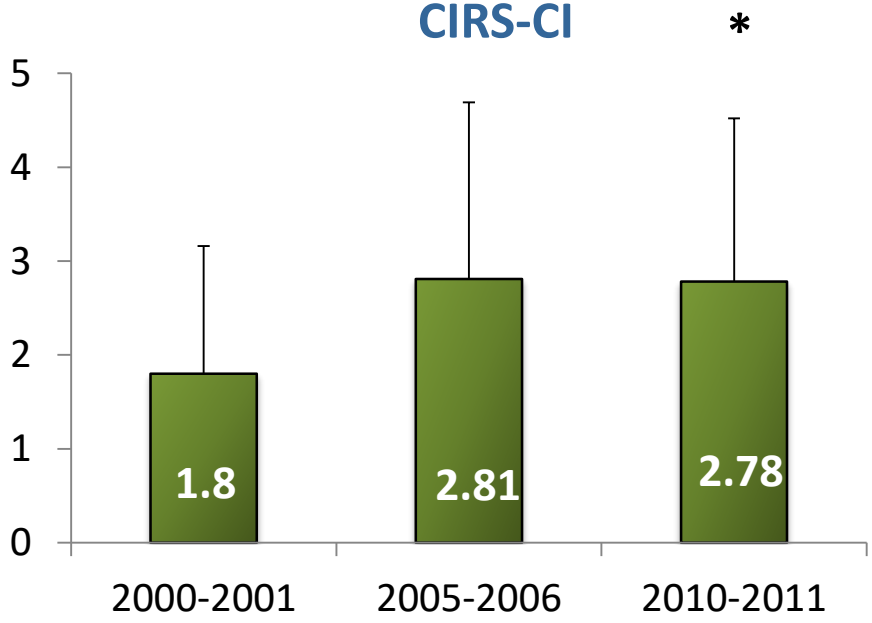

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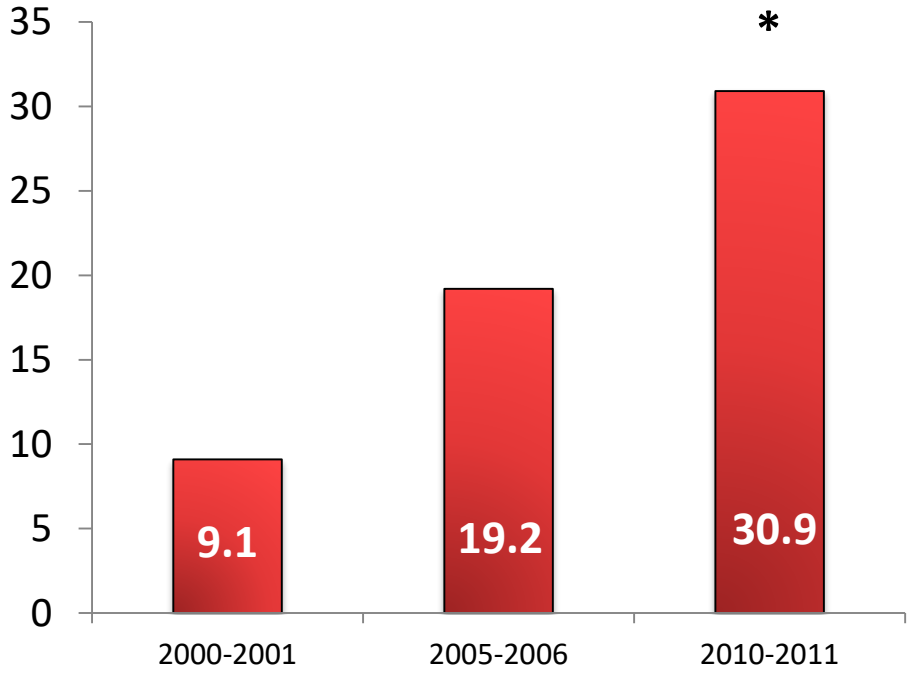
Obesity (BMI>30 Kg/m2)



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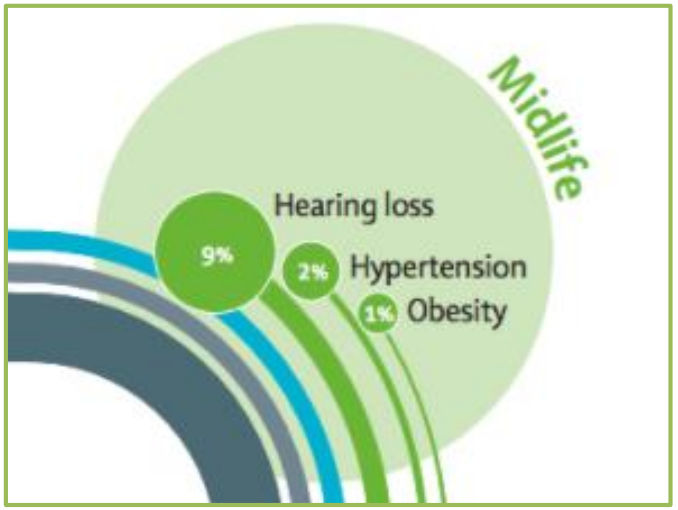
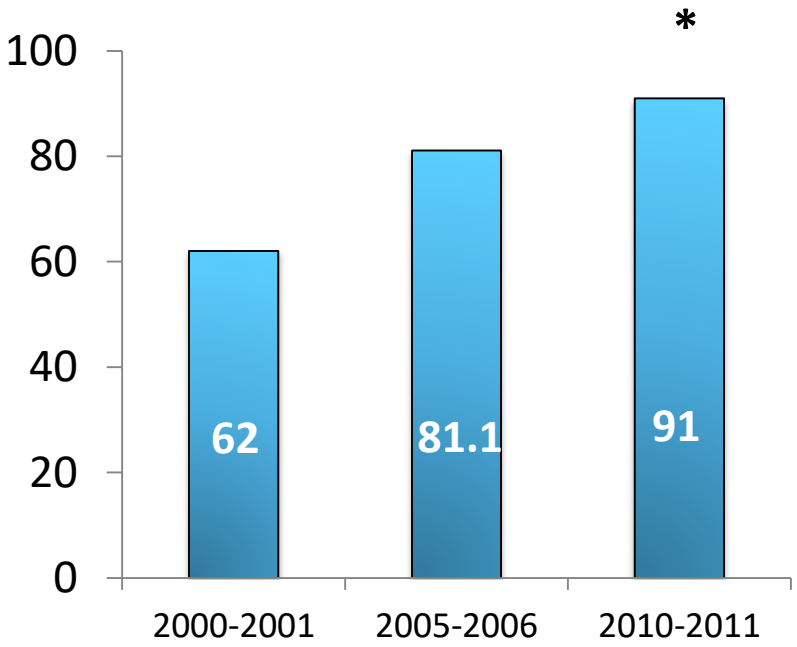


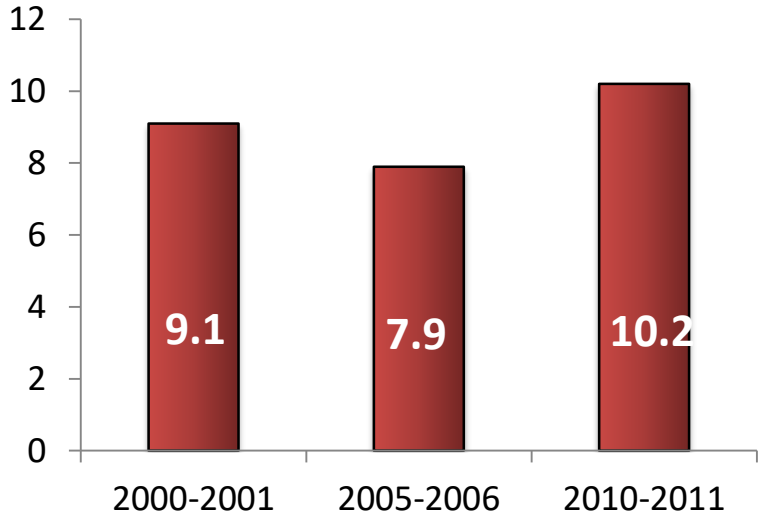
Hyperthension (JNCVII)



I dati sono espressi in frequenza %

*p=0.039

Smoking habit



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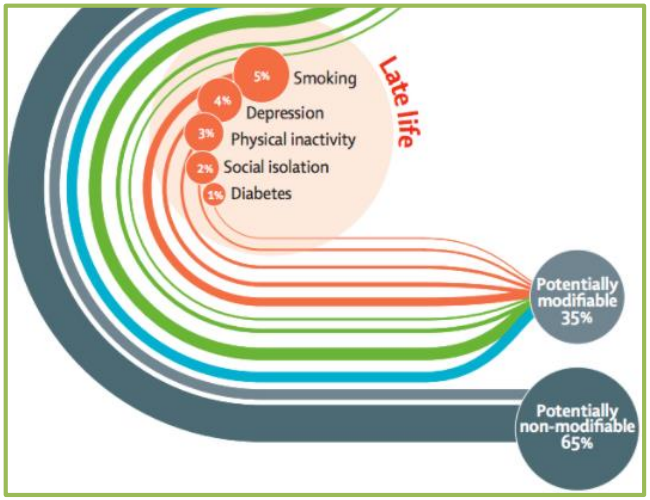
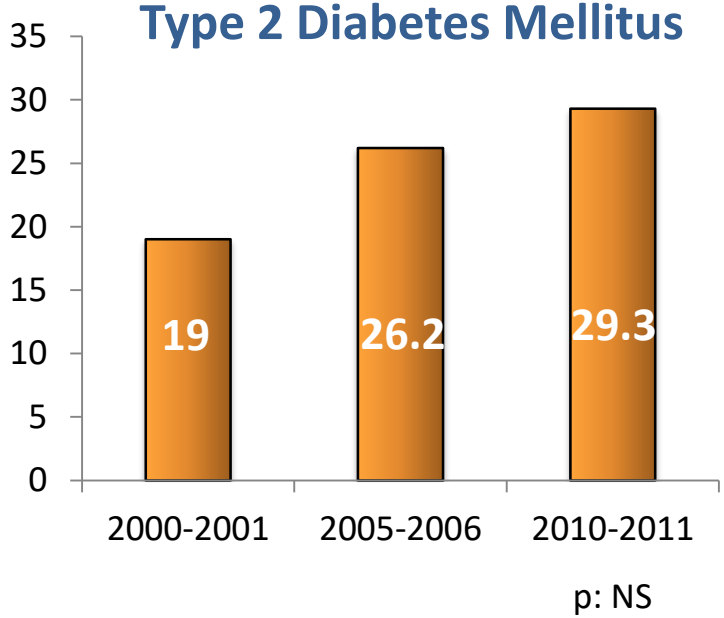


Type 2 Diabetes Mellitus



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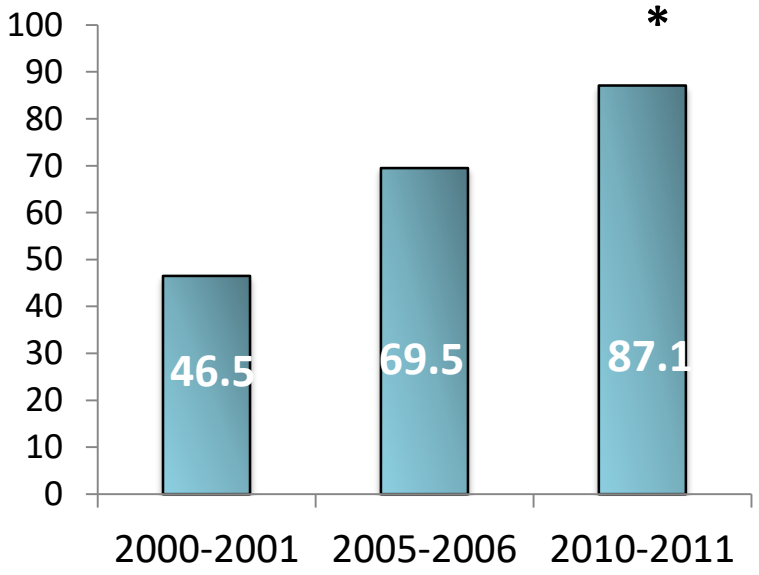
Depression (DSM IV)



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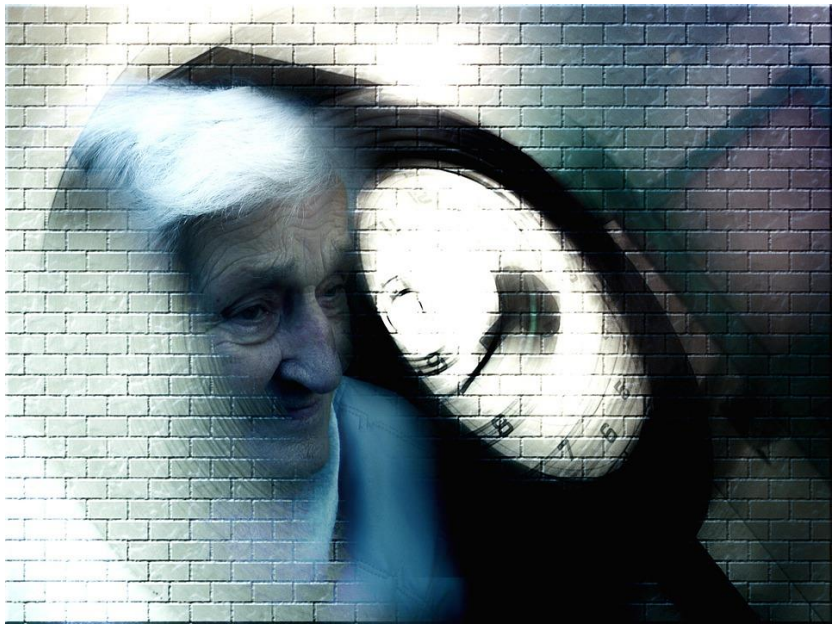
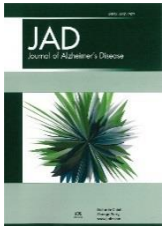
I dati sono espressi in frequenza %

Cognitive decline and Alzheimer's disease: when only prevention makes sense



The future of Alzheimer's disease therapy might be viewed as a combination approach or multitargeted therapeutic "cocktail"

- **Understanding early changes in Alzheimer's disease and mild cognitive impairment**
- **Developing surrogate markers for AD and MCI**

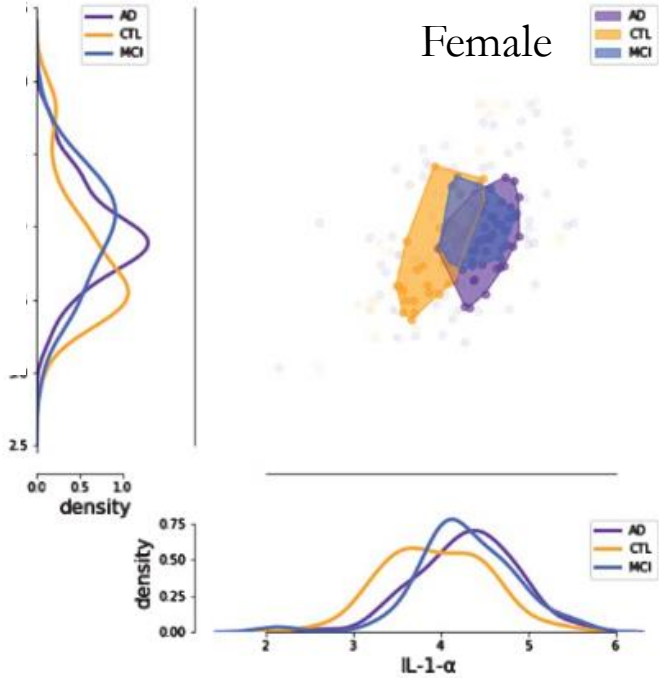


Cognitive Decline and Alzheimer's Disease in Old Age: A Sex-Specific Cytokinome Signature

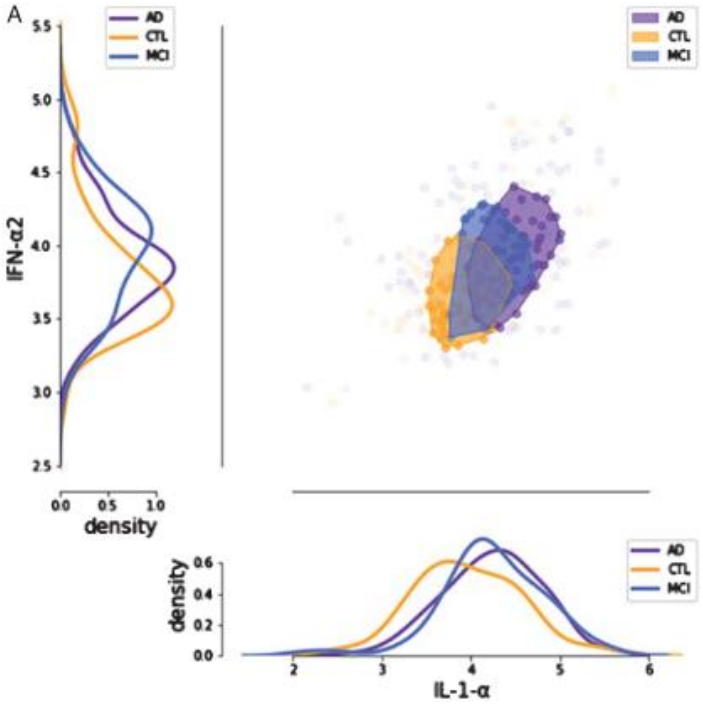
Virginia Boccardi[a,*], Lucia Paolacci[a], Daniel Remondini[b], Enrico Giampieri[b], Giulia Poli[c], Nico Curti[b], Roberta Cecchetti[a], Alfredo Villa[d], Carmelinda Ruggiero[a], Stefano Brancorsini[c] and Patrizia Mecocci[a]

[a]*Institute of Gerontology and Geriatrics, Department of Medicine, University of Perugia, Italy*
[b]*Department of Physics and Astronomy, University of Bologna, and INFN Bologna, Bologna, Italy*
[c]*Department of Experimental Medicine, Section of Terni, University of Perugia, Perugia, Italy*
[d]*Department of Clinical Pathology, S.M. della Misericordia Hospital, Perugia, Italy*

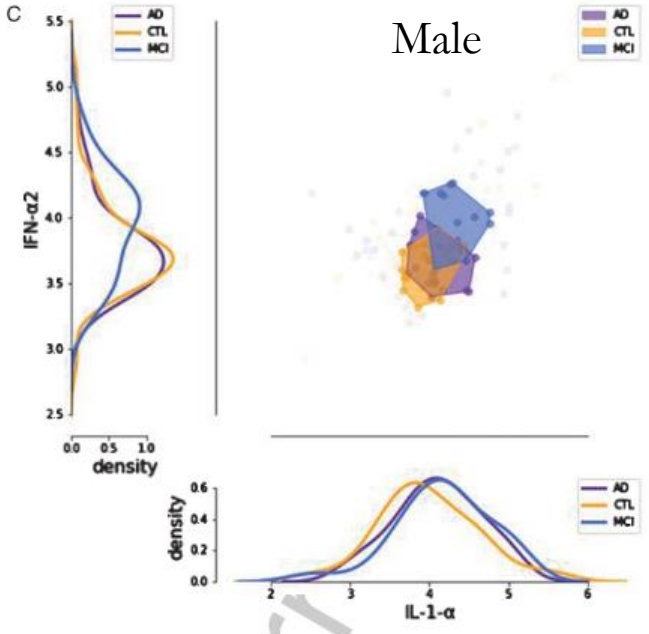
Female



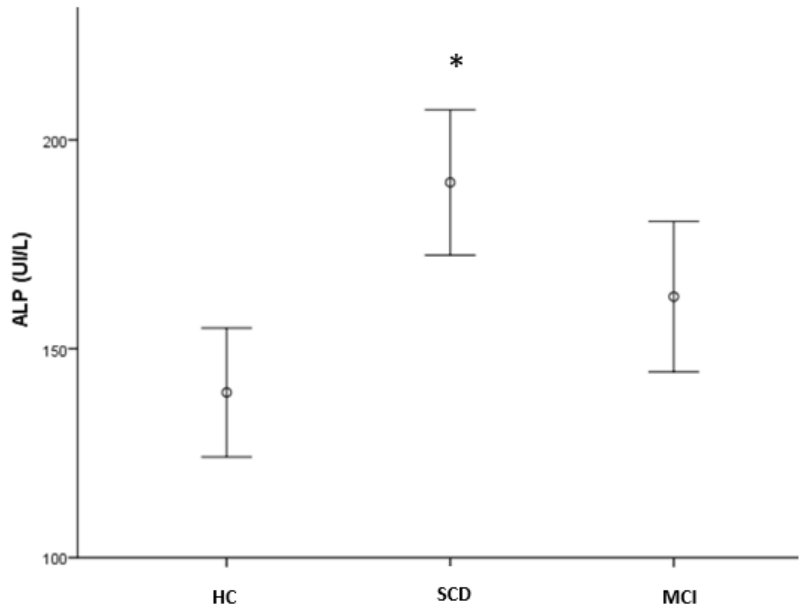
All population



Male



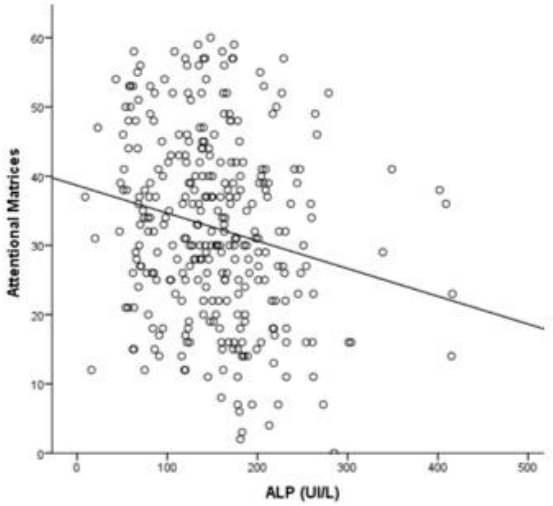
Serum alkaline phosphatase is elevated and inversely correlated with cognitive functions in subjective cognitive decline: results from the ReGAl 2.0 project



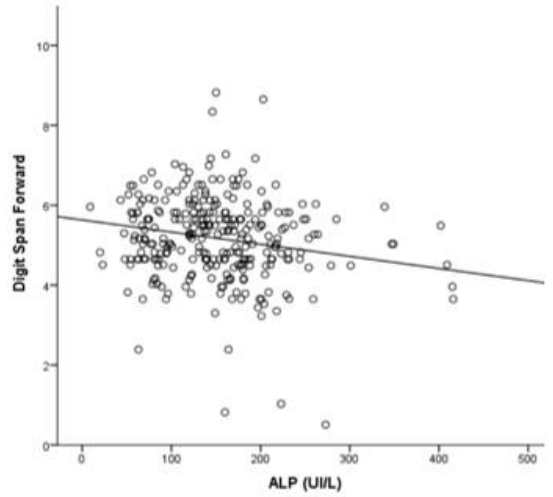
Data are expressed as means ± Standard Deviation (SD).

HC= Healthy Control; SCD= Subjective Cognitive Decline; MCI = Mild Cognitive Impairment.

*SCD vs HC (p=0.001) by Tukey's post hoc



r= -0.245, p=0.001 by simple Pearson's correlation.



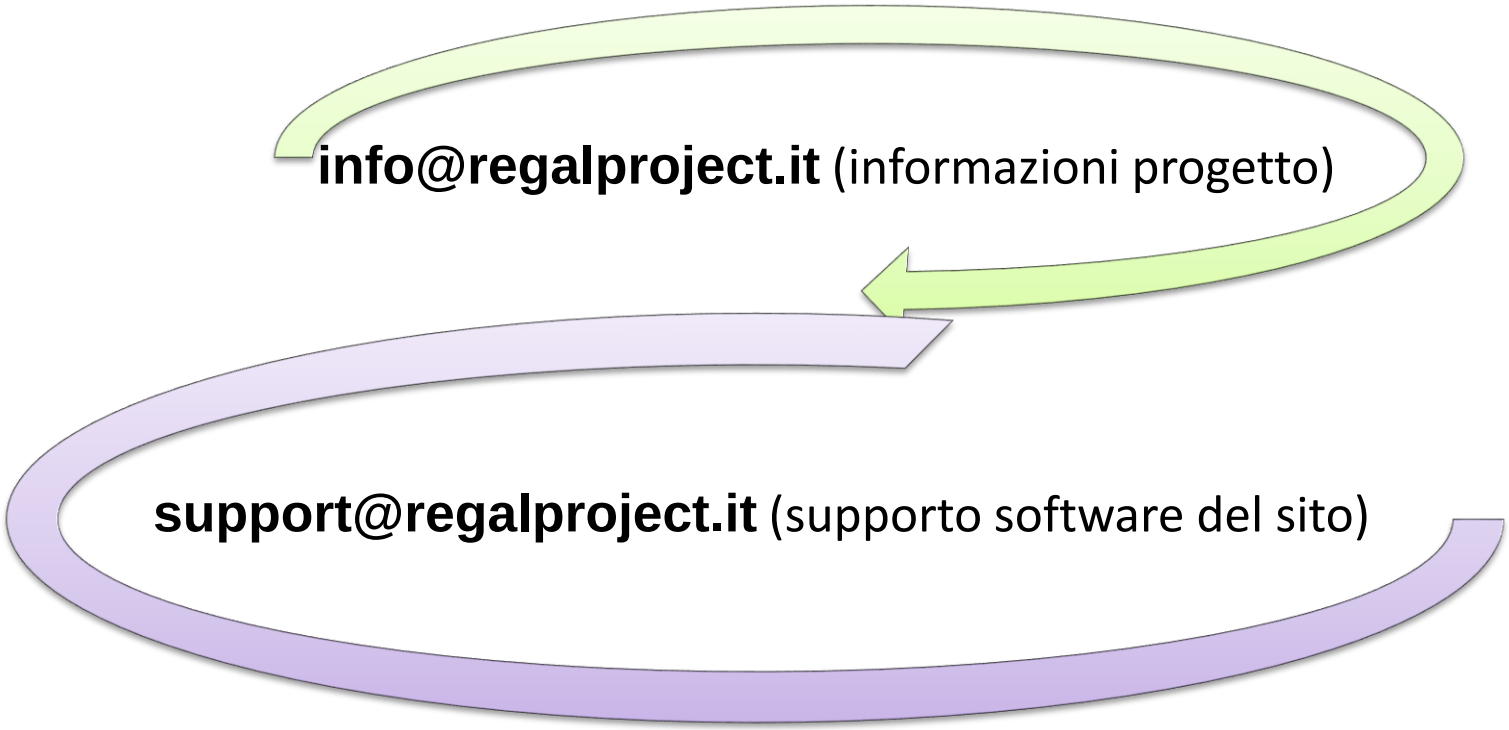
r= -0.227, p=0.005 by simple Pearson's correlation.

Under review

"The hope is that the combination of tailored prevention approach, the early identification of the person at risk, comorbidities treatment and the intervention with novel disease-modifying therapeutics will allow individ... free from the scourge of demen... retain their valuable memories and self-identity"

Grazie per l'attenzione

CONTATTI



Link: http://www.regalproject.eu
http://www.regalproject.it