Il deterioramento cognitivo, fattori di rischio e biomarcatori: GLP-1 nel ridurre infiammazione ed aterosclerosi

Genova,15 Novembre 2025

Dott. Claudio Ivaldi

Geriatra

Responsabile CDCD ASL 3

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15/11/2025

Dott. Claudio Ivaldi

Sommario

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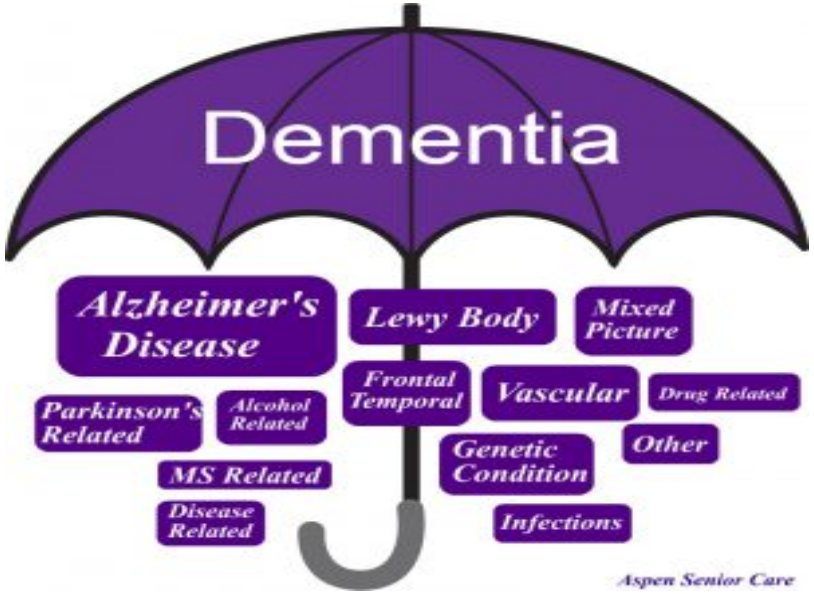
Che cosa è la Demenza?

"Demenza" è un termine a ombrello, che raccoglie molte malattie che coinvolgono la memoria, le altre funzioni cognitive e i comportamenti tali da interferire in maniera significativa con la capacità di mantenere le comuni attività della vita quotidiana



World Health Organization

Sebbene l'invecchiamento sia il principale fattore di rischio noto per demenza, questa non è una caratteristica dell'invecchiamento normale

Che cosa è la demenza?

E’ una sindrome (insieme di segni e sintomi) caratterizzata da:

- Disturbi cognitivi
- Disturbi non cognitivi

che determinano incapacità di compiere le attività della vita quotidiana

Disturbo neurocognitivo maggiore (DSM 5 TR)

- **A. Evidenza di una perdita significativa rispetto ad un precedente livello di performances in uno o più domini cognitivi (attenzione complessa, funzioni esecutive, memoria ed apprendimento, linguaggio, funzione percettivo-motoria o cognizione sociale) sulla base di :**
  - **1. preoccupazione del soggetto, di un informatore affidabile, o del clinico che vi sia stato un significativo declino delle funzioni cognitive; e**
  - **2. Compromissione significativa delle prestazioni cognitive, preferibilmente documentata da test neurospicologici standardizzati o, in loro assenza, da altra valutazione clinica quantificata**
  - **B. I deficit cognitivi interferiscono con l'indipendenza nelle attività quotidiane (richiesta assistenza almeno nelle attività strumentali complesse, come pagare le bollette o gestire i farmaci)**
  - **C. I deficit cognitivi non si verificano esclusivamente nel contesto di un delirium**
  - **D. I deficit cognitivi non sono meglio spiegati da un altro disturbo mentale (es, depressione maggiore, schizofrenia)**
  - ***Specificare se AD, FTD, LBD, VaD, TBI, Substance/Med use, HIV, malattia da prioni, PD, Hungtington disease, altra condizione medica, non specificato***

Disturbo neurocognitivo minore (DSM 5 TR)

- **A. Evidenza di una modesto declino cognitivo rispetto ad un precedente livello di prestazioni in uno o più domini cognitivi (attenzione complessa, funzioni esecutive, memoria ed apprendimento, linguaggio, funzione percettivo-motoria o cognizione sociale) sulla base di :**
  - **1. preoccupazione del soggetto, di un informatore affidabile, o del clinico che vi sia stato un lieve declino delle funzioni cognitive; e**
  - **2. Modesta compromissione delle prestazioni cognitive, preferibilmente documentata da test neurospicologici standardizzati o, in loro assenza, da altra valutazione clinica quantificata**
  - **B. I deficit cognitivi non interferiscono con l'indipendenza nelle attività quotidiane (le attività strumentali complesse, come pagare le bollette o gestire i farmaci, sono conservate, ma richiedono uno sforzo maggiore, strategie compensatorie o adattamento)**
  - **C. I deficit cognitivi non si verificano esclusivamente nel contesto di un delirium**
  - **D. I deficit cognitivi non sono meglio spiegati da un altro disturbo mentale (es, depressione maggiore, schizofrenia)**
  - ***Specificare se AD, FTD, LBD, VaD, TBI, Substance/Med use, HIV, malattia da prioni, PD, Hungtington disease, altra condizione medica, non specificato***

ADL e IADL

ADL

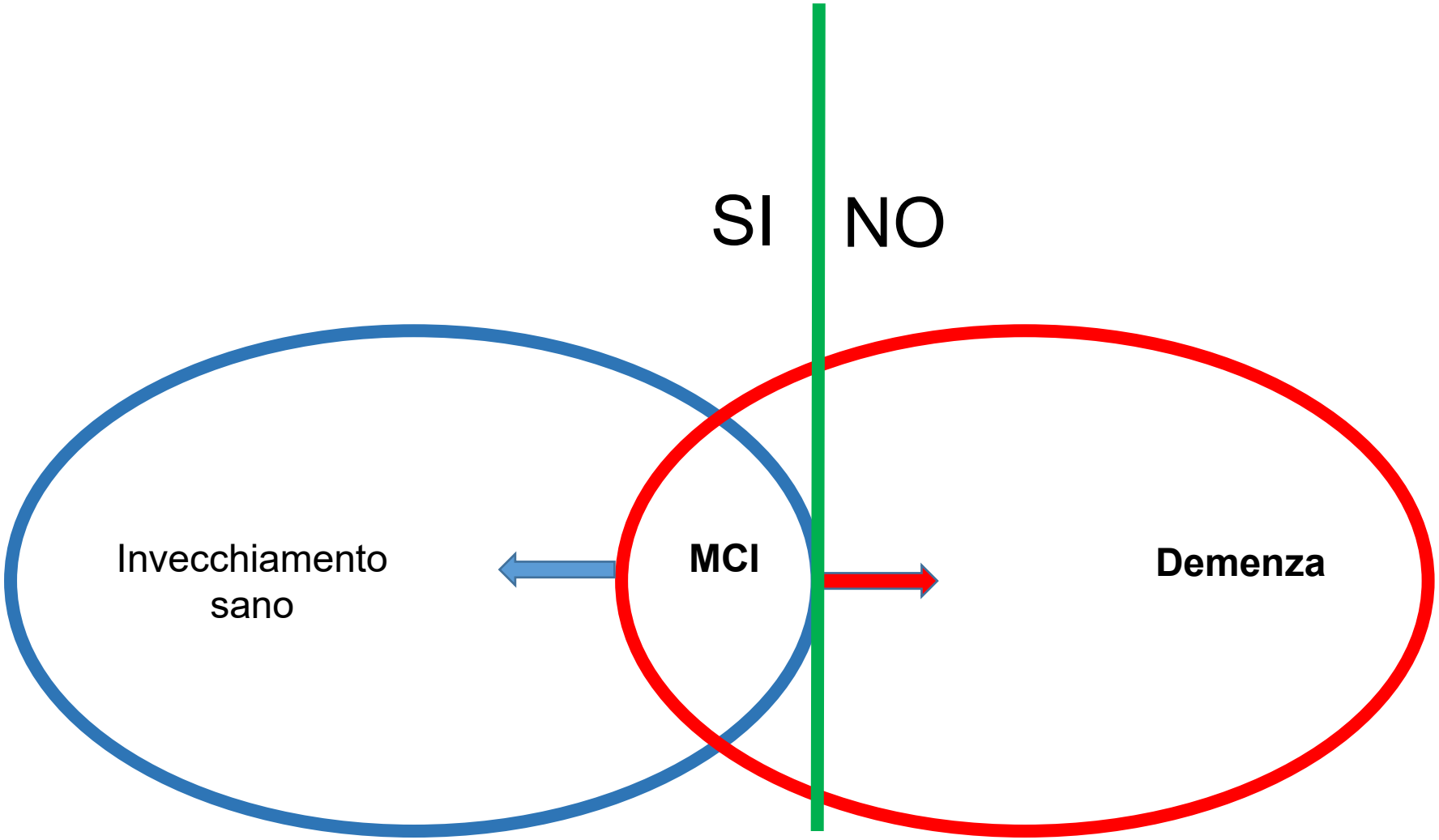
Activity of Daily Living

- Mangiare
- Spostarsi
- Uso WC
- Lavarsi
- Vestirsi
- Continenza

IADL

Instrumental Activity of Daily Living

- Usare telefono
- Fare la spesa
- Preparare i pasti
- Pulire la casa
- Lavare i panni
- Uscire
- Farmaci
- Finanze



Autonomia nelle attività della vita quotidiana ?

THE BARE ESSENTIALS

NON TUTTE LE DEMENZE SONO CAUSATE DA MALATTIA DI ALZHEIMER

Dementia

Maartje I Kester,[1] Philip Scheltens[2]

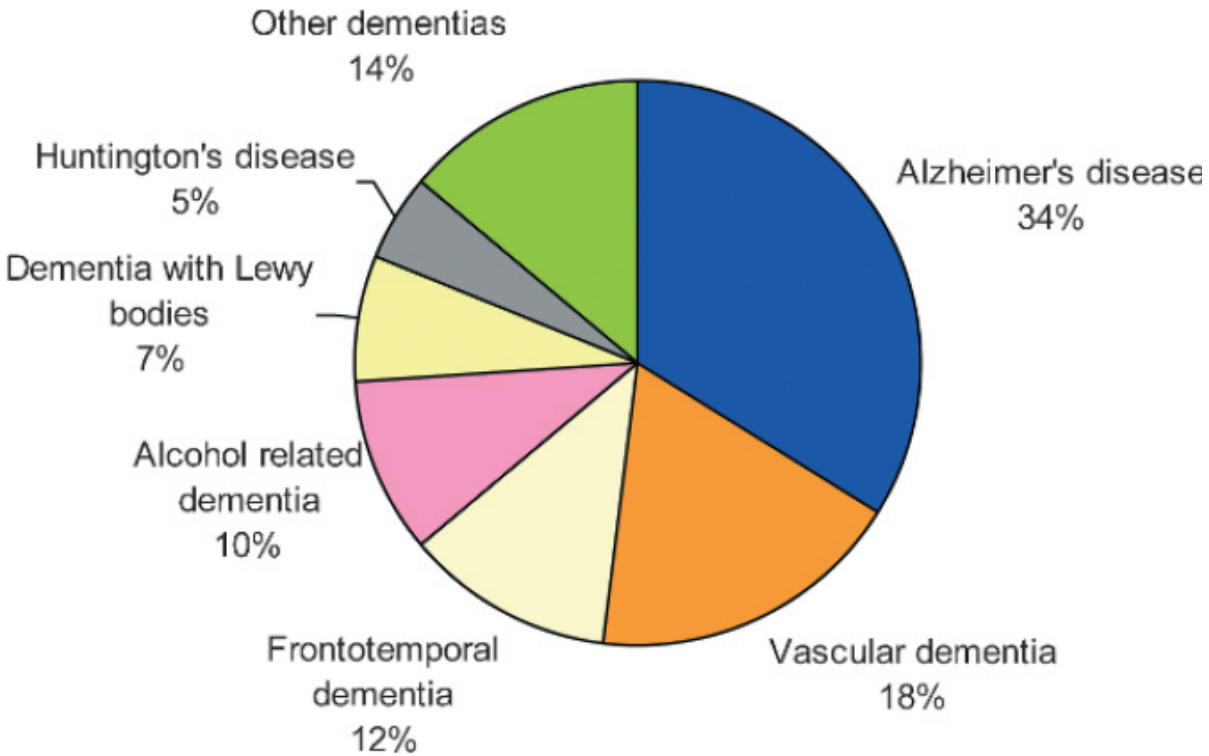


Figure 3 Causes of dementia with young onset (<65 years) (based on Harvey *et al*, see further reading).

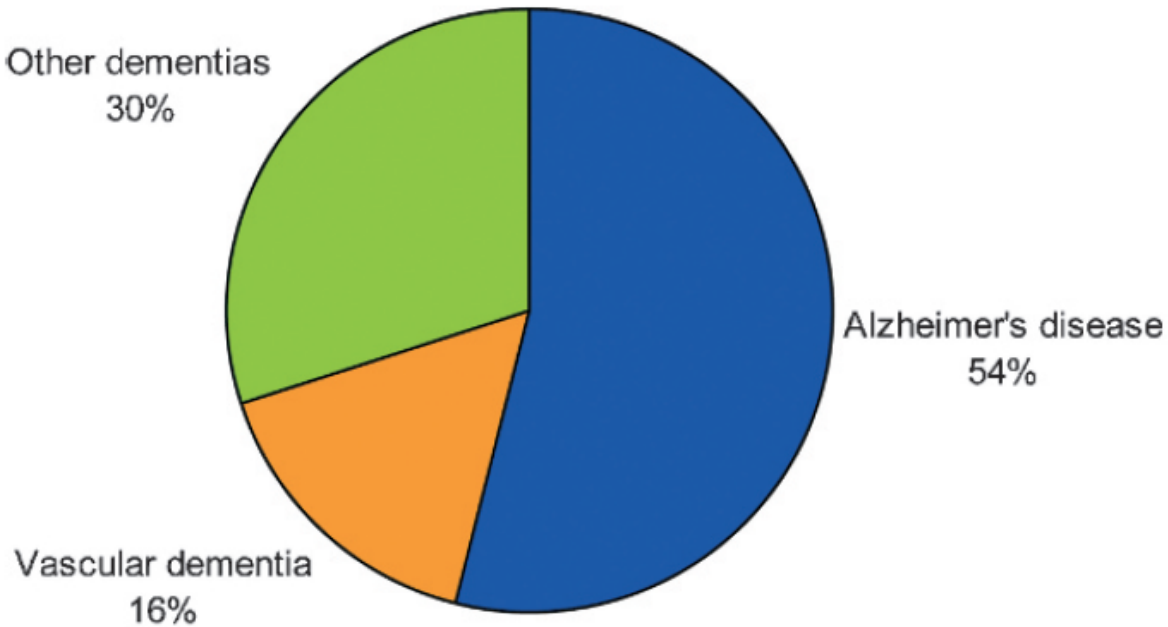
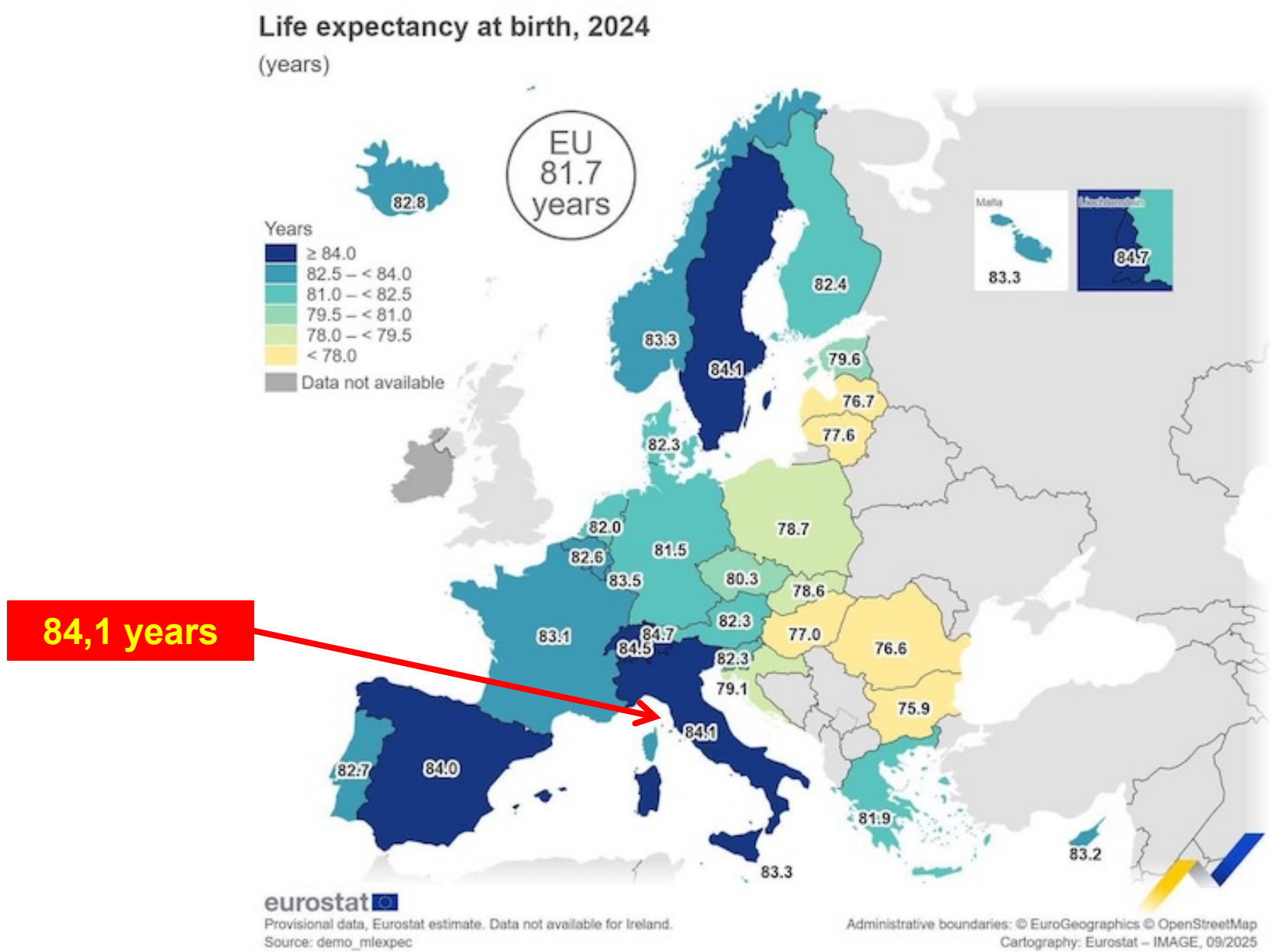


Figure 2 Causes of dementia with late onset (≥65 years) (based on Lobo *et al*, see further reading).

Epidemiologia


Life expectancy at birth, 2024
(years)
EU
81.7
years
Years
≥ 84.0
82.5 – < 84.0
81.0 – < 82.5
79.5 – < 81.0
78.0 – < 79.5
< 78.0
Data not available
82.8
83.3
84.1
82.4
79.6
76.7
77.6
82.3
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Malta
83.3
84.7
84,1 years
eurostat
Provisional data, Eurostat estimate. Data not available for Ireland.
Source: demo_mlexpec
Administrative boundaries: © EuroGeographics © OpenStreetMap
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I numeri della Liguria

Tabella 1.1 Liguria. Casi prevalenti demenza ≥65 anni (*late onset*)

	Maschi			Femmine			Totale	
	Popolazione	Tassi x 100	Casi	Popolazione	Tassi x 100	Casi	Popolazione	Casi
65-69	47.178	0,9	425	51.642	1,1	568	98.820	993
70-74	43.422	2,1	912	50.376	2,2	1.108	93.798	2.020
75-79	37.873	4,6	1.742	47.780	5,6	2.676	85.653	4.418
80-84	31.132	9,0	2.802	44.630	13,3	5.936	75.762	8.738
85-89	18.084	13,9	2.514	31.298	26,4	8.263	49.382	10.776
90+	8.634	31,2	2.694	22.775	38,9	8.859	31.409	11.553
Totale	**186.323**	**6,0**	**11.088**	**248.501**	**11,0**	**27.410**	**434.824**	**38.498**

Tabella 1.3 Liguria. Casi prevalenti Mild Cognitive Impairment ≥60 anni

	Maschi			Femmine			Totale	
	Popolazione	Tassi x 100	Casi	Popolazione	Tassi x 100	Casi	Popolazione	Casi
60-69	101.996	4,0	4.080	110.563	4,8	5.307	212.559	9.387
70-79	81.295	5,7	4.634	98.156	5,8	5.693	179.451	10.327
80-89	49.216	7,1	3.494	75.928	7,1	5.391	125.144	8.885
Totale	**232.507**	**5,3**	**12.208**	**284.647**	**5,8**	**16.391**	**517.154**	**28.599**

Tabella 1.2 Liguria. Casi prevalenti demenza 35-64 anni (*early onset*)

	Maschi			Femmine			Totale	
	Popolazione	Tassi x 100.000	Casi	Popolazione	Tassi x 100.000	Casi	Popolazione	Casi
35-39	38.160	0,0	0	36.377	4,6	2	74.537	2
40-44	40.743	3,7	2	41.072	11,1	5	81.815	6
45-49	52.598	23,5	12	53.921	10,2	5	106.519	18
50-54	61.513	38,4	24	63.098	63,2	40	124.611	63
55-59	63.663	177,1	113	66.723	152,5	102	130.386	214
60-64	54.818	285,3	156	58.921	306,7	181	113.739	337
Totale	**311.495**	**98,4**	**307**	**320.112**	**104,4**	**334**	**631.607**	**641**

67.738

Provincia di Genova	
Casi di demenza	**21.489** (di cui **345** < 65 anni)
Casi di *Mild Cognitive Impairment*	**15.546**
Totale	**37.035**
+ caregiver (x 2)	**74.070**

$$\frac{\textbf{Persone coinvolte (demenza + MCI + caregiver)}}{\textbf{Popolazione residente}} = \frac{\textbf{74.070}}{\textbf{819.320}} = \boxed{\textbf{9,0 \%}}$$

Fattori di rischio

14 Fattori di rischio per demenza

1 Sedentarietà

2 Fumo

3 Eccesso di alcool

4 Inquinamento dell'aria

5 Trauma cranico

6 Isolamento sociale

7 Bassa scolarità

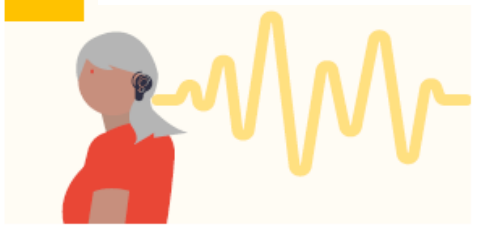
8 Obesità

9 Ipertensione

10 Diabete

11 Depressione

12 Deficit dell'udito



13 Colesterolo LDL alto

14 Deficit della vista

Source: *Dementia prevention, intervention, and care: 2024 report of the Lancet standing Commission, Livingston, Gill et al. The Lancet, Volume 404, Issue 10452, 572–628*

www.alzint.org

Alzheimer's Disease International
The global voice on dementia

14 Fattori di rischio per demenza

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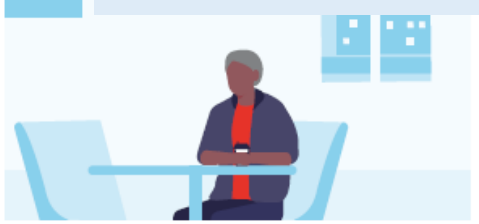


7 Bassa scolarità

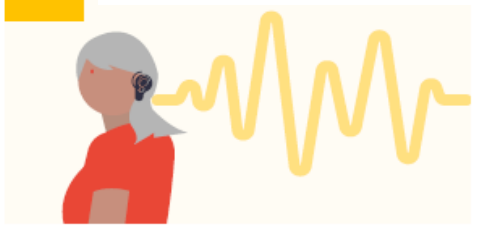
8 Obesità

9 Ipertensione

10 Diabete

11 Depressione

12 Deficit dell'udito



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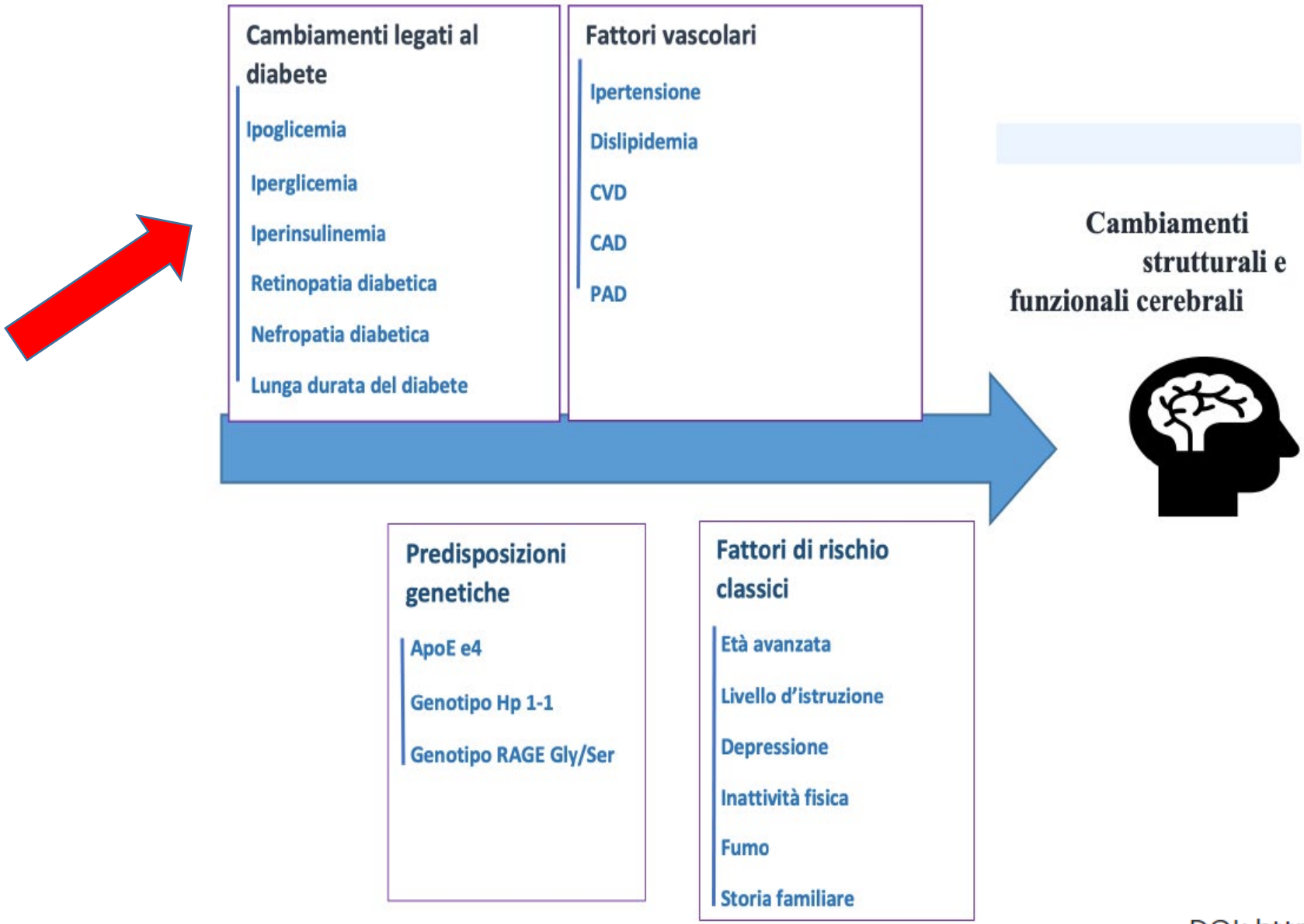
The global voice on dementia

Principali meccanismi coinvolti nel danno strutturale e funzionale cerebrale nel paziente diabetico



DOI: https://doi.org/10.30682/ildia2204b

Diabete e disturbi cognitivi

The Lancet Commissions

Dementia prevention, intervention, and care: 2024 report of the *Lancet* standing Commission

Gill Livingston, Jonathan Huntley, Kathy Y Liu, Sergi G Costafreda, Geir Selbæk, Suvarna Alladi, David Ames, Sube Banerjee, Alistair Burns, Carol Brayne, Nick C Fox, Cleusa P Ferri, Laura N Gitlin, Robert Howard, Helen C Kales, Mika Kivimäki, Eric B Larson, Noeline Nakasujja, Kenneth Rockwood, Quincy Samus, Kokoro Shirai, Archana Singh-Manoux, Lon S Schneider, Sebastian Walsh, Yao Yao, Andrew Sommerlad, Naaheed Mukadam**

Diabetes

The Lancet Commissions

Dementia prevention, intervention, and care: 2024 report of the *Lancet* standing Commission

Gill Livingston, Jonathan Huntley, Kathy Y Liu, Sergi G Costafreda, Geir Selbæk, Suvarna Alladi, David Ames, Sube Banerjee, Alistair Burns, Carol Brayne, Nick C Fox, Cleusa P Ferri, Laura N Gitlin, Robert Howard, Helen C Kales, Mika Kivimäki, Eric B Larson, Noeline Nakasujja, Kenneth Rockwood, Quincy Samus, Kokoro Shirai, Archana Singh-Manoux, Lon S Schneider, Sebastian Walsh, Yao Yao, Andrew Sommerlad*, Naaheed Mukadam*

- We previously discussed type 2 diabetes as a risk factor in late life for development of dementia. New evidence suggests that age of onset makes a difference, with midlife, but not necessarily late-life, diabetes onset increasing the risk of dementia.
    - In a prospective cohort study of 10 095 participants, the risk for dementia increased for every 5-year decrease in age of type 2 diabetes onset (HR 1·24, 95% CI 1·06–1·46), until aged over 70 years at onset.25 Diabetes should be classified as a midlife risk for dementia.
- It is unclear whether diabetes is not a risk factor for dementia at older ages or whether the absence of evidence showing significant risk is because of short follow-ups and few studies.
- WHO concludes that diabetes in late life might have a detrimental effect on brain health and dementia risk. Long illness duration and poorly controlled diabetes increase the risk of dementia.
- Our understanding of the mechanism by which diabetes increases the risk of dementia is incomplete.
    1. Long-term microvascular and macrovascular complications are well established in diabetes, and the causal mechanism likely incorporates a strong vascular component, including stroke risk.
    2. Peripheral insulin resistance leads to decreased insulin signalling in the CNS, followed by alteration in brain metabolism. Insulin resistance is a common molecular mechanism linking diabetes and Alzheimer’s disease: it leads to increased amyloid β toxicity, tau hyperphosphorylation, oxidative stress, and neuroinflammation.
    3. Increased concentrations of systemic inflammatory markers (eg, CRP) were associated with the diabetes-associated increased dementia risk.

Diabetes

The Lancet Commissions

Dementia prevention, intervention, and care: 2024 report of the *Lancet* standing Commission

- It is unclear whether effective treatment of diabetes ameliorates dementia risk per se, particularly as taking large quantities of oral medication and insulin is related to increased severity of diabetes. Strict, intensive treatment compared with standard diabetic control, however, does not decrease the risk of dementia.

- Some evidence suggests that people taking some types of anti-diabetic medication might be less at risk of dementia.
    1. A systematic review, meta-analysis, and network analysis of 27 studies (1 590 757 participants), which did not report heterogeneity, identified that cohort studies indicated that SGLT2 inhibitors (OR 0·41, 95% CI 0·22–0·76), GLP-1 receptor agonists (0·34, 0·14–0·85), and DPP-4 inhibitors (0·78, 0·61–0·99) were associated with dementia risk reduction, whereas sulfonylureas were associated with increased risk (1·43, 1·11–1·82).186 Metformin was not associated with a decreased or increased risk (0·71, 0·46–1·08). A study in UK primary care reported a significantly lower risk of dementia in 114 628 people with diabetes initiating metformin than in 95 609 people who were not on medication for their diabetes (HR 0·88, 95% CI 0·84–0·92).
    2. A meta-analysis of three RCTs and a cohort study of GLP-1 receptor agonists in people with type 2 diabetes identified a lower dementia rate in people who were randomly assigned to the drug than in those on placebo (15 820 participants; HR 0·47, 95% CI 0·25–0·86) and in people on GLP-1 receptor agonists in the nationwide Danish cohort (120 054 individuals; 0·89, 0·86–0·93).
    3. Another meta-analysis of observational studies of 819 511 people with type 2 diabetes and a mean follow-up of 4·5 years (range 1·3–7·2) reported similar findings, with less subsequent dementia in users of SLGT2 inhibitors (three studies; RR 0·62, 95% CI 0·39–0·97; I^2=82·5%), GLP-1 receptor agonists (four studies; 0·72, 0·54–0·97; I^2=91·3%), and DPP-4 inhibitors (seven studies; 0·84, 0·74–0·94; I^2=88·6%) than in people not using these medications, but the analysis reported high heterogeneity.
    4. People with diabetes might not be taking medication because their diabetes is well controlled without medication or because it is not well treated, which might account for the heterogeneity between studies. Evidence from RCTs also exists for the protective effect of GLP-1 receptor agonists.
    5. In a Taiwanese population of 31 384 propensity-matched pairs (including matching for chronic kidney disease with diabetes) who were followed up for 5 years, people who were adherent to metformin had a 72% lower risk of developing dementia than people who did not adhere.
    6. Novel study designs, such as mendelian randomisation or target trial emulation, might help to address potential confounding by indication, whereby high blood sugar leads to particular prescriptions or harms people who do not take medication.
    7. The Look AHEAD study recruited 3751 people aged 45–76 years with type 2 diabetes and overweight or obesity and randomly assigned them to a 10-year intervention of increased exercise and decreased calorie intake or diabetes support and education.192 This RCT was terminated because the interim analysis showed that the intervention had no effect on death from vascular outcomes or myocardial infarction, stroke, or severe angina. Cognitive outcome was measured at follow-up, controlling for baseline education but not cognition. There was a strong inverse relationship between HbA1c concentration and cognition over both groups. Cognitive function was not related to group allocation or to weight loss. Overall improved control of diabetes, but not very low blood sugar or weight loss without improved diabetic control, might attenuate the risk of dementia and be a way of decreasing dementia risk.

- Weight loss might also help to control diabetes and therefore might also affect cognition.

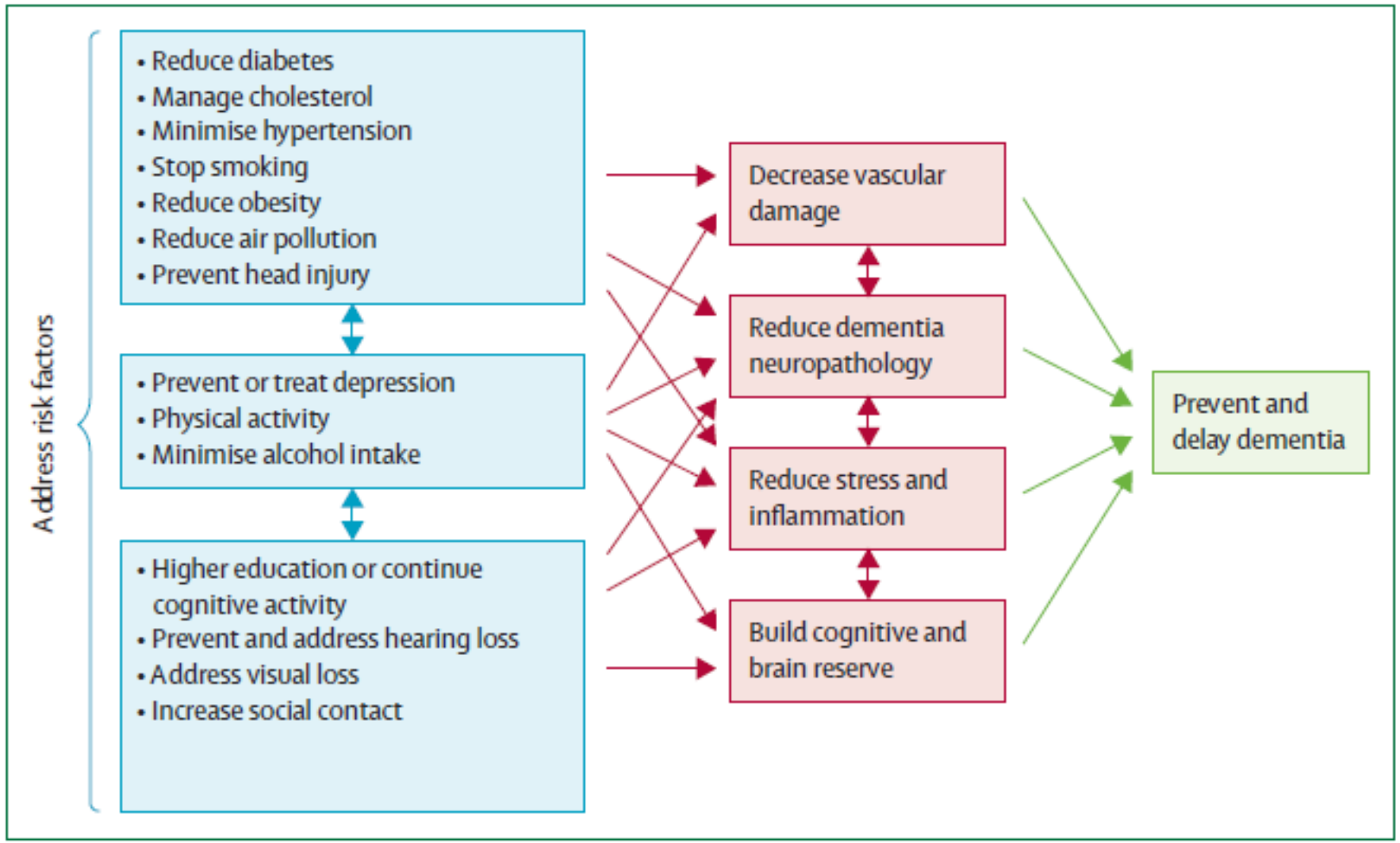


Figure 2: **Possible brain mechanisms for enhancing or maintaining cognitive reserve and risk reduction of potentially modifiable risk factors in dementia**

Ipoglicemia come fattore di rischio di demenza

Table 4. Subgroup Analyses of Hypoglycemia and Dementia Risk[a]

No. of Hypoglycemic Episodes	No. With Dementia/ No. in Group	Hazard Ratio (95% Confidence Interval)	
		Adjusted for Age (as Time Scale), BMI, Race/Ethnicity, Education, Sex, and Duration of Diabetes	Additionally Adjusted for 7-Year Mean HbA_{1c} Level, Diabetes Treatment, and Years of Insulin Use
Without stroke	890/11 633		
1		1.32 (1.01-1.73)	1.23 (1.02-1.76)
2		2.00 (1.31-3.04)	1.70 (1.11-2.60)
≥3		1.97 (1.08-3.30)	1.62 (0.98-2.71)
Without end-stage renal disease	1722/16 084		
1		1.30 (1.09-1.55)	1.30 (1.08-1.56)
2		1.70 (1.27-2.29)	1.62 (1.18-2.23)
≥3		2.04 (1.32-3.15)	1.93 (1.21-3.09)
Not African American	1617/14 780		
1		1.23 (1.04-1.46)	1.24 (1.03-1.48)
2		1.75 (1.34-2.29)	1.69 (1.26-2.26)
≥3		1.88 (1.26-2.76)	1.85 (1.23-2.80)

Abbreviations: BMI, body mass index; HbA_{1c}, glycated hemoglobin.
Analyses combined using Cox proportional hazard models.

Episodi ipoglicemici severi con ospedalizzazione,si associano ad un elevato rischio di demenza, maggiore in caso di eventi ripetuti.
Il risultato era indipendente dal controllo glicemico e dalla presenza di comprbidità

JAMA. 2009;301(15):1565-1572.

Effect of Hypoglycemia on Brain Structure in People With Type 2 Diabetes: Epidemiological Analysis of the ACCORD-MIND MRI Trial

Diabetes Care 2014;37:3279–3285 | DOI: 10.2337/dc14-0973

Compared with participants without HA, those with HA had marginally significant less atrophy (less decrease in TBV) from baseline to 40 months (29.55 [95% CI 215.21, 23.90] vs. 215.38 [95% CI 216.64, 214.12], P = 0.051), and no significant increase of AWM volume (2.06 [95% CI 1.71, 2.49] vs. 1.84 [95% CI 1.76, 1.91], P = 0.247). In addition, no unexpected local signal changes or volume loss were seen on hypoglycemic participants' brain MRI scans.

GLP-1 (glucagon-like peptide 1)

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glp-1 and alzheimer's disease

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RESULTS BY YEAR

551 results

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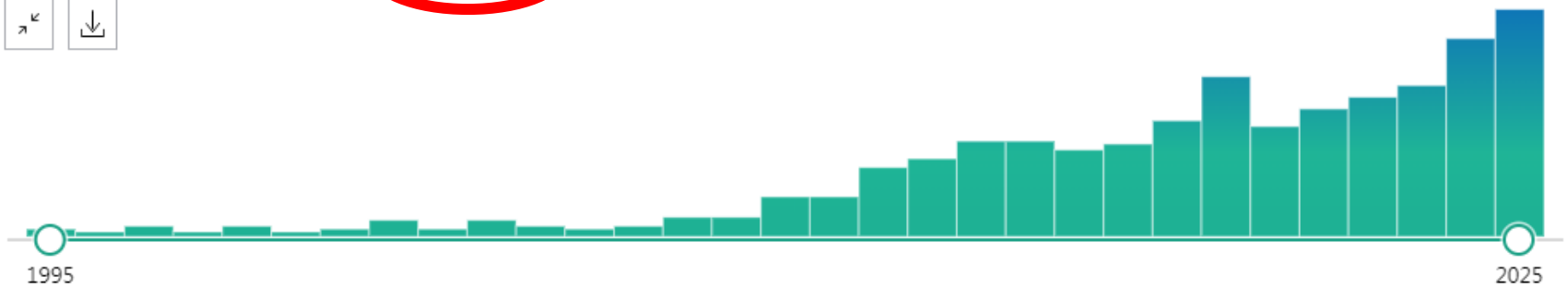

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glp-1 and dementia

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RESULTS BY YEAR

422 results

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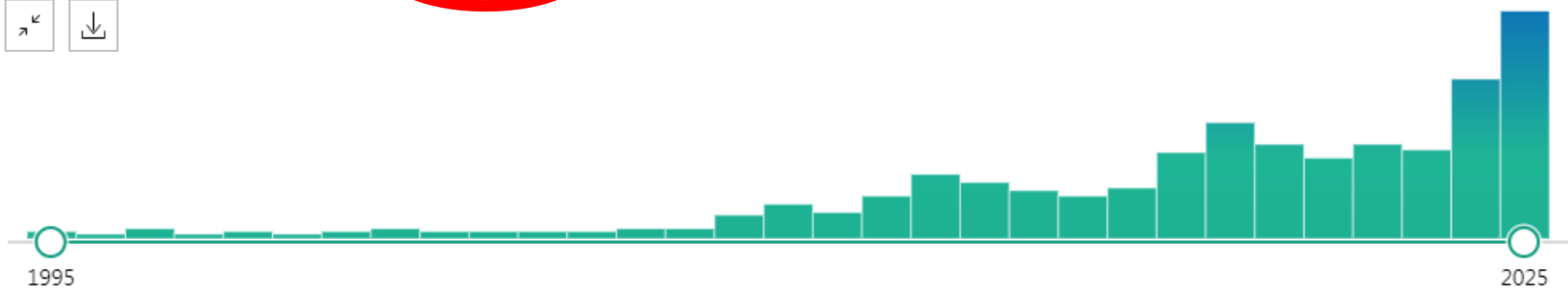

Journal of Alzheimer's Disease 4 (2002) 487–496
IOS Press

The glucagon-like peptides: a new genre in therapeutic targets for intervention in Alzheimer's disease

TracyAnn Perry* and Nigel H. Greig
Laboratory of Neuroscience, Section of Drug Design & Development, Gerontology Research Center, National Institute on Aging, National Institutes of Health, 5600 Nathan Shock Drive, Baltimore, MD 21224, USA

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EDITED BY
Jianbo Zhou,
Beijing Tongren Hospital, Capital Medical University, China

REVIEWED BY
Finbarr P. M. O'Harte,
Ulster University, United Kingdom
Venkateswarlu Kanamarlapudi,
Swansea University Medical School, United Kingdom

*CORRESPONDENCE
Jun Xu
xujun0304@163.com

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Du H, Meng X, Yao Y and Xu J (2022) The mechanism and efficacy of GLP-1 receptor agonists in the treatment of Alzheimer's disease

The mechanism and efficacy of GLP-1 receptor agonists in the treatment of Alzheimer's disease

Haiyang Du[1], Xiaoyu Meng[2,3], Yu Yao[1] and Jun Xu[1*]

[1]Division of Orthopedics, Department of Orthopedics, The Second Affiliated Hospital of Harbin Medical University, Harbin, China, [2]Division of Endocrinology, Department of Internal Medicine, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China, [3]Branch of National Clinical Research Center for Metabolic Diseases, Hubei, China

Since type 2 diabetes mellitus (T2DM) is a risk factor for Alzheimer's disease (AD) and both have the same pathogenesis (e.g., insulin resistance), drugs used to treat T2DM have been gradually found to reduce the progression of AD in AD models. Of these drugs, glucagon-like peptide 1 receptor (GLP-1R) agonists are more effective and have fewer side effects. GLP-1R agonists have reducing neuroinflammation and oxidative stress, neurotrophic effects, decreasing Aβ deposition and tau hyperphosphorylation in AD models, which may be a potential drug for the treatment of AD. However, this needs to be verified by further clinical trials. This study aims to summarize the current information on the mechanisms and effects of GLP-1R agonists in AD.

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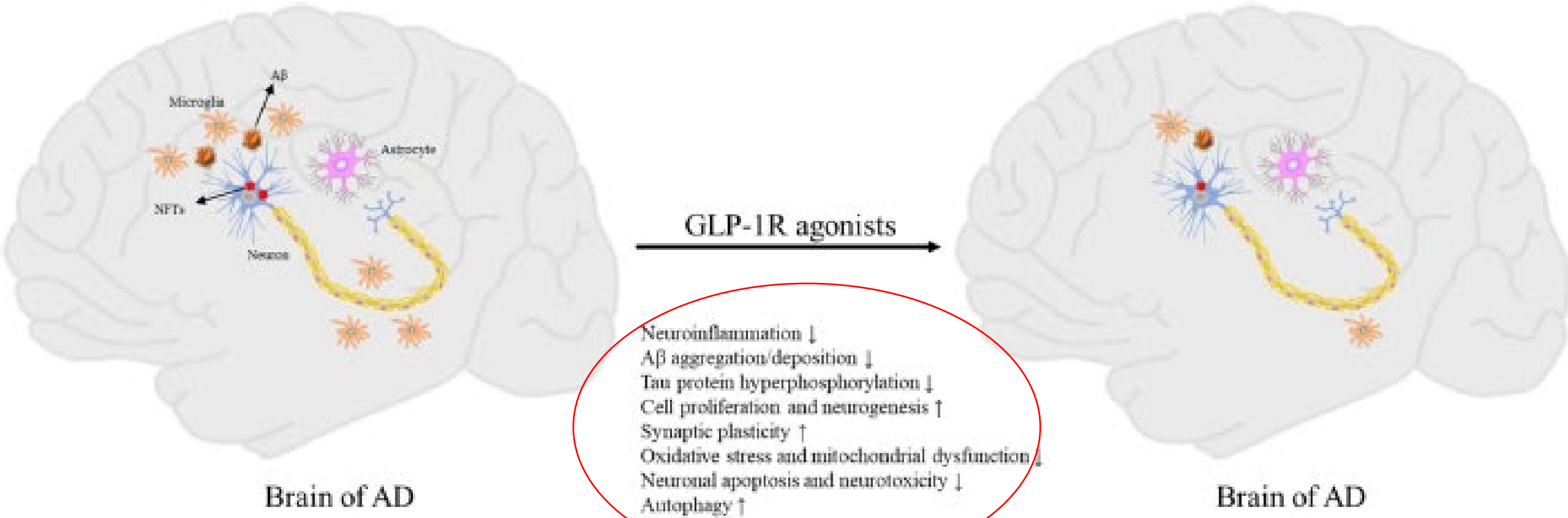
Jianbo Zhou,
Beijing Tongren Hospital, Capital

The mechanism and efficacy of GLP-1 receptor agonists in the treatment of Alzheimer's disease



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EDITED BY
Jianbo Zhou,
Beijing Tongren Hospital, Capital Medical University, China

REVIEWED BY
Finbarr P. M. O'Harte,
Ulster University, United Kingdom
Venkateswarlu Kanamarlapudi,
Swansea University Medical School, United Kingdom

*CORRESPONDENCE
Jun Xu
xujun0304@163.com

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The mechanism and efficacy of GLP-1 receptor agonists in the treatment of Alzheimer's disease

Haiyang Du[1], Xiaoyu Meng[2,3], Yu Yao[1] and Jun Xu[1*]

[1]Division of Orthopedics, Department of Orthopedics, The Second Affiliated Hospital of Harbin Medical University, Harbin, China, [2]Division of Endocrinology, Department of Internal Medicine, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China, [3]Branch of National Clinical Research Center for Metabolic Diseases, Hubei, China

Since type 2 diabetes mellitus (T2DM) is a risk factor for Alzheimer's disease (AD) and both have the same pathogenesis (e.g., insulin resistance), drugs used to treat T2DM have been gradually found to reduce the progression of AD in AD models. Of these drugs, glucagon-like peptide 1 receptor (GLP-1R) agonists are more effective and have fewer side effects. GLP-1R agonists have reducing neuroinflammation and oxidative stress, neurotrophic effects, decreasing Aβ deposition and tau hyperphosphorylation in AD models, which may be a potential drug for the treatment of AD. However, this needs to be verified by further clinical trials. This study aims to summarize the current information on the mechanisms and effects of GLP-1R agonists in AD.

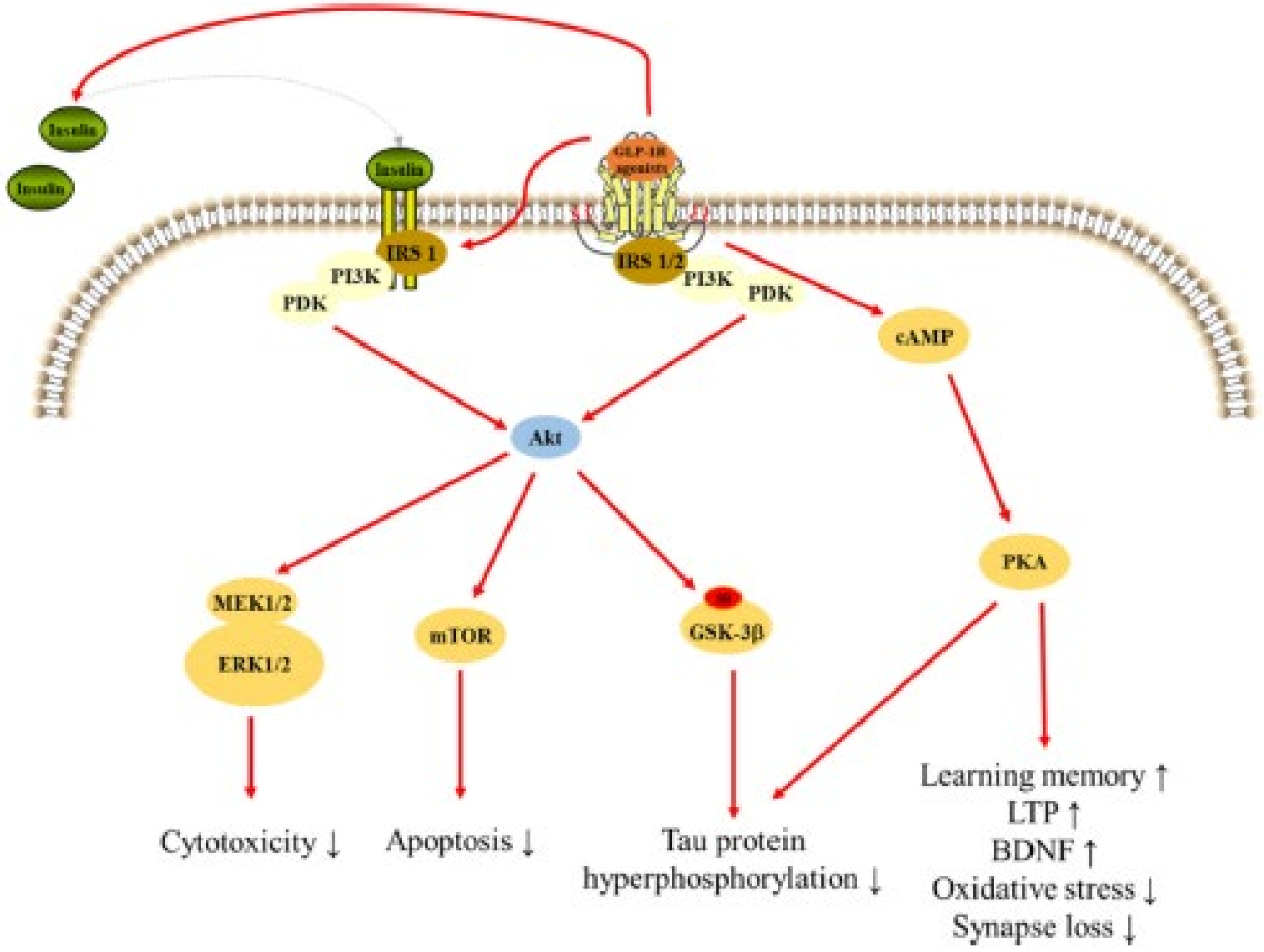
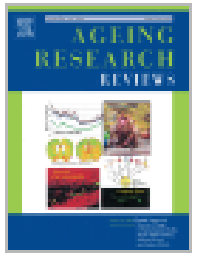


FIGURE 3
Signaling pathways for GLP-1R agonists in regulating cellular events in AD.

Ageing Research Reviews
Volume 98, July 2024, 102343



Type 2 diabetes mellitus/obesity drugs: A neurodegenerative disorders savior or a bridge too far?

Katherine O. Kopp [a], Elliot J. Glotfelty [b], Yazhou Li [a], Debomoy K. Lahiri [c d], Nigel H. Greig [a]

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https://doi.org/10.1016/j.arr.2024.102343

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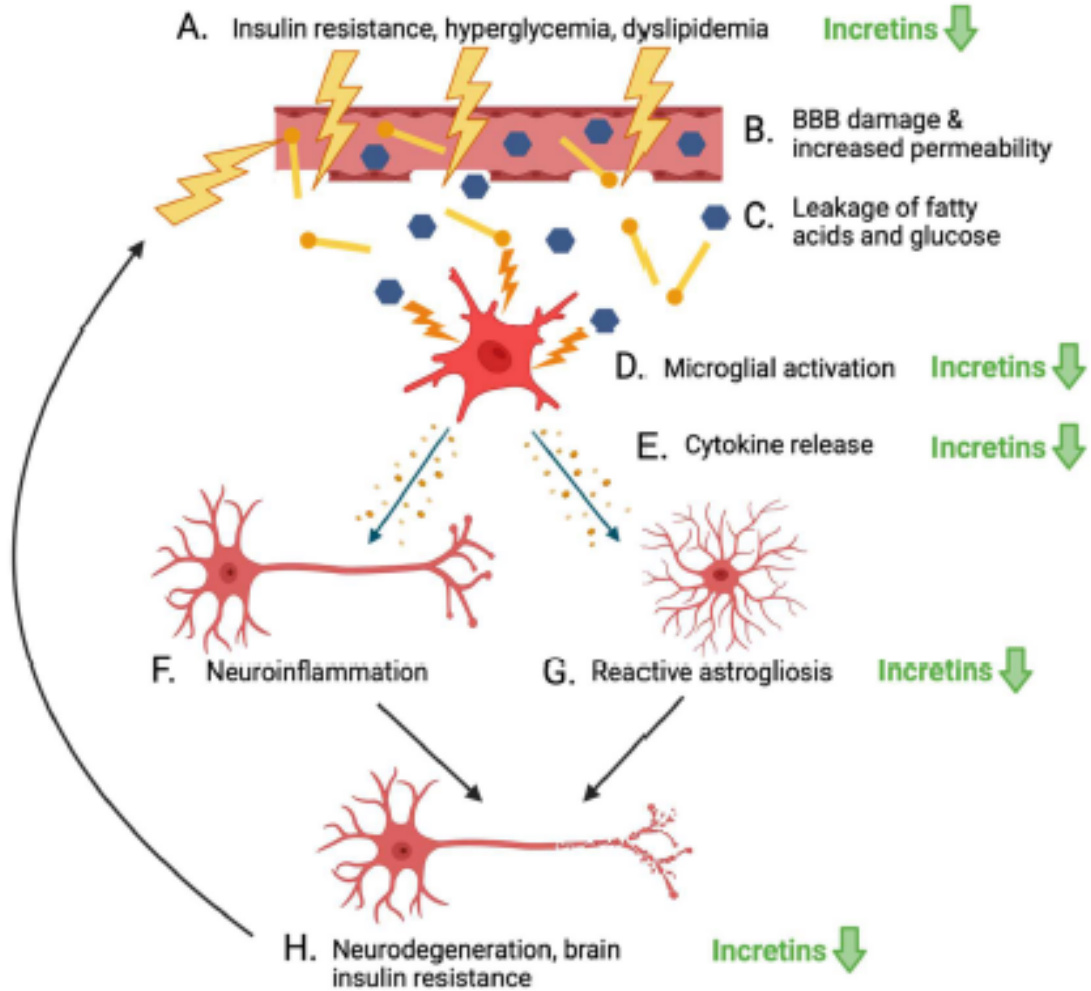

Figure 1. Hallmarks of neurodegeneration mitigated by incretins. Insulin resistance, hyperglycemia and dyslipidemia (A), often all consequences of T2DM, can damage and increase permeability of the blood-brain barrier (B). This damage results in brain infiltration with free fatty acids, excessive glucose, and other aberrant molecules (C), thus activating microglia into a pro-inflammatory phenotype (D). These pro-inflammatory microglia release a variety of proteins, including cytokines (E) that stimulate neuroinflammation (F) and reactive astrogliosis (G). Chronic neuroinflammation fuels the development of brain insulin resistance and the progression of neurodegenerative disease (H). Preclinical studies demonstrate incretin action at multiple points within this cycle of neurodegenerative disease. Figure adapted from Kopp et al., 2022 (Kopp et al., 2022).

Journal of Alzheimer's Disease Reports 8 (2024) 777–789
DOI 10.3233/ADR-230181
IOS Press

Systematic Review

Clinical Evidence for GLP-1 Receptor Agonists in Alzheimer's Disease: A Systematic Review

Yulin Liang[a], Vincent Doré[b,c], Christopher C. Rowe[c,d,e] and Natasha Krishnadas[c,e,*]

Neurodegeneration

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Review

Glucagon-like peptide-1 receptor agonists for major neurocognitive disorders

Riccardo De Giorgi,[1,2] Ana Ghenciulescu,[1] Courtney Yotter,[1] Maxime Taquet,[1] Ivan Koychev [1,3]

De Giorgi R, *et al*. *J Neurol Neurosurg Psychiatry* 2025;**96**:870–883. doi:10.1136/jnnp-2024-335593

OPEN ACCESS

Review

Glucagon-like peptide-1 receptor agonists for major neurocognitive disorders

Riccardo De Giorgi,[1,2] Ana Ghenciulescu,[1] Courtney Yotter,[1] Maxime Taquet,[1] Ivan Koychev [1,3]

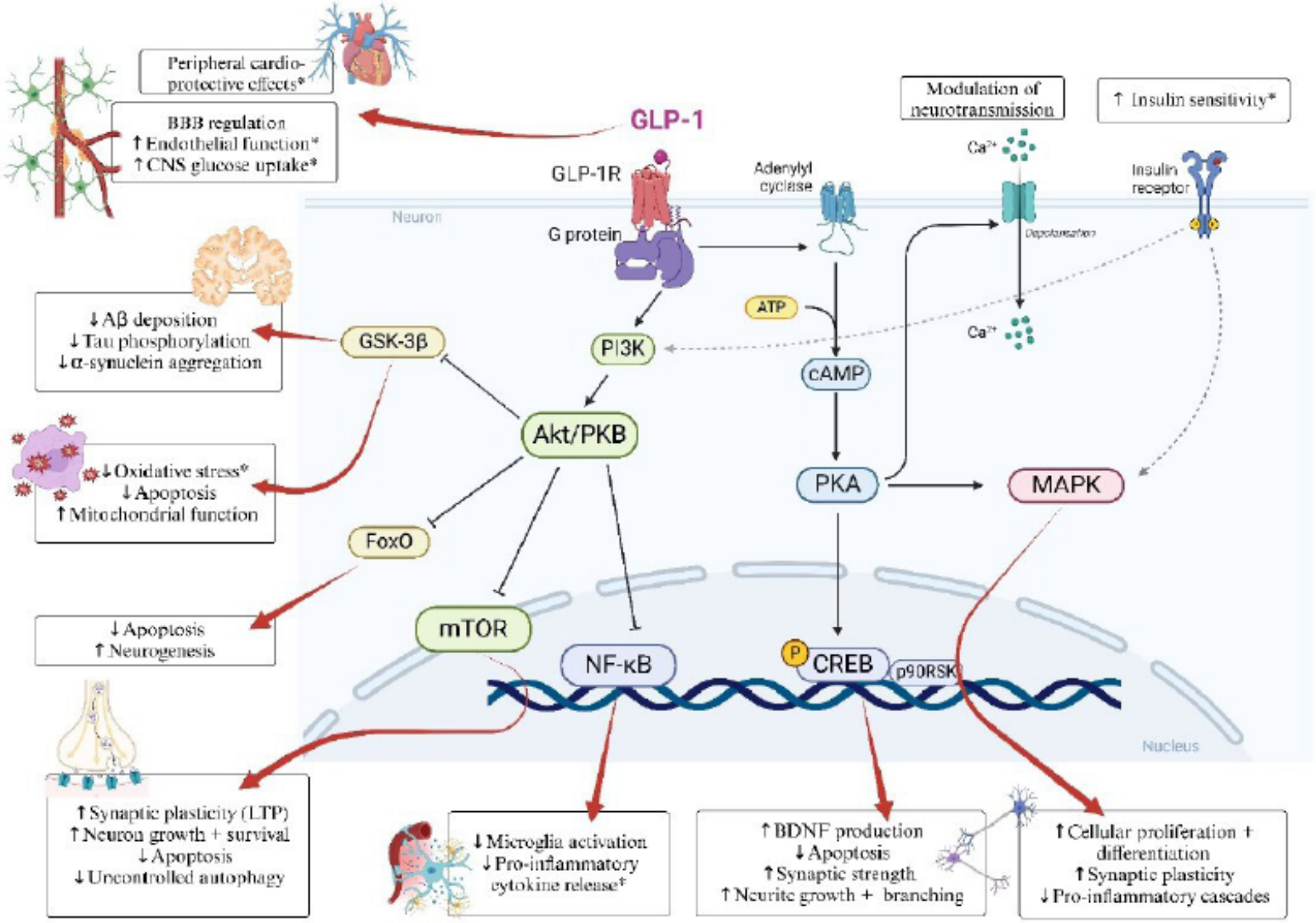

Figure 1 Molecular pathways underlying the putative neuroprotective effects of glucagon-like peptide-1 receptor agonists (GLP-1RAs). *Findings confirmed in human studies. ↑ denotes increase/enhance; ↓ denotes decrease/inhibit. Aβ, amyloid-beta; Akt/PKB, protein kinase B; BBB, blood-brain barrier; BDNF, brain-derived neurotrophic factor; cAMP, cyclic adenosine monophosphate; CNS, central nervous system; CREB, cAMP response element-binding protein; FOXO, forkhead box protein O; GSK-3β, glycogen synthase kinase 3 beta; LTP, long-term potentiation; MAPK, mitogen-associated protein kinase; mTOR, mammalian target of rapamycin; NF-κB, nuclear factor kappa B; p90RSK, p90 ribosomal kinase; PI3K, phosphoinositide 3-kinase; PKA, protein kinase

Exploring the link between GLP-1 receptor agonists and dementia: A comprehensive review

Journal of Alzheimer's Disease Reports
Volume 9: 1–8

DOI: 10.1177/25424823251342182
journals.sagepub.com/home/alr

Mallika Chuansangeam[1], Pawit Phadungsaksawasdi[2], Hyo Jin Park[3] and Yuan-Han Yang[4,5,6]

Abstract

Type 2 diabetes mellitus (T2DM) and insulin resistance are associated with an increased risk of cognitive decline and dementia, including Alzheimer's disease (AD). Glucagon-like peptide-1 receptor agonists (GLP-1RAs), originally developed for glycemic control, are emerging as neuroprotective agents. This review evaluates the evidence regarding the effects of GLP-1RAs in populations with T2DM-both with and without cognitive impairment, as well as in individuals diagnosed with AD. We conducted a comprehensive literature search and identified ten studies for inclusion: six randomized controlled trials, one prospective open-label study, and three observational studies. GLP-1RAs consistently demonstrated cognitive benefits in patients with T2DM, even in the absence of metabolic improvements. In cases of early dementia or AD, GLP-1RA treatment preserved brain metabolism and connectivity but did not significantly alter amyloid or tau biomarkers. Notably, cognitive improvements were most evident in individuals with higher body mass index (BMI) or obesity. While some studies reported neural functional changes via imaging, direct modifications in established AD biomarkers were not consistently observed. In conclusion, GLP-1RAs may improve cognitive outcomes and brain function, particularly in the early stages of neurodegeneration and among high-risk T2DM populations. Further well-designed clinical trials are needed to evaluate the impacts of GLP-1RAs on both the clinical progression and underlying pathology of dementia.

Original Article

MOLECULAR METABOLISM

GLP-1 improves the supportive ability of astrocytes to neurons by promoting aerobic glycolysis in Alzheimer's disease

Jiaping Zheng [1], Yunzhen Xie [2], Lingjia Ren [1], Liqin Qi [1], Li Wu [1,4], Xiaodong Pan [3], Jianxing Zhou [2], Zhou Chen [2,**], Libin Liu [1,*]

Journal of Alzheimer's Disease Reports 8 (2024) 777–789
DOI 10.3233/ADR-230181
IOS Press

Systematic Review

Clinical Evidence for GLP-1 Receptor Agonists in Alzheimer's Disease: A Systematic Review

Yulin Liang[a], Vincent Doré[b,c], Christopher C. Rowe[c,d,e] and Natasha Krishnadas[c,e,*]

Conclusions

In conclusion, this systematic review summarized the clinical studies on GLP-1 RA in AD and found that GLP-1 RA show no benefit in lowering/clearing Aβ plaques and tau neurofibrillary tangles deposition or improving cognition, but highlights the metabolic and neuroprotective potential for this class of drugs. Due to the limitations of the included studies, further studies with large sample sizes are needed to draw more definite conclusions.

Neuropharmacology 253 (2024) 109952

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

Glucagon-like peptide-1 class drugs show clear protective effects in Parkinson's and Alzheimer's disease clinical trials: A revolution in the making?

Christian Hölscher

Henan Academy of Innovations in Medical Science, Neurodegeneration Research Group, 451100 Xinzheng, Henan province, China

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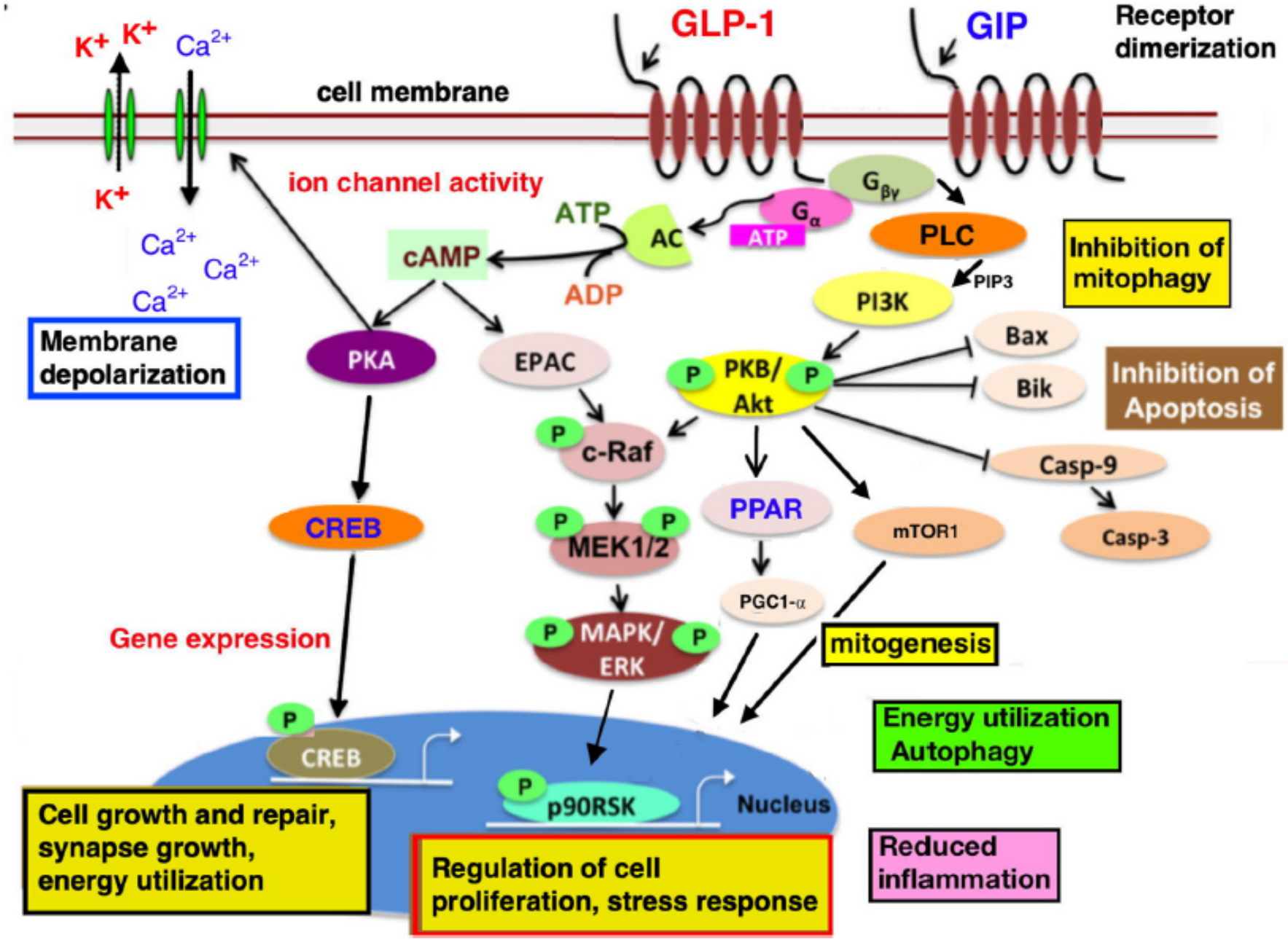
ABSTRACT

Parkinson's disease (PD) is a complex syndrome for which there is no disease-modifying treatment on the market. However, a group of drugs from the Glucagon-like peptide-1 (GLP-1) class have shown impressive improvements in clinical phase II trials. Exendin-4 (Bydureon), Liraglutide (Victoza, Saxenda) and Lixisenatide (Adlyxin), drugs that are on the market as treatments for diabetes, have shown clear effects in improving motor activity in patients with PD in phase II clinical trials. In addition, Liraglutide has shown improvement in cognition and brain shrinkage in a phase II trial in patients with Alzheimer disease (AD). Two phase III trials testing the GLP-1 drug semaglutide (Wegovy, Ozempic, Rybelsus) are ongoing. This perspective article will summarize the clinical results obtained so far in this novel research area. We are at a crossroads where GLP-1 class drugs are emerging as a new treatment strategy for PD and for AD. Newer drugs that have been designed to enter the brain easier are being developed already show improved effects in preclinical studies compared with the older GLP-1 class drugs that had been developed to treat diabetes. The future looks bright for new treatments for AD and PD.


GLP-1
GIP
Receptor dimerization
cell membrane
K+ K+ Ca2+
K+
Ca2+ Ca2+ Ca2+
ion channel activity
Membrane depolarization
ATP
AC
cAMP
ADP
Gβγ
Gα
ATP
PLC
PIP3
PI3K
Inhibition of mitophagy
PKA
EPAC
P PKB/ Akt P
Bax
Bik
Inhibition of Apoptosis
P c-Raf
Casp-9
Casp-3
CREB
P MEK1/2 P
PPAR
mTOR1
PGC1-α
Gene expression
P MAPK/ ERK P
mitogenesis
P CREB
Energy utilization Autophagy
P p90RSK
Nucleus
Cell growth and repair, synapse growth, energy utilization
Regulation of cell proliferation, stress response
Reduced inflammation

Trial fase III: RCT in pazienti con demenza lieve

frontiers in Aging Neuroscience

ORIGINAL RESEARCH
published: 24 May 2016
doi: 10.3389/fnagi.2016.00108

2016

In Alzheimer's Disease, 6-Month Treatment with GLP-1 Analog Prevents Decline of Brain Glucose Metabolism: Randomized, Placebo-Controlled, Double-Blind Clinical Trial

*Michael Gejl[1,2], Albert Gjedde[2,3], Lærke Egefjord[1,2], Arne Møller[2], Søren B. Hansen[2], Kim Vang[2], Anders Rodell[2], Hans Brændgaard[4], Hanne Gottrup[4], Anna Schacht[2], Niels Møller[5], Birgitte Brock[1,6] and Jørgen Rungby[1,7]**

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2024

Liraglutide Trial Was Negative Four Years Ago, Still Negative Today

Series - Alzheimer's Association International Conference (AAIC) 2024: Part 4 of 20: L

ARTICLE | COMMENTS | REFERENCES | FURTHER READING

09 Aug 2024

Among the hundreds of studies presented at this year's Alzheimer's Association International Conference, held July 27 to August 1 in Philadelphia, one of the few that made a splash in the news was not news at all. Perhaps spurred by a press release, media outlets reported that tantalizing results from a small trial suggested that the GLP-1 agonist liraglutide might shield the brain from dementia. The results were featured by Reuters, The Guardian, STAT News, USA Today, Forbes, CNN, and various national news channels.

ANNOTATE

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DOI: 10.1002/alz.057848

DRUG DEVELOPMENT

PODIUM PRESENTATION

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ELAD

Evaluation of liraglutide in the treatment of Alzheimer's disease

Paul Edison[1] | Grazia Daniela Femminella[1] | Craig W. Ritchie[2] | Clive Holmes[3] | Clive Ballard[37]

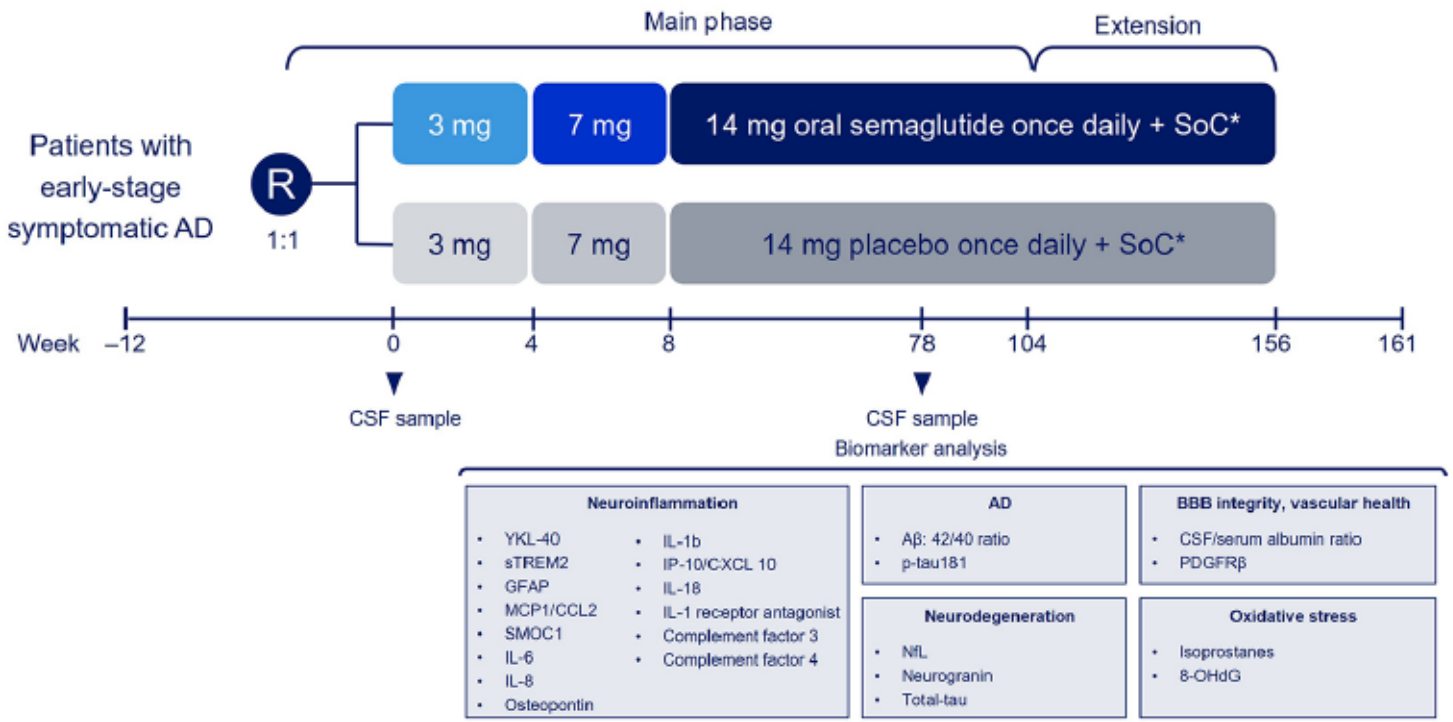
Cummings *et al. Alzheimer's Research & Therapy* (2025) 17:14
https://doi.org/10.1186/s13195-024-01666-7

Alzheimer's Research & Therapy

RESEARCH Open Access

evoke and evoke+: design of two large-scale, double-blind, placebo-controlled, phase 3 studies evaluating efficacy, safety, and tolerability of semaglutide in early-stage symptomatic Alzheimer's disease

Jeffrey L. Cummings[1,15*], Alireza Atri[2,3,4], Howard H. Feldman[5], Oskar Hansson[6,7], Mary Sano[8], Filip K. Knop[9,10,11,12], Peter Johannsen[12], Teresa León[12] and Philip Scheltens[13,14]



I farmaci antidiabetici prevengono il rischio di demenza ?

Received: 11 May 2023 | Revised: 12 September 2023 | Accepted: 29 September 2023

DOI: 10.1111/dom.15331

ORIGINAL ARTICLE

WILEY

Diabetes, antidiabetic medications and risk of dementia: A systematic umbrella review and meta-analysis

Alvin Kuate Defo BSc[1] | Veselko Bakula BSc[2] | Alessandro Pisaturo MSc[3] |
Christopher Labos MD[1] | Simon S. Wing MD[4] | Stella S. Daskalopoulou MD[1,5]

Aims:
The objective of this umbrella review and meta-analysis was to evaluate the effect of diabetes on risk of dementia, as well as the mitigating effect of antidiabetic treatments.

Conclusions:
Metformin, thiazolidinediones, pioglitazone, GLP1RAs and SGLT2is were associated with reduced risk of dementia. More longitudinal studies aimed at determining their relative benefit in different populations should be conducted.

I farmaci antidiabetici prevengono il rischio di demenza ?

Research

JAMA Neurology | Original Investigation

Cardioprotective Glucose-Lowering Agents and Dementia Risk
A Systematic Review and Meta-Analysis

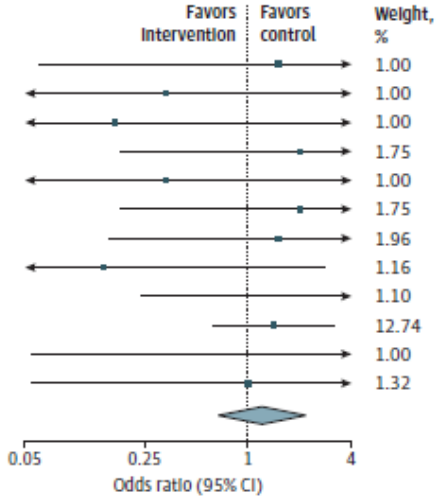
Allie Seminer, MSc; Alfredi Mulihano; Clare O'Brien, MD; Finn Krewer, PhD; Maria Costello, PhD; Conor Judge, PhD; Martin O'Donnell, PhD; Catriona Reddin, MD

JAMA Neurol. 2025;82(5):450-460. doi:10.1001/jamaneurol.2025.0360
Published online April 7, 2025.

Figure 1. Association of Glucose-Lowering Therapy With All-Cause Dementia

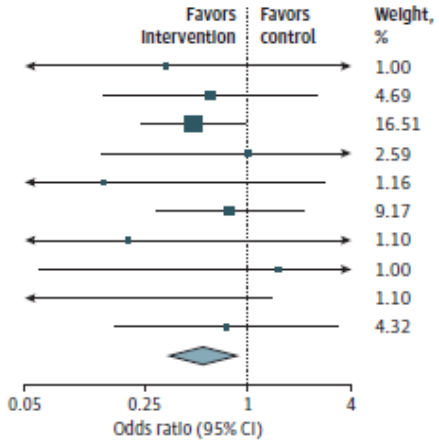
A SGLT2I

Study	Intervention event	Total	Control event	Total	ARR (95% CI)	Odds ratio (95% CI)
Zinman et al,[28] 2015	1	4687	0	2333	-0.01 (-0.09 to 0.07)	1.49 (0.06 to 36.68)
Neal et al,[29] 2017	0	2907	1	2905	0.03 (-0.06 to 0.13)	0.33 (0.01 to 8.18)
Neal et al,[29] 2017	0	2888	1	1442	0.09 (-0.09 to 0.26)	0.17 (0.01 to 4.09)
Wiviott et al,[32] 2019	2	8582	1	8578	-0.01 (-0.05 to 0.03)	2.00 (0.18 to 22.05)
McMurray et al,[31] 2019	0	2373	1	2371	0.04 (-0.07 to 0.16)	0.33 (0.01 to 8.18)
Perkovic et al,[30] 2019	2	2202	1	2199	-0.05 (-0.20 to 0.11)	2.00 (0.18 to 22.05)
Cannon et al,[35] 2020	3	5499	1	2747	-0.02 (-0.11 to 0.08)	1.50 (0.16 to 14.42)
Packer et al,[34] 2020	0	1863	3	1867	0.16 (-0.05 to 0.37)	0.14 (0.01 to 2.77)
Heerspink,[33] 2020	2	2152	0	2152	-0.09 (-0.25 to 0.06)	5.00 (0.24 to 104.30)
Anker et al,[36] 2021	14	2997	10	2991	-0.13 (-0.45 to 0.19)	1.40 (0.62 to 3.15)
Pelkert et al,[37] 2022	1	3131	0	3132	-0.03 (-0.12 to 0.06)	3.00 (0.12 to 73.72)
Herrington et al,[38] 2023	1	3304	1	3305	0.00 (-0.08 to 0.08)	1.00 (0.06 to 16.00)
RE model for subgroup: Q = 6.40; *df* = 11; *P* = .85; I^2 = 0%					-0.01 (-0.04 to 0.02)	1.20 (0.67 to 2.17)



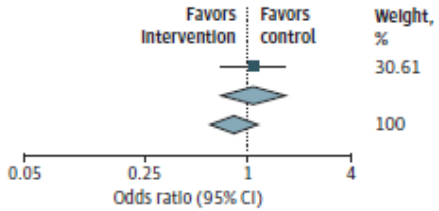
B GLP-1RA

Study	Intervention event	Total	Control event	Total	ARR (95% CI)	Odds ratio (95% CI)
Pfeffer et al,[39] 2015	0	3034	1	3034	0.03 (-0.06 to 0.12)	0.33 (0.01 to 8.18)
Marso et al,[51] 2016	3	1648	5	1649	0.12 (-0.21 to 0.46)	0.60 (0.14 to 2.51)
Marso et al,[40] 2016	12	4668	25	4672	0.28 (0.02 to 0.53)	0.48 (0.24 to 0.95)
Holman et al,[41] 2017	2	7356	2	7396	0.00 (-0.05 to 0.05)	1.01 (0.14 to 7.14)
Hernandez et al,[47] 2018	0	4731	3	4732	0.06 (-0.02 to 0.15)	0.14 (0.01 to 2.77)
Gerstein et al,[42] 2019	7	4949	9	4952	0.04 (-0.12 to 0.20)	0.78 (0.29 to 2.09)
Husain et al,[43] 2019	0	1591	2	1592	0.13 (-0.09 to 0.34)	0.20 (0.01 to 4.17)
Gerstein et al,[44] 2021	1	2717	0	1359	-0.02 (-0.15 to 0.12)	1.50 (0.06 to 36.88)
Frias et al,[45] 2022	0	304	2	102	2.26 (-0.74 to 5.27)	0.07 (0.00 to 1.39)
Lincoff et al,[48] 2023	3	8803	4	8801	0.01 (-0.05 to 0.07)	0.75 (0.17 to 3.35)
RE model for subgroup: Q = 4.72; *df* = 9; *P* = .86; I^2 = 0%					0.07 (0.02 to 0.1)	0.55 (0.35 to 0.86)



C Pioglitazone

Study	Intervention event	Total	Control event	Total	ARR (95% CI)	Odds ratio (95% CI)
TOMORROW, 2021	39	1545	46	1949	-0.16 (-1.20 to 0.87)	1.07 (0.70 to 1.65)
RE model for subgroup: Q = 0; *df* = 0; *P* = 1.00; I^2 = 0%					-0.2 (-1.0 to 0.9)	1.07 (0.70 to 1.65)
Heterogeneity: τ^2 = 0.04; χ^2 = 17.23; *P* = .75; I^2 = 6.6%					0.02 (-1.0 to 0.06)	0.83 (0.60 to 1.14)
Test for overall effect: *z* = -1.17; *P* = .24						

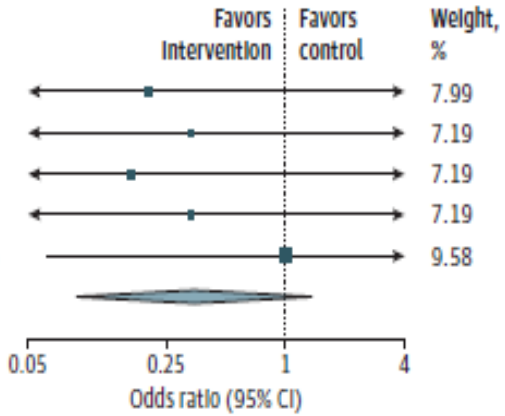


ALL-CAUSES DEMENTIA

Figure 2. Association of Glucose-Lowering Therapy With Vascular Dementia

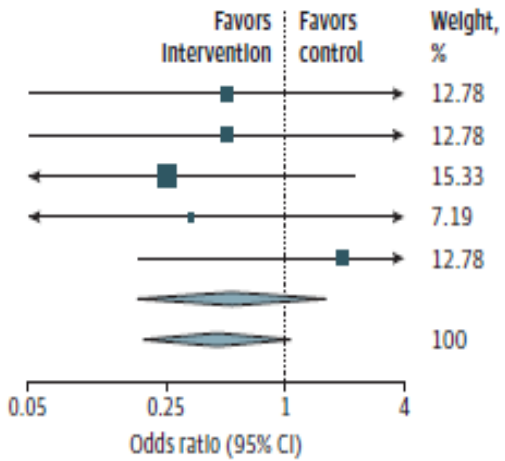
A SGLT2I

Study	Intervention event	Total	Control event	Total	ARR (95% CI)	Odds ratio (95% CI)
Wiviott et al,[32] 2019	0	8582	2	8578	0.02 (-0.02 to 0.06)	0.20 (0.01 to 4.16)
McMurray et al,[31] 2019	0	2373	1	2371	0.04 (-0.07 to 0.16)	0.33 (0.01 to 8.18)
Cannon et al,[35] 2020	0	5499	1	2747	0.05 (-0.05 to 0.14)	0.17 (0.01 to 4.09)
Packer et al,[34] 2020	0	1863	1	1867	0.05 (-0.09 to 0.20)	0.33 (0.01 to 8.20)
Anker et al,[36] 2021	1	2997	1	2991	0.00 (-0.09 to 0.09)	1.00 (0.06 to 15.96)
RE model for subgroup: Q = 0.89; df = 4; P = .93; I^2 = 0%					0.03 (-0.0004 to 0.06)	0.35 (0.09 to 1.36)



B GLP-1RA

Study	Intervention event	Total	Control event	Total	ARR (95% CI)	Odds ratio (95% CI)
Marso et al,[40] 2016	1	4668	2	4672	0.02 (-0.05 to 0.09)	0.50 (0.05 to 5.52)
Holman et al,[41] 2017	1	7356	2	7396	0.01 (-0.03 to 0.06)	0.50 (0.05 to 5.54)
Gerstein et al,[42] 2019	1	4949	4	4952	0.06 (-0.03 to 0.15)	0.25 (0.03 to 2.24)
Husain et al,[43] 2019	0	1591	1	1592	0.06 (-0.11 to 0.24)	0.33 (0.01 to 8.19)
Lincoff et al,[48] 2023	2	8803	1	8801	-0.01 (-0.05 to 0.03)	2.00 (0.18 to 22.06)
RE model for subgroup: Q = 1.71; df = 4; P = .79; I^2 = 0%					0.02 (-0.009 to 0.05)	0.54 (0.18 to 1.61)
Heterogeneity: τ^2 = 0; χ^2 = 2.84; P = .97; I^2 = 0%						
Test for overall effect: z = -1.82; P = .07						

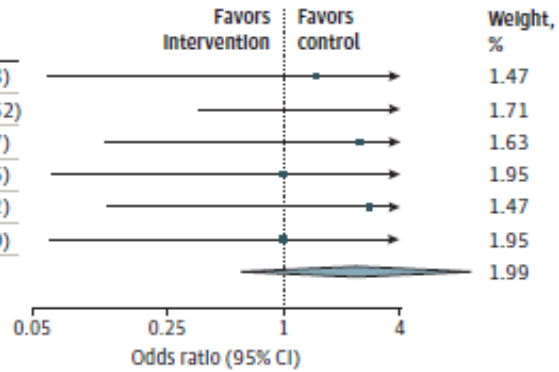


VASCULAR DEMENTIA

Figure 3. Association of Glucose-Lowering Therapy With Alzheimer Dementia

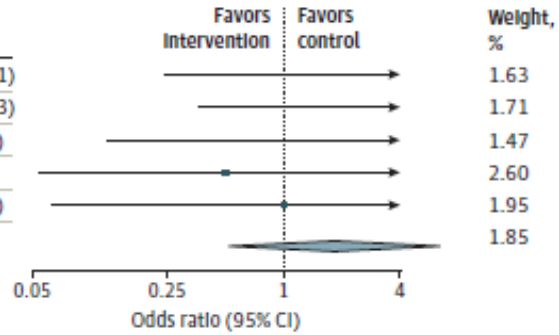
A SGLT2I

Study	Intervention event	Total	Control event	Total	ARR (95% CI)	Odds ratio (95% CI)
Zinman et al,[28] 2015	1	4687	0	2333	-0.01 (-0.09 to 0.07)	1.49 (0.06 to 36.68)
Wiviott et al,[32] 2019	3	8582	0	8578	-0.03 (-0.08 to 0.01)	7.00 (0.36 to 135.52)
Cannon et al,[35] 2020	2	5499	0	2747	-0.03 (-0.10 to 0.05)	2.50 (0.12 to 52.07)
Anker et al,[36] 2021	1	2997	1	2991	0.00 (-0.09 to 0.09)	1.00 (0.06 to 15.96)
Pelkert et al,[37] 2022	1	3131	0	3132	-0.03 (-0.12 to 0.06)	3.00 (0.12 to 73.72)
Herrington et al,[38] 2023	1	3304	1	3305	0.00 (-0.08 to 0.08)	1.00 (0.06 to 16.00)
RE model for subgroup: Q = 1.28; df = 5; P = .94; I^2 = 0%					-0.02 (-0.05 to 0.00011)	1.99 (0.59 to 6.71)



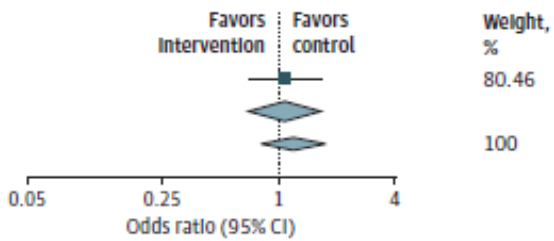
B GLP-1RA

Study	Intervention event	Total	Control event	Total	ARR (95% CI)	Odds ratio (95% CI)
Marso et al,[40] 2016	2	4668	0	4672	-0.04 (-0.12 to 0.03)	5.01 (0.24 to 104.31)
Holman et al,[41] 2017	3	7356	0	7396	-0.04 (-0.09 to 0.01)	7.04 (0.36 to 136.33)
Hernandez et al,[47] 2018	1	4731	0	4732	-0.02 (-0.08 to 0.04)	3.00 (0.12 to 73.69)
Gerstein et al,[42] 2019	1	4949	2	4952	0.02 (-0.05 to 0.09)	0.50 (0.05 to 5.52)
Lincoff et al,[48] 2023	1	8803	1	8801	0.00 (-0.03 to 0.03)	1.00 (0.06 to 15.99)
RE model for subgroup: Q = 2.61; df = 4; P = .62; I^2 = 0%					-0.02 (-0.04 to 0.005)	1.85 (0.52 to 6.57)



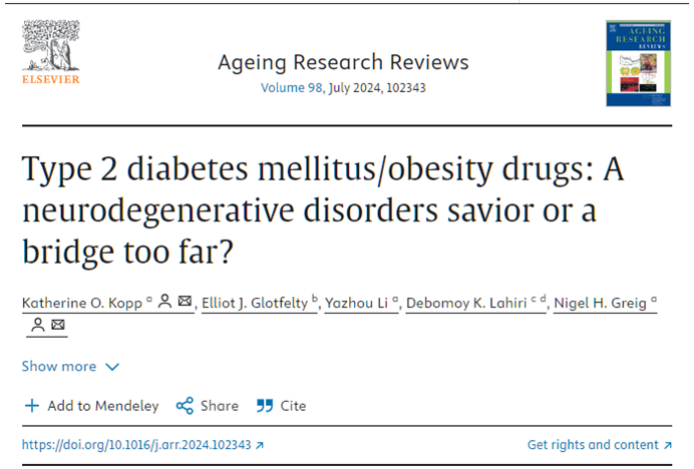
C Pioglitazone

Study	Intervention event	Total	Control event	Total	ARR (95% CI)	Odds ratio (95% CI)
TOMORROW, 2021	39	1545	46	1949	-0.16 (-1.20 to 0.87)	1.07 (0.70 to 1.65)
RE model for subgroup: Q = 0; df = 0; P = 1.00; I^2 = 0%					-0.2 (-1.0 to 0.9)	1.07 (0.70 to 1.65)
Heterogeneity: τ^2 = 0; χ^2 = 5.28; P = .92; I^2 = 0%					-0.02 (-0.06 to 0.01)	1.20 (0.82 to 1.77)
Test for overall effect: z = .93; P = .35						



ALZHEIMER DEMENTIA

CONCLUSIONS AND RELEVANCE While cardioprotective glucose-lowering therapies were not associated with an overall reduction in all-cause dementia, this meta-analysis of randomized clinical trials found that glucose lowering with GLP-1RAs was associated with a statistically significant reduction in all-cause dementia.



Ageing Research Reviews
Volume 98, July 2024, 102343

Type 2 diabetes mellitus/obesity drugs: A neurodegenerative disorders savior or a bridge too far?

Katherine O. Kopp [c], Elliot J. Glotfelty [b], Yazhou Li [a], Debomoy K. Lahiri [c d], Nigel H. Greig [a]

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https://doi.org/10.1016/j.arr.2024.102343 Get rights and content

An increasing preclinical, clinical, and epidemiological literature suggests that select incretin mimetics may provide an effective treatment strategy, but ‘which ones’ for ‘which disorders’ and ‘when’ remain key open questions.

Gestione delle persone con demenza/*Mild Cognitive Impairment* e patologie fisiche croniche coesistenti

69	In persone con demenza o *Mild Cognitive Impairment* e almeno una comorbilità fisica cronica, per il trattamento delle condizioni in comorbilità (per esempio ipertensione, malattie cardiovascolari, diabete di tipo 2, deficit sensoriali, disturbi urinari) si fa riferimento al trattamento standard di ciascuna condizione, tenendo in considerazione la situazione clinica della singola persona e fatta eccezione nei casi in cui la somministrazione del trattamento standard determini un rischio e/o danno superiore al beneficio atteso (vedere la Tabella 6 a p. 105).	FORTE POSITIVA ADATTATA

Riposizionamento di trattamenti farmacologici

86	Non offrire i seguenti trattamenti allo specifico scopo di rallentare la progressione della demenza di Alzheimer o di rallentare o impedire la conversione da *Mild Cognitive Impairment* a demenza: · farmaci antidiabetici; · farmaci antipertensivi; · statine; · farmaci antinfiammatori non steroidei (FANS), incluso l'acido acetilsalicilico.	FORTE NEGATIVA

Che fare ?

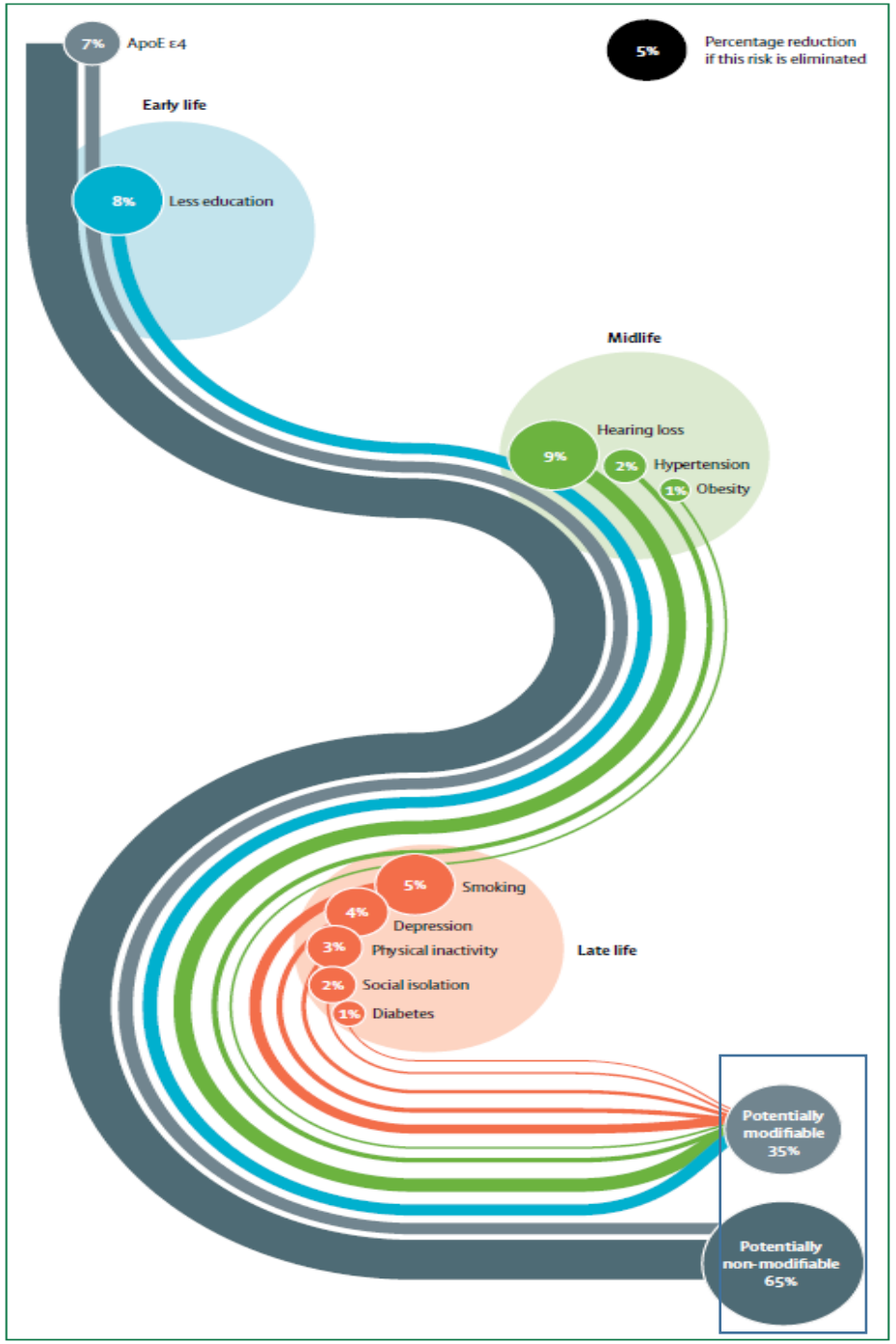
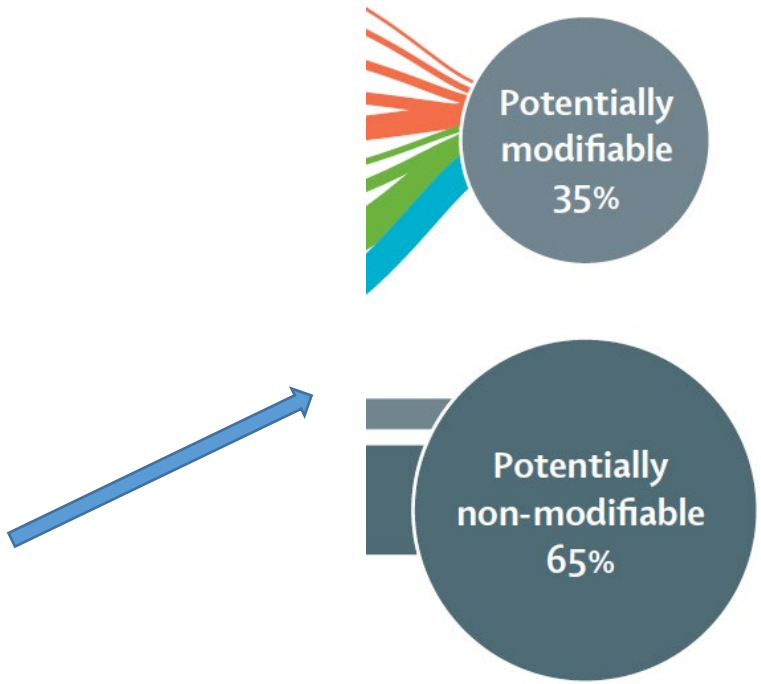


Figure 4: **Life-course model of contribution of modifiable risk factors to dementia**
Numbers are rounded to nearest integer. Figure shows potentially modifiable or non-modifiable risk factors.

The Lancet Commissions

Dementia prevention, intervention, and care

Gill Livingston, Andrew Sommerlad, Vasiliki Orgeta, Sergi G Costafreda, Jonathan Huntley, David Ames, Clive Ballard, Sube Banerjee, Alistair Burns, Jiska Cohen-Mansfield, Claudia Cooper, Nick Fox, Laura N Gitlin, Robert Howard, Helen C Kales, Eric B Larson, Karen Ritchie, Kenneth Rockwood, Elizabeth L Sampson, Quincy Samus, Lon S Schneider, Geir Selbæk, Linda Teri, Naaheed Mukadam



2017

www.thelancet.com Published online July 20, 2017 http://dx.doi.org/10.1016/S0140-6736(17)31363-6

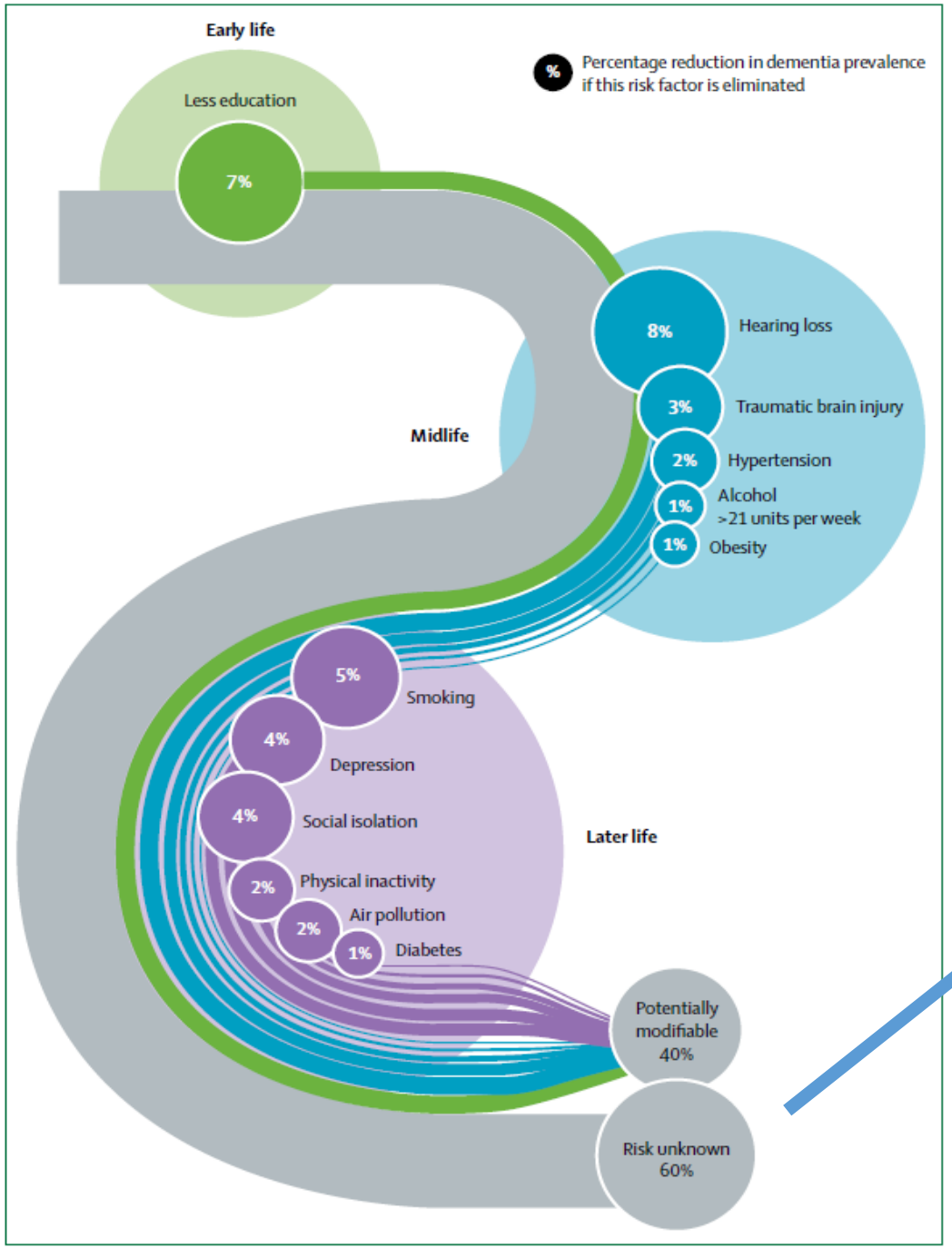
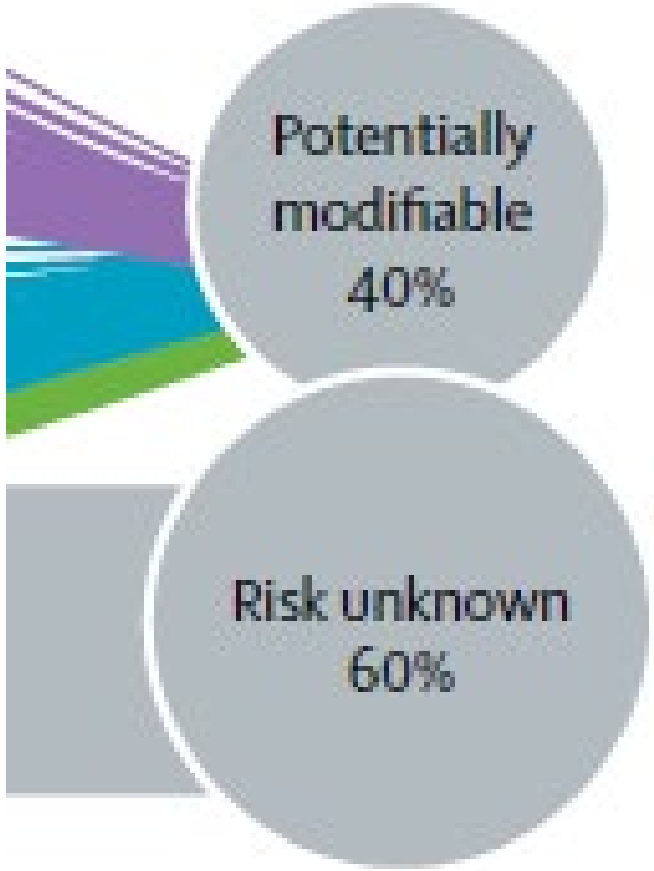
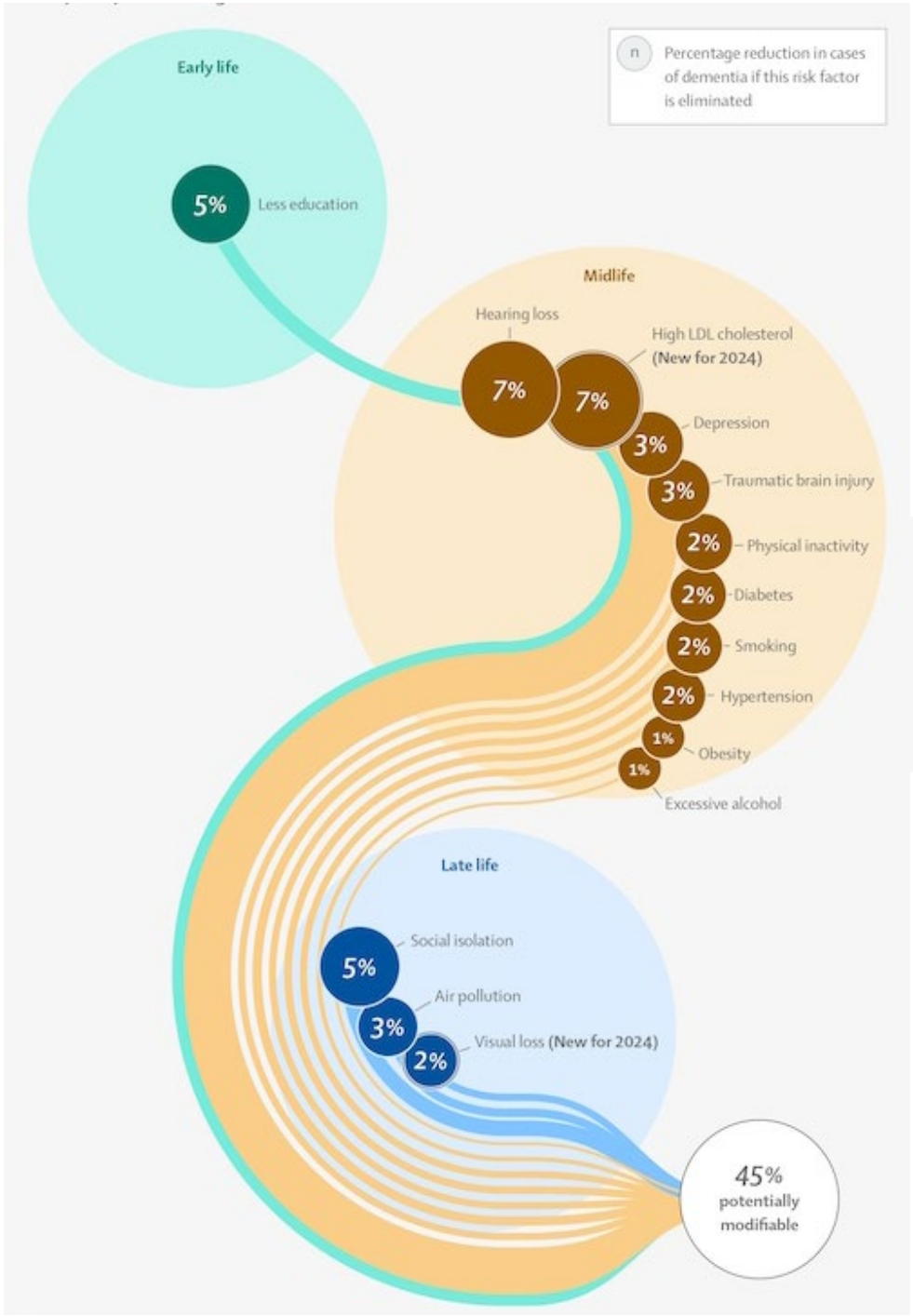


Figure 7: **Population attributable fraction of potentially modifiable risk factors for dementia**

Dementia prevention, intervention, and care: 2020 report of the *Lancet* Commission



2020



2024

Risk factors for dementia — 2024 update

The 2024 update to the standing Lancet Commission on dementia prevention, intervention, and care adds two new risk factors (high LDL cholesterol and vision loss) and indicates that nearly half of all dementia cases worldwide could be prevented or delayed by addressing 14 modifiable risk factors.

Livingston G, Huntley J, Liu KY, et al. Dementia prevention, intervention, and care: 2024 report of the Lancet standing Commission. *The Lancet* 2024; published online July 31. https://doi.org/10.1016/S0140-6736(24)01296-0.

THE LANCET

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JAMA Network Open

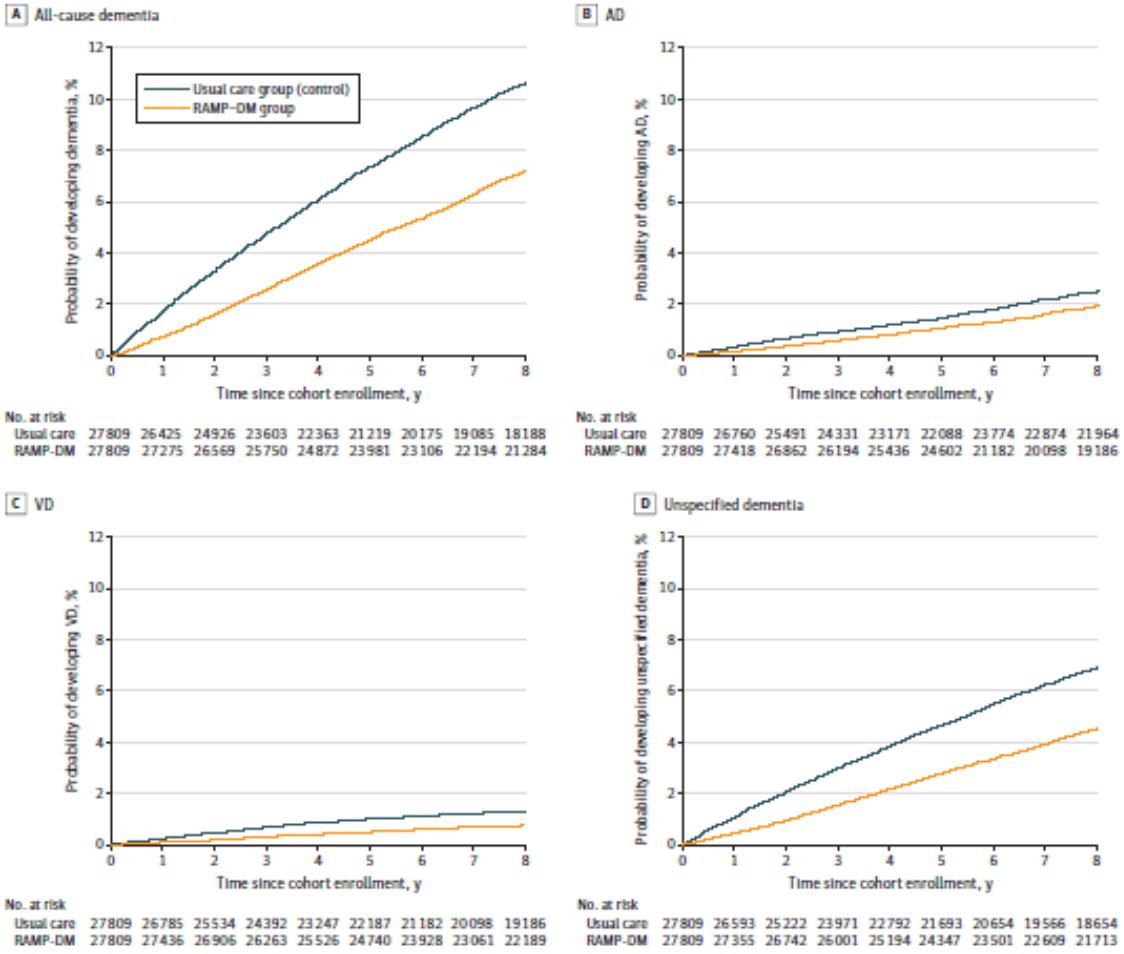
Original Investigation | Public Health

JAMA Network Open. 2024;7(2):e2355733. doi:10.1001/jamanetworkopen.2023.55733

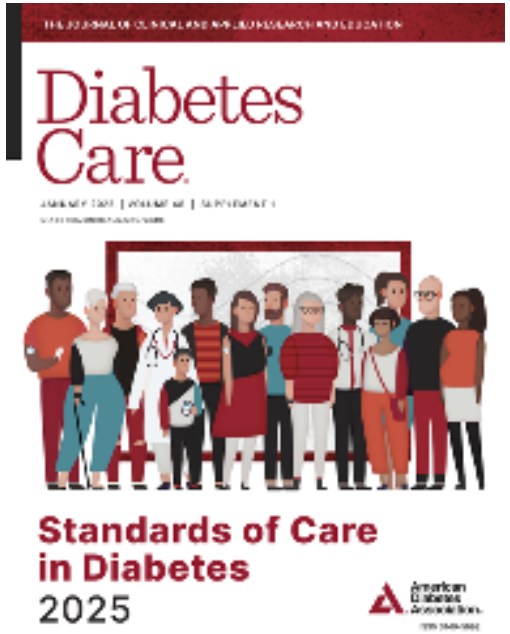
Risk of Dementia Among Patients With Diabetes in a Multidisciplinary, Primary Care Management Program

Kailu Wang, PhD; Shi Zhao, PhD; Eric Kam-Pui Lee, MBBS, MSc; Susan Zi-May Yau, MBChB; Yushan Wu, PhD; Chi-Tim Hung, MBBS, MSc; Eng-Kiong Yeoh, MBBS

Figure. Time to Dementia Incidence in RAMP-DM vs Usual Care Group



AD indicates Alzheimer disease; RAMP-DM, Risk Assessment and Management Program-Diabetes Mellitus; VD, vascular dementia.



13. Older Adults: Standards of Care in Diabetes—2025

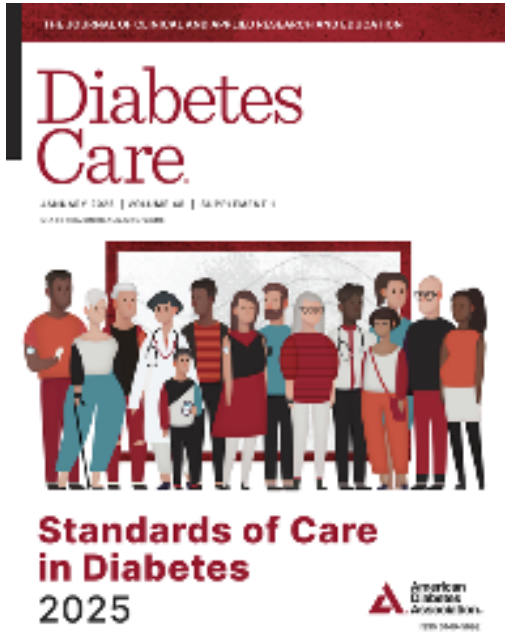
*American Diabetes Association Professional Practice Committee**

Diabetes Care 2025;48(Suppl. 1):S266–S282 | https://doi.org/10.2337/dc25-S013

Recommendations

13.1 Assess the medical, psychological, functional (self-management abilities), and social domains in older adults with diabetes to provide a framework to determine goals and therapeutic approaches for diabetes management. **B**

13.2 Screen at least annually for geriatric syndromes (e.g., cognitive impairment, depression, urinary incontinence, falls, persistent pain, and frailty), hypoglycemia, and polypharmacy in older adults with diabetes, as they may affect diabetes management and diminish quality of life. **B**



NEUROCOGNITIVE FUNCTION

Recommendation

13.3 Screening for early detection of mild cognitive impairment or dementia should be performed for adults 65 years of age or older at the initial visit, annually, and as appropriate. **B**

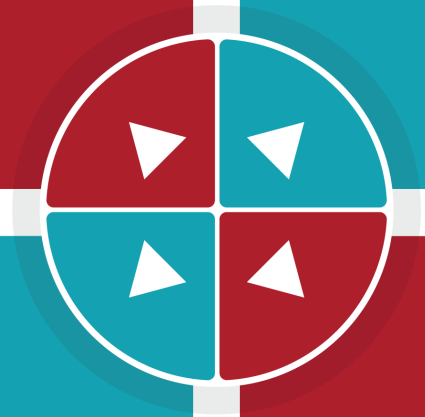
Using the 4Ms Framework of Age-Friendly Health Systems to Address Person-Specific Issues That Can Affect Diabetes Management

MENTATION

- Self-administration of medications
- Ability to use diabetes technology
- Anxiety, depression, and diabetes distress
- Mild cognitive impairment or dementia
- Coping skills and self-care

MEDICATIONS

- Treatment burden
- Affordability or insurance coverage
- End-organ disease or complications affecting medication choice
- Polypharmacy
- History of adverse medication effects
- Social and family support
- Risk of hypoglycemia, hypoglycemia unawareness, and fear of hypoglycemia



MOBILITY

- Foot complications
- Functional ability
- Frailty and sarcopenia
- Leg weakness
- Neuropathy
- Vision and hearing impairment

WHAT MATTERS MOST

- Discussing goals and expectations
- Symptom and disease burden
- Meal and treatment preferences (e.g., injections and glucose monitoring)
- Risks, burdens, and benefits of treatment
- Loneliness, social isolation, and overall quality of life
- Life expectancy

Se c’è un dubbio….

- Lamenta problemi di memoria ?
- Non ricorda la terapia ?
- Non fa esami prescritti ?
- E’ solo ?
- Non appare «compliante» con la terapia ?
- Non appare congruo nei discorsi ?
- E’ trasandato/trascurato ?
- E’ ripetititvo ?

Quale strumento utilizzare ?

GPCOG – Test Di Screening Per La Demenza Per Medici Di Medicina Generale

Versione Italiana Trial (di Dr. Pirani Alessandro autorizzato dagli autori)

Brodaty H., Pond D., Kemp N. M., Luscombe G., Harding L., Berman K., Huppert F. A.. JAGS, 50: 530-534; 2002

A) Valutazione Del Paziente *Ogni domanda deve essere formulata una volta sola.*

Richiamo 1° fase **Nominativo ed indirizzo da richiedere nella 2°f ase**

1. *"Le dirò un nome ed un indirizzo. Dopo che li avrò detti, desidero che lei li ripeta. Ricordi questo nome ed indirizzo perché le chiederò di ripetermeli fra qualche minuto.* (Consentite un massimo di 4 tentativi ma non date punteggio)

"Mario Rossi, Via Libertà 42, Pavia"

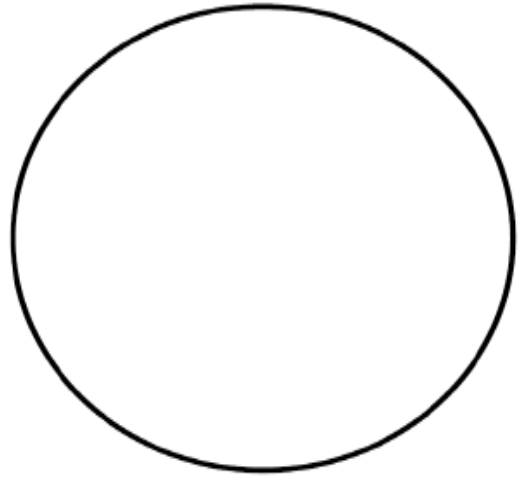
		Corretto	Sbagliato
Orientamento Temporale			
2. Mi dica la data di oggi?	(si accetta solo data esatta)	1	0
Funzionalità Visuospaziale - Disegno dell'Orologio (usare un cerchio prestampato)			
3.Inserisca i numeri delle ore (devono essere posizionati correttamente)		1	0
4. Per piacere disegni le lancette in modo che segnino le ore 11:10		1	0
Informazione 5. Può raccontarmi una notizia importante appresa da TV o giornali?			
_____		1	0
Richiamo 2° fase 6. Mi può ripetere il nome e l'indirizzo che le avevo chiesto di ricordare?			
	Mario	1	0
	Rossi	1	0
	Via Libertà	1	0
	42	1	0
	Pavia	1	0
Punteggio. 9 : normale; < 5: cognitivamente deteriorato; 5-8: border	**TOTALE**		

Linee guida per il punteggio

Disegno dell'Orologio. Domanda 3: risposta corretta se i numeri 12, 3, 6 e 9 sono collocati senza errori nel cerchio ed anche i restanti numeri delle ore sono inseriti in modo congruo. Domanda 4, risposta corretta se le lancette sono puntate sui numeri 11 e 2 anche qualora la lancetta lunga non venga chiaramente distinta da quella corta.

Informazione. Non sono necessarie risposte particolarmente dettagliate: l'importante è che l'intervistato dimostri buona conoscenza di un evento importante riportato dai media di recente. Se vengono date risposte generiche, tipo "guerra" "molta pioggia", chiedete dettagli: se l'intervistato non li fornisce classificare "sbagliato".

www. demenzemedicinagenerale.net



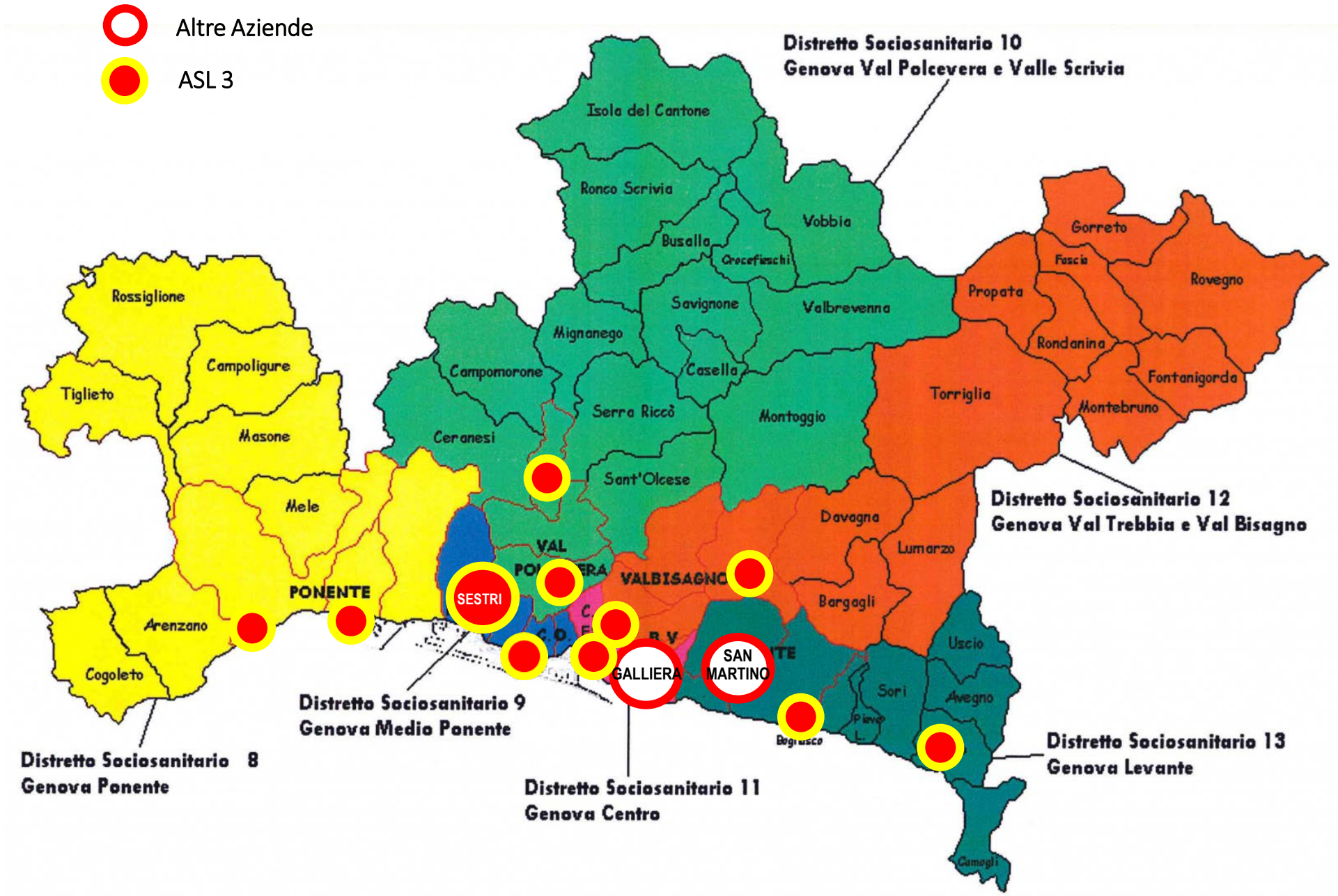
B) Intervista al Famigliare/Conoscente Chiedete: *"Rispetto a qualche anno fa, il paziente.:"*

	SI	NO	Non so	N/A
I. ... ha più difficoltà a ricordare cose che gli sono successe di recente?	0	1		
II. ...ha più difficoltà a rievocare conversazioni di pochi giorni prima?	0	1		
III. ..quando parla, ha più difficoltà a trovare le parole giuste o sbaglia le parole più spesso?	0	1		
IV. .è meno capace di gestire denaro e affari (ad es. pagare conti, programmare le spese)?	0	1		
V. . è meno capace di gestire ed assumere i suoi farmaci autonomamente?	0	1		
VI. richiede più assistenza per utilizzare i mezzi di trasporto (sia privati che pubblici)?	0	1		
Punteggio. Sezione A) = 5-8 + Sezione B) ≤ 3 Confermano Deterioramento Cognitivo	**TOTALE**			

Pirani A, Brodaty H, Martini E, et al. *The validation of the Italian version of the GPCOG (GPCOG-It): a contribution to crossnational implementation of a screening test for dementia in general practice.* Int Psychogeriatrics 2010;22:82-90.

Centri per Disturbi Cognitivi e le Demenze
CDCD

- Geriatri
- Neurologi
- Psicologo
- Infermieri
- Assistenti sanitari
- Assistenti sociali
- Fisioterapista; Logopedista; Foniatra; Fisiatra (consulenze al bisogno)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Distretto Sociosanitario 10
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Isola del Cantone
Ronco Scrivia
Vobbia
Busalla
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Savignone
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Mignanego
Campomorone
Casella
Serra Riccò
Montoggio
Ceranesi
Sant'Olcese
Rossiglione
Campoligure
Tiglieto
Masone
Mele
PONENTE
Arenzano
Cogoleto
Distretto Sociosanitario 8
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SESTRI
VAL
VALBISAGNO
Distretto Sociosanitario 9
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ARS LIGURIA
AGENZIA REGIONALE SANITARIA

REGIONE LIGURIA

INDIRIZZI PER
L'ADOZIONE DEL "PIANO regionale demenze"

Centro per i Disturbi Cognitivi e per le Demenze
Obiettivi:

- Garantire una diagnosi adeguata e tempestiva
- Migliorare la qualità di vita dei pazienti e dei loro familiari, fornendo specifiche indicazioni terapeutiche ed assistenziali durante tutto il decorso della malattia
- Garantire interventi assistenziali individualizzati in collaborazione con i Medici di Medicina Generale (MMG)
- Promuovere il mantenimento delle funzioni cognitive e dell'autonomia funzionale, attraverso percorsi riabilitativi appositamente sviluppati
- Favorire la permanenza del paziente al domicilio il più a lungo possibile
- Favorire l'accesso alla rete dei servizi sia istituzionali che non
- Fornire supporto psicologico ai familiari che assistono le persone affette da demenza
- Svolgere attività informativa e formativa sia per i familiari degli utenti che per il personale dei Servizi coinvolti.
- Adeguare, espandere e specializzare la rete dei servizi
- Contribuire alla conoscenza della malattia e della sua gestione sia a livello scientifico, che informativo e culturale

Il CDCD costituisce il centro di coordinamento di tutti i soggetti coinvolti a vario titolo nel processo di cura del paziente con demenza

Come arrivare ai CDCD ?

- Il paziente si rivolge al Medico di Famiglia

- Valutazione (GPCog)
- Valutazione cause secondarie

- Invio per valutazione specialistica neurologica, geriatrica o psichiatrica al CDCD di zona (prenotazione a CUP) con «visita neurologica/geriatrica CDCD per decadimento cognitivo/demenza/Disturbo neurocognitivo/deficit mnesico»

Centri per i Disturbi Cognitivi e le Demenze (CDCD)

CARTA DEI SERVIZI

Presentazione

Il Centro per i Disturbi Cognitivi e Demenze (CDCD) nasce per fornire un adeguato percorso diagnostico e di presa in carico dei soggetti affetti da disturbi cognitivi e demenza e dei loro familiari:

- Il CDCD è il **centro di coordinamento** di tutti i soggetti coinvolti a vario titolo nel processo di cura e assistenza della persona e della sua famiglia, in primis il MMG e la rete dei servizi, anche non sanitari (Comuni, Terzo settore, ecc.)
- Il CDCD rappresenta l'**evoluzione della UVA** (Unità di Valutazione Alzheimer) e si configura quale organizzazione a rete, in cui un ambulatorio centrale coordina e sovraintende l'attività di altri ambulatori e servizi periferici, in continuità con l'assistenza di base, fornita dal MMG.

Per ogni ragione all'interno degli ambulatori opera un gruppo di lavoro multidisciplinare composto da diversi specialisti e figure professionali, che lavorano secondo protocolli condivisi.

Obiettivi del CDCD sono:

- Garantire una diagnosi adeguata e tempestiva
- Migliorare la qualità di vita dei pazienti e dei loro familiari, fornendo specifiche indicazioni terapeutiche ed assistenziali durante tutto il decorso della malattia

DOCUMENTI

- Centri per i disturbi cognitivi e le demenze (CDCD) - Pieghevole informativo (106.24 Kb)

CONTENUTI CORRELATI

- Sedi e contatti Centri per i Disturbi Cognitivi e le Demenze (CDCD)
- Struttura Complessa Neurologia

COSA SONO I DISTURBI COGNITIVI

I DISTURBI COGNITIVI sono caratterizzati dalla compromissione delle funzioni mentali di una persona e si possono manifestare con perdita di memoria, difficoltà nell'attenzione, alterazioni del linguaggio, disorientamento spaziale e temporale.

Possono interferire con la capacità di eseguire azioni consuete della quotidianità (gestire la casa, organizzare la spesa, assumere i farmaci, gestire il denaro ecc.) e/o causare cambiamenti psicologici e comportamentali.

I disturbi cognitivi possono avere diverse cause che devono essere valutate da un esperto; tra queste, malattie degenerative (Alzheimer, Parkinson, ecc.) ma anche problematiche cerebro-vascolari, metaboliche, psichiatriche, ecc.

Quando questi disturbi sono tanto severi da causare la perdita dell'autonomia si parla di Demenza.

Nel **CDCD** un'equipe di esperti potrà definire l'entità del problema, diagnosticare le condizioni patologiche che lo causano e ottimizzare la cura, aiutando il paziente ed i familiari lungo tutto il percorso della malattia.

Come arrivare da noi

NB: Al CDCD si accede su indicazione del Medico di Medicina Generale con richiesta di visita (neurologica o geriatrica) per "disturbi cognitivi/deficit mnesico/demenza" e prenotabile tramite CUP.

Come contattarci

Per prenotazioni
CUP ASL 3 tel 800 098543
Per informazioni sui servizi offerti e per problematiche cliniche dei pazienti (già conosciuti):
WhatsApp 3392904262
(ore 11.30-13.30 giorni feriali)
Email CDCDponente@asl3.liguria.it

Organizzazione sul territorio

Gli ambulatori CDCD di ASL 3 sono presenti capillarmente in ogni distretto sociosanitario

Accedi con QRcode per l'elenco degli ambulatori presenti nel tuo distretto

o visita www.asl3.liguria.it

Asl3
Sistema Sanitario Regionale Liguria

CENTRI *per i* DISTURBI COGNITIVI *e le* DEMENZE (CDCD)

Chi siamo

Il Centro per i Disturbi Cognitivi e per le Demenze (**CDCD**) è un servizio specialistico ambulatoriale che si occupa della cura dei disturbi cognitivi e delle demenze, coordinando tutti i soggetti coinvolti nel processo di cura e assistenza della persona e della sua famiglia (Medico di medicina generale, ASL, Comuni, RSA, Centri Diurni, Volontariato)

Il **CDCD** fornisce un percorso diagnostico e di presa in carico dei soggetti affetti da disturbi cognitivi e demenza e dei loro familiari.

Nei **CDCD** opera un gruppo di lavoro multidisciplinare composto da Medici specialisti, Psicologi, Infermieri Assistenti Sociali, Assistenti Sanitari e altri professionisti (Fisioterapista, Logopedista, ecc.) esperti nel campo dei disturbi cognitivi.

I **CDCD** sono coordinati dalla Rete Regionale Demenze di Regione Liguria e da ALISA.

Cosa offriamo

- Una rete di ambulatori specialistici dedicati alla presa in carico complessiva del paziente, in collaborazione con il Medico di base
- Indicazioni terapeutiche per il trattamento cognitivo comportamentale e farmacologico dei disturbi della memoria e del comportamento
- Supporto e indicazioni per il mantenimento a casa delle persone con demenza
- Facilitazione di accesso alla rete dei servizi Regionali, ASL, Comunali, Centri Diurni, RSA
- Fornitura di ausili e presidi
- Informazione e formazione a chi assiste le persone con demenza

Percorso informativo per chi si occupa di persone con demenza

I professionisti del **CDCD** hanno realizzato alcuni video informativi dedicati alle famiglie e caregiver (chi si prende cura del paziente) con indicazioni finalizzate alla migliore assistenza della persona con demenza

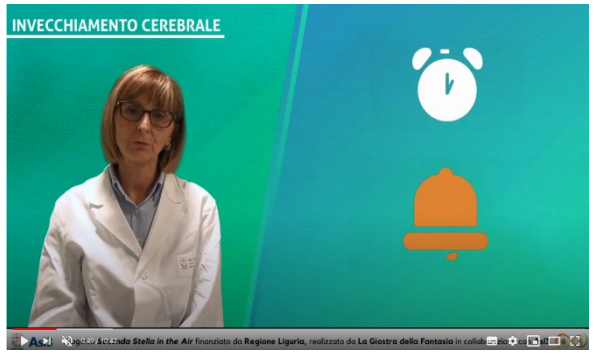
(Percorso realizzato in collaborazione con "La Giostra della Fantasia"– ONLUS)

Accedi con QRcode per l'elenco dei video disponibili

o visita www.asl3.liguria.it

Corso per familiari «online»

LEZIONI	Specialista
Cosa è la demenza	Geriatra
Cosa sono I CDCD	Geriatra
Invecchiamento cerebrale	Geriatra
Diagnosi demenza: strumenti a disposizione del clinico	Neurologo
Cosa è la malattia di Alzheimer	Neurologo
Demenza vascolare	Neurologo
Demenza fronto temporale	Neurologo
Reazioni psicologiche delle persone con demenza	Psicologa
BPSD	Psichiatra
Rapporto tra attività motoria e disturbi cognitivi	Fisioterapista
Difficoltà deglutizione	Logopedista
Diritti umani, capacità e incapacità di agire, persone con demenza, valore della persona	Assistente Sociale
Supporti legislativi a favore di persone affette da disturbi cognitivi	Assistente Sociale
Supporti legislativi, grave e gravissima disabilità	Assistente Sociale
I Centri Diurni	Assistente Sanitario
Servizi di assistenza domiciliare: DOGE	Assistente Sociale
Rete dei servizi semiresidenziali e residenziali del Comune di Genova	Assistente Sociale
Segretariato sociale	Assistente Sociale



Grazie per l’attenzione !