Sharp vs Scattered

Carolyn K Clevenger DNP

AGENDA

Diagnosing Alzheimer's Disease

New Medications

Dementia Care Programs


10
Warning Signs
Recent memory loss
Difficulty with regular tasks
Loss of initiative
Time and place disorientation
Abstract thinking problems
Language problems
Mood and behavior changes
Poor judgment
Personality changes
Misplacing items

AD8 Screener (> 2 "yes"… should test further)

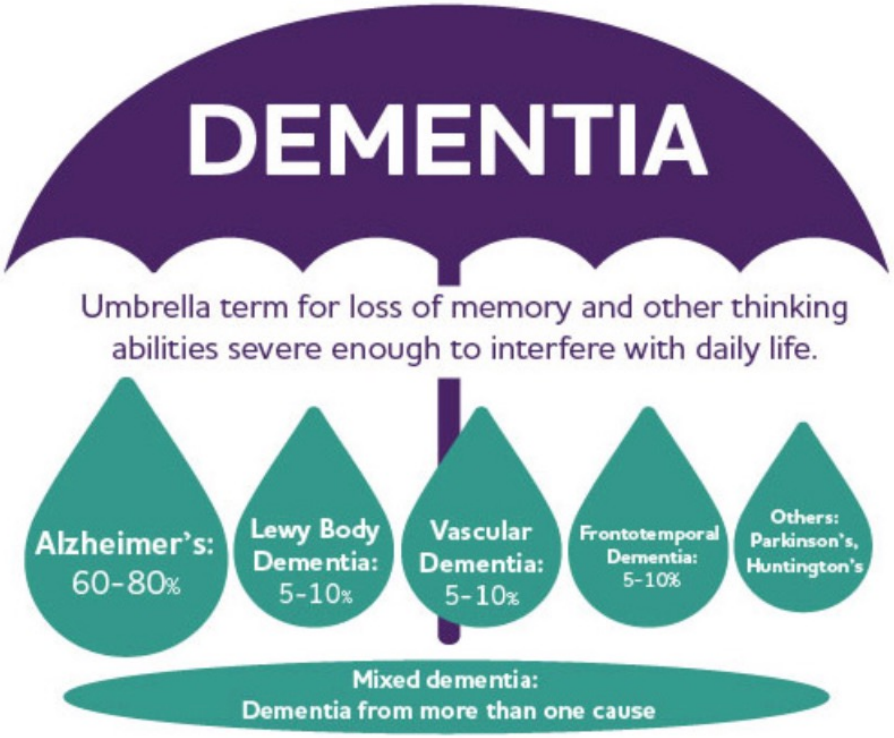
	YES, a change	NO, no change	N/A, don't know
Problems with judgment (making decisions, bad financial decisions, problems with thinking)			
Less interest in hobbies or activities			
Repeats the same things over and over (questions, stories, statements)			
Trouble learning how to use a tool, appliance, or gadget			
Forgets correct month or year			
Trouble handling complicated financial affairs (balancing checkbook, income taxes, paying bills)			
Trouble remembering appointments			
Daily problems with thinking and/or memory			

Know your brain, know yourself

Brain health and why it matters

Coming Soon: BrainCheck

Get baseline score to monitor over time



What We've Said

Dementia is an umbrella term

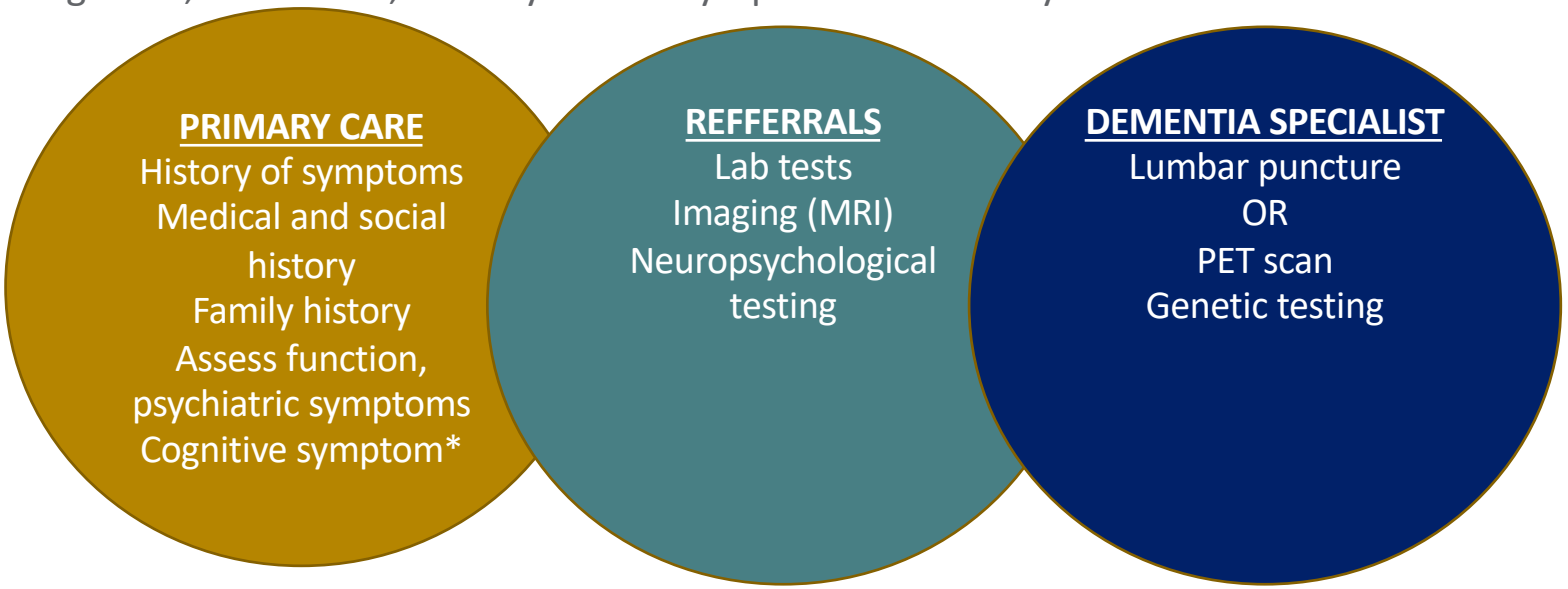
Alzheimer's disease is one type

How Do We Know It’s Alzheimer’s?

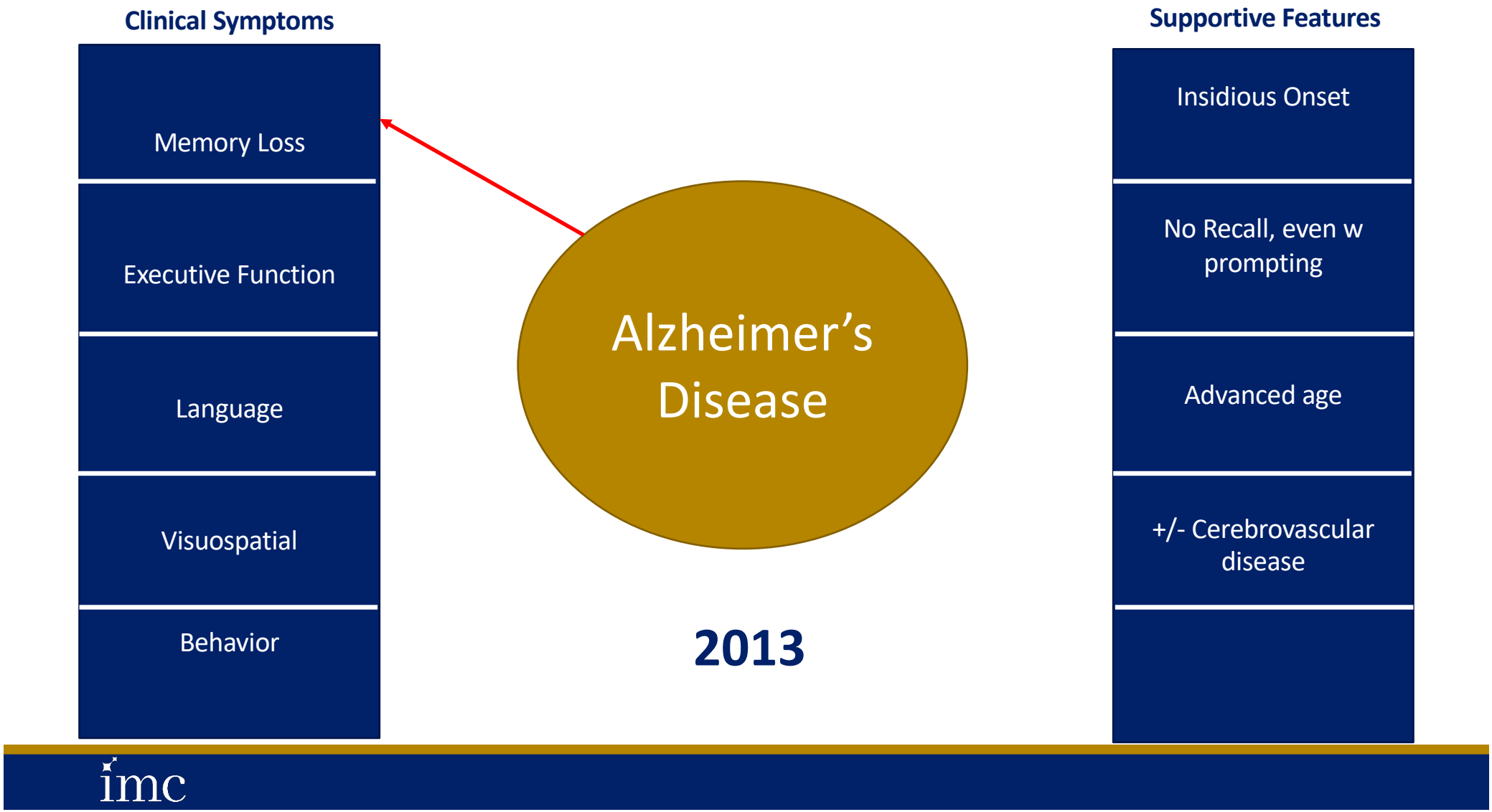
The Answer has Changed

Diagnostic Evaluation (DETEcD-ADRD)

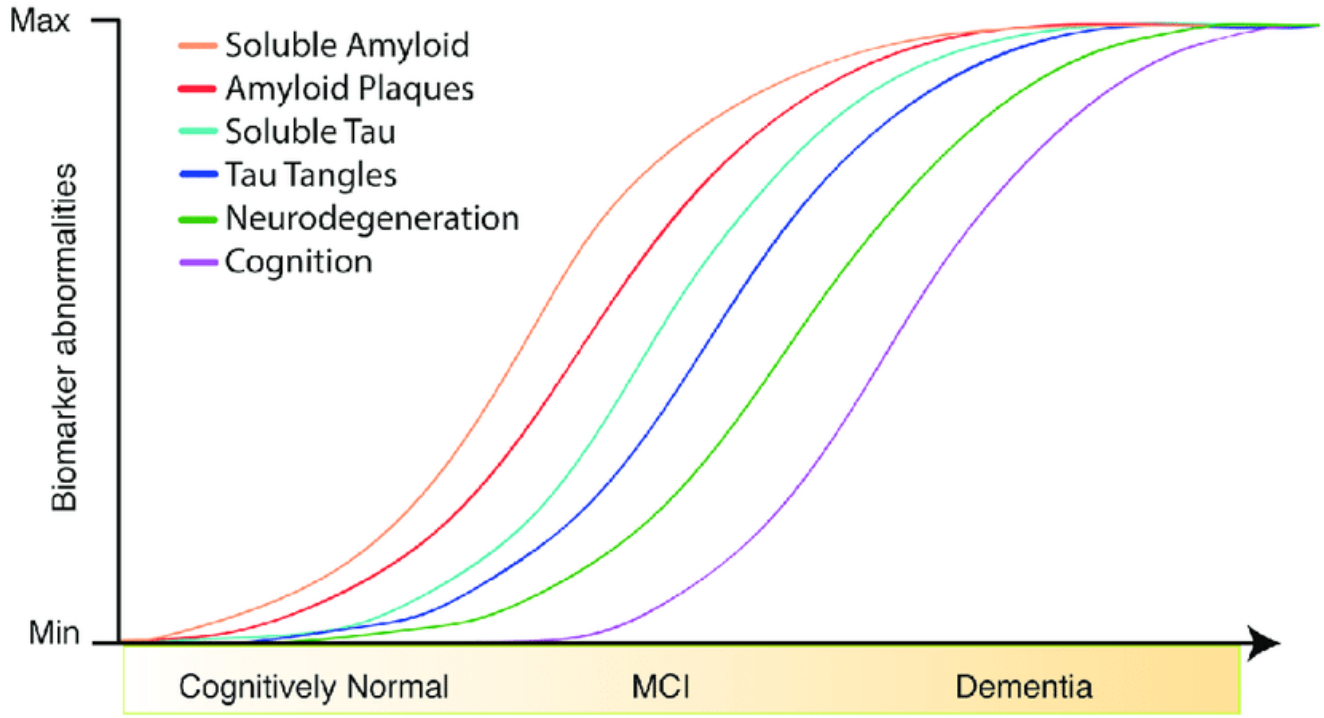
Cognitive, Functional, and Psychiatric symptoms most likely due to Alzheimer's



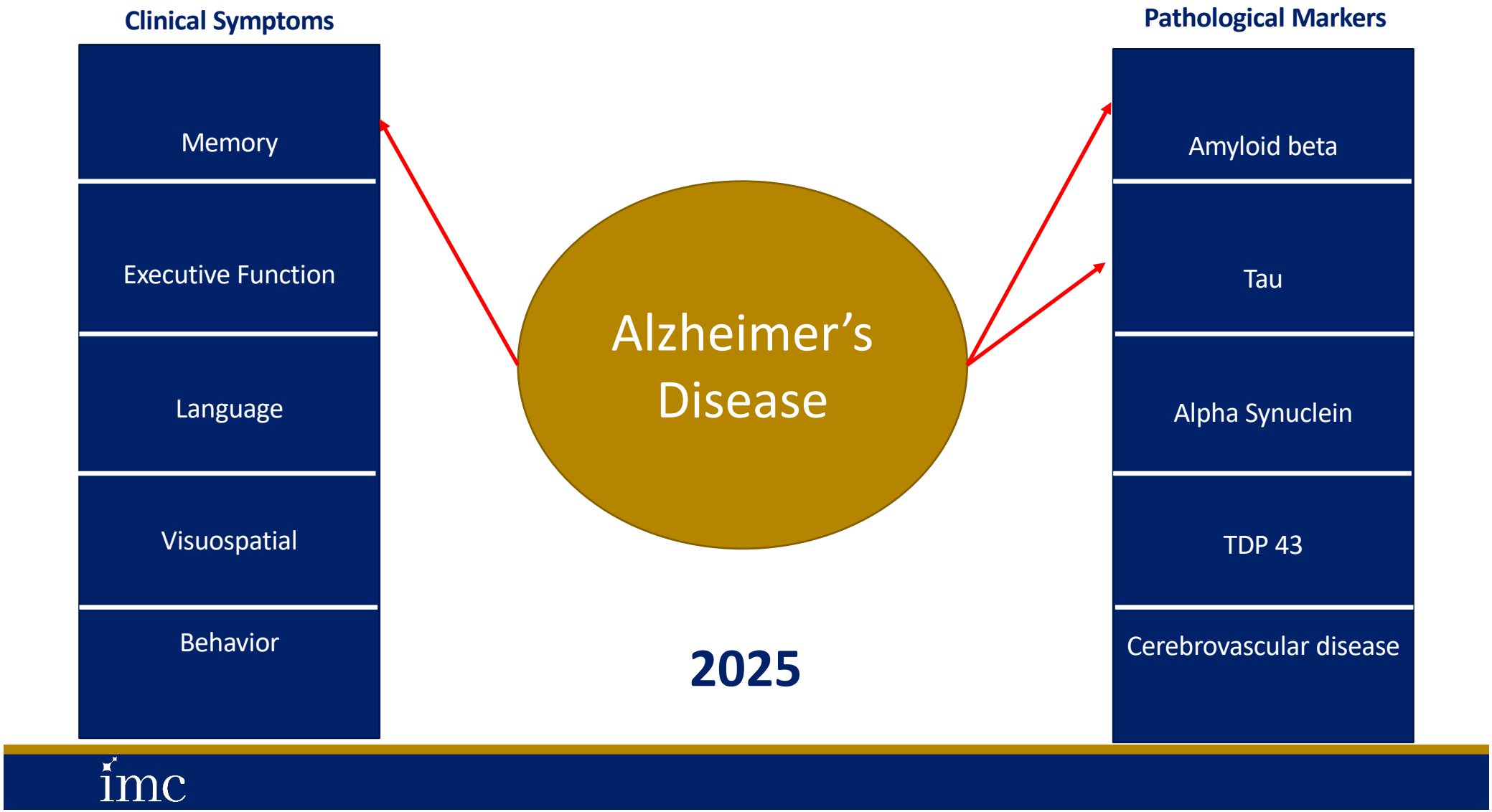
Dickerson, B. C., Atri, A., Clevenger, C., Karlawish, J., Knopman, D., Lin, P. J., Norman, M., Onyike, C., Sano, M., Scanland, S., & Carrillo, M. (2024). Alzheimer's Association clinical practice guideline for the Diagnostic Evaluation, Testing, Counseling, and Disclosure of Suspected Alzheimer's Disease and Related Disorders (DETeCD-ADRD): Executive summary of recommendations for primary care. Alzheimer's & Dementia : the Journal of the Alzheimer's Association, 10.1002/alz.14333. https://doi.org/10.1002/alz.14333


Clinical Symptoms
Memory Loss
Executive Function
Language
Visuospatial
Behavior
Alzheimer's Disease
2013
Supportive Features
Insidious Onset
No Recall, even w prompting
Advanced age
+/- Cerebrovascular disease

Disease Progression of Alzheimer's: ATN



Jack, C. R., Wiste, H. J., Lesnick, T. G., Weigand, S. D., Knopman, D. S., Vemuri, P., Pankratz, V. S., Senjem, M. L., Gunter, J. L., Mielke, M. M., Lowe, V. J., Boeve, B. F., Petersen, R. C. (2013). Brain beta-amyloid load approaches a plateau. Neurology, 80, 890-6. https://dx.doi.org/10.1212/WNL.0b013e3182840bbe


Clinical Symptoms
Memory
Executive Function
Language
Visuospatial
Behavior
Alzheimer's Disease
2025
Pathological Markers
Amyloid beta
Tau
Alpha Synuclein
TDP 43
Cerebrovascular disease

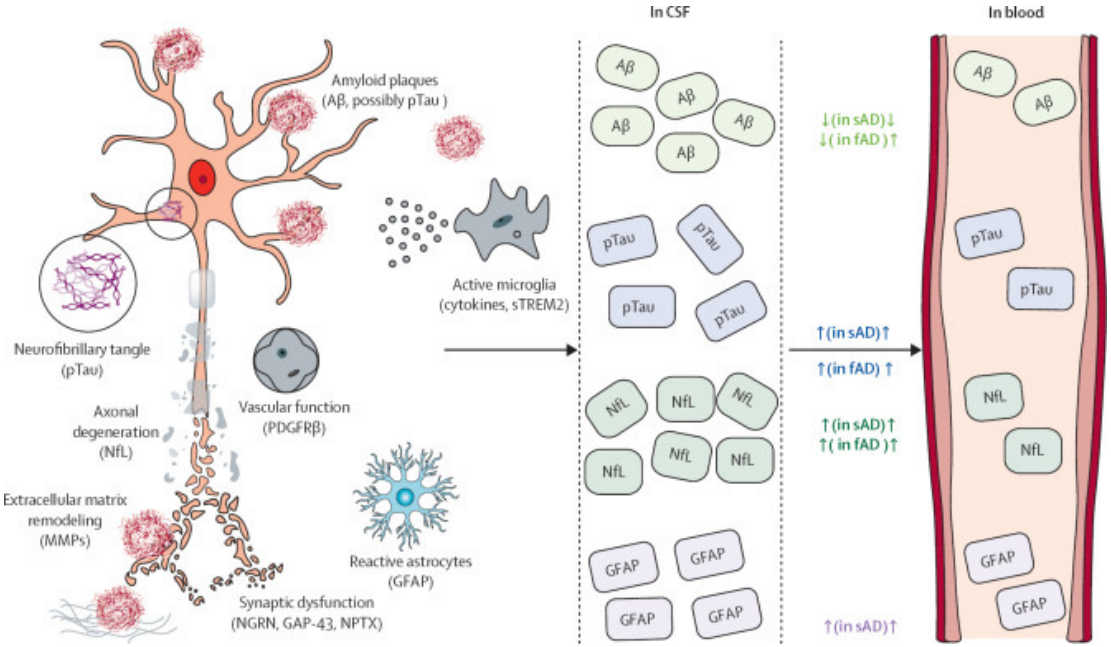
Blood Testing

Amyloid

Tau

Neurodegeneration

(+Genetics)



Teunissen CE, Verberk IMW, Thijssen EH, et al. Blood-based biomarkers for Alzheimer's disease: towards clinical implementation. *Lancet Neurol*. 2022;21(1):66-77. doi:10.1016/S1474-4422(21)00361-6

Real-World Challenges in Diagnostics

Blood Tests are not diagnostic all by themselves

- Available at chain laboratory companies though
- FDA approved, not Medicare/insurer covered

Neurotracers (like Amyvid, Tauvid) are gold standard

- FDA approved, Medicare coverage in certain cases

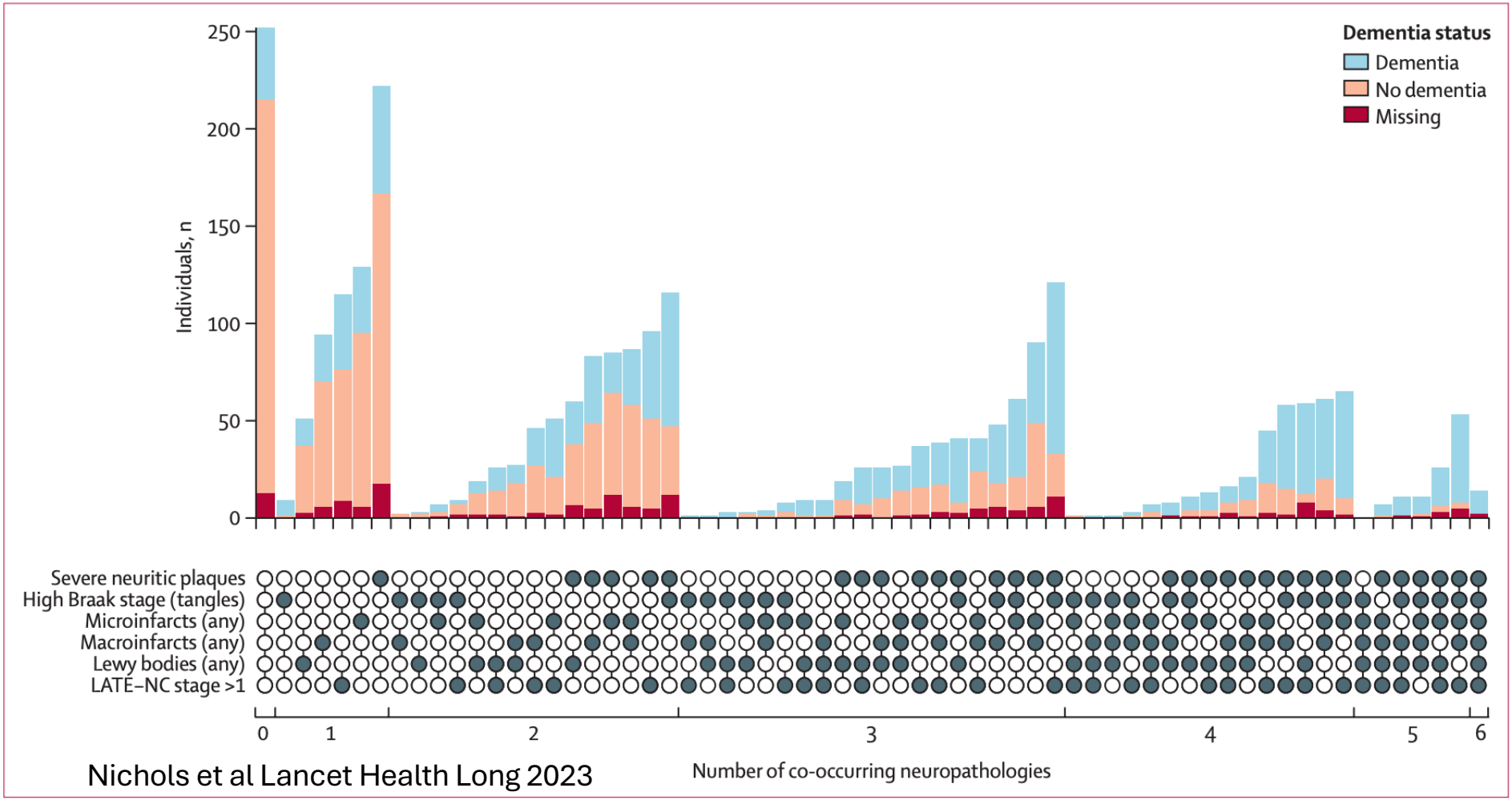
Lumbar punctures

- Both FDA approved and Medicare covered
- Requires specialized skillset

Among Individuals who Meet Clinical Criteria for Probable AD Dementia

A/T/N score	NIA-AA classification
A-/T-/N-	Dementia, unlikely due to AD
A+/T-/N-	Intermediate likelihood; probable AD dementia based on clinical criteria
A+/T+/N-	High likelihood; probable AD dementia; based on clinical criteria
A+/T-/N+	High likelihood; probable AD dementia; based on clinical criteria
A+/T+/N+	High likelihood AD pathophysiology
A-/T+/N-	Probable AD dementia; based on clinical criteria
A-/T-/N+	Intermediate likelihood; probable AD dementia based on clinical criteria
A-/T+/N+	Intermediate likelihood; probable AD dementia based on clinical criteria

Jack, C. R., Bennett, D. A., Blennow, K., Carrillo, M. C., Feldman, H. H., Frisoni, G. B., Hampel, H., Jagust, W. J., Johnson, K. A., Knopman, D. S., Petersen, R. C., Scheltens, P., Sperling, R. A., Dubois, B. (2016). A/t/n: an unbiased descriptive classification scheme for alzheimer disease biomarkers. Neurology, 87, 539-47. https://dx.doi.org/10.1212/WNL.0000000000002923



Nichols et al Lancet Health Long 2023

Current Medication Treatments for Cognitive Diseases

Symptomatic Treatments

ARICEPT (DONEPEZIL) OR EXELON (RIVASTIGMINE) PATCH

Keep more acetylcholine in the brain

Improves attention

Slows disease progression

NAMENDA (MEMANTINE)

Maintains ability to do self-care, delays nursing home admission

Preserves brain cells by preventing overstimulation (“neuroexcitability”)

OTHER SYMPTOMS

Anxiety
Mood swings
Psychosis
Depression
Agitation
Insomnia

Anti-Amyloid Drugs

FOR AMYLOID-DOMINANT ALZHEIMER'S DISEASE

imc

Pipeline of Amyloid Treatments

Amyloid Hypothesis: amyloid beta protein is the *primary* cause of synaptic dysfunction and neurodegeneration in AD

Study of Anti-AD Clinical Trials, 2002-2012

Most drugs were Anti-Amyloid beta (70 of 146)

- Advanced stage 1 to stage 2: 28% (72% attrition rate)
- Advanced stage 2 to stage 3: 2% (98% attrition rate)

"Overall, 244 compounds were assessed in the decade of 2002 through 2012 and one was approved for marketing; excluding the 14 compounds currently in Phase 3, the success rate for advancing agents for regulatory approval is 0.4% (99.6% attrition)."

Also, in 2014: Solanezumab and bapineuzumab trials unsuccessful

Cummings JL, Morstorf T, Zhong K. (2014). Alzheimer's disease drug-development pipeline: few candidates, frequent failures. Alzheimers Res Ther. 6(4):37. doi: 10.1186/alzrt269. PMID: 25024750; PMCID: PMC4095696.

Story of Aducanumab

ARTICLE

doi:10.1038/nature19323

The antibody aducanumab reduces Aβ plaques in Alzheimer's disease

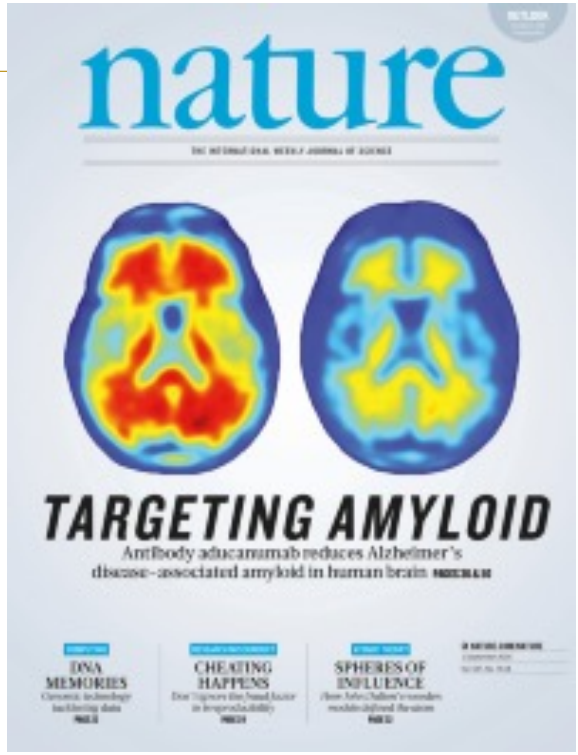
Jeff Sevigny[1]*, Ping Chiao[1]*, Thierry Bussière[1]*, Paul H. Weinreb[1]*, Leslie Williams[1], Marcel Maier[2], Robert Dunstan[1], Stephen Salloway[3], Tianle Chen[1], Yan Ling[1], John O'Gorman[1], Fang Qian[1], Mahin Arastu[1], Mingwei Li[1], Sowmya Chollate[1], Melanie S. Brennan[1], Omar Quintero-Monzon[1], Robert H. Scannevin[1], H. Moore Arnold[1], Thomas Engber[1], Kenneth Rhodes[1], James Ferrero[1], Yaming Hang[1], Alvydas Mikulskis[1], Jan Grimm[2], Christoph Hock[2,4], Roger M. Nitsch[2,4]§ & Alfred Sandrock[1]§

Alzheimer's disease (AD) is characterized by deposition of amyloid-β (Aβ) plaques and neurofibrillary tangles in the brain, accompanied by synaptic dysfunction and neurodegeneration. Antibody-based immunotherapy against Aβ to trigger its clearance or mitigate its neurotoxicity has so far been unsuccessful. Here we report the generation of aducanumab, a human monoclonal antibody that selectively targets aggregated Aβ. In a transgenic mouse model of AD, aducanumab is shown to enter the brain, bind parenchymal Aβ, and reduce soluble and insoluble Aβ in a dose-dependent manner. In patients with prodromal or mild AD, one year of monthly intravenous infusions of aducanumab reduces brain Aβ in a dose- and time-dependent manner. This is accompanied by a slowing of clinical decline measured by Clinical Dementia Rating—Sum of Boxes and Mini Mental State Examination scores. The main safety and tolerability findings are amyloid-related imaging abnormalities. These results justify further development of aducanumab for the treatment of AD. Should the slowing of clinical decline be confirmed in ongoing phase 3 clinical trials, it would provide compelling support for the amyloid hypothesis.

The amyloid hypothesis posits that Aβ-related toxicity is the primary cause of synaptic dysfunction and subsequent neurodegeneration that underlies the progression characteristic of AD[1,2]. Genetic, neuropathological, and cell biological evidence strongly suggest that targeting Aβ could be beneficial for patients with AD[3,4]. So far, attempts at therapeutically targeting Aβ have not been successful[5–7], casting doubt on the validity of the amyloid hypothesis. However, the lack of success may

Removal of brain Aβ plaques in patients with AD

In the PRIME study, 165 patients were randomized and treated between October 2012 and January 2014 at 33 sites in the United States. Patients with a clinical diagnosis of prodromal or mild AD and visually positive Aβ PET scan[9] were given monthly intravenous infusions of placebo or aducanumab at doses of 1, 3, 6 or 10 mg kg^{-1} for one year. Of these patients, 125 completed and 40 discontinued treatment, most com-



Sevigny, J., Chiao, P., Bussiere, T., Weinreb, P. H., Williams, L., Maier, M., Dunstan, R., Salloway, S., Chen, T., Ling, Y., O'Gorman, J., Qian, F., Arastu, M., Li, M., Chollate, S., Brennan, M. S., Quintero-Monzon, O., Scannevin, R. H., Arnold, H. M., Engber, T., Rhodes, K., Ferrero, J., Hang, Y., Mikulskis, A., Grimm, J., Hock, C., Nitsch, R. M., Sandrock, A. (2016). The antibody aducanumab reduces abeta plaques in alzheimer's disease. Nature, 537, 50-6. https://dx.doi.org/10.1038/nature19323

Measuring "Success"

Amyloid Measures, PET

Standard Uptake Value ratio (SUVr) in serial scans

Change from baseline amyloid SUVr and tau PET

Also, change in brain volume

Amyloid Measures, Lumbar puncture

Change from baseline relevant amyloid proteins and phosphorylated tau and total tau as measured by CSF analysis

Clinical Measures

MMSE

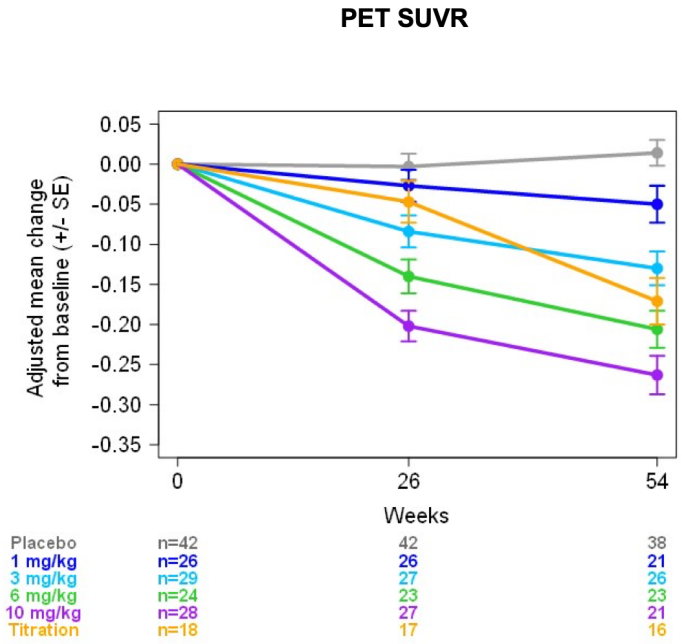
Alzheimer's Disease AS-Cog (ADASCog)

- **13**-item scale to assess severity of symptoms in memory, orientation, language, praxis, and reason

Clinical Dementia Rating Scale-Sum of Boxes (CDR-SB)

- 6 boxes measuring memory, orientation, judgment and problem-solving, community affairs, home and hobbies, and personal care
- Score range 0-18, higher indicates advanced stage

Aducanumab: Biomarker Results



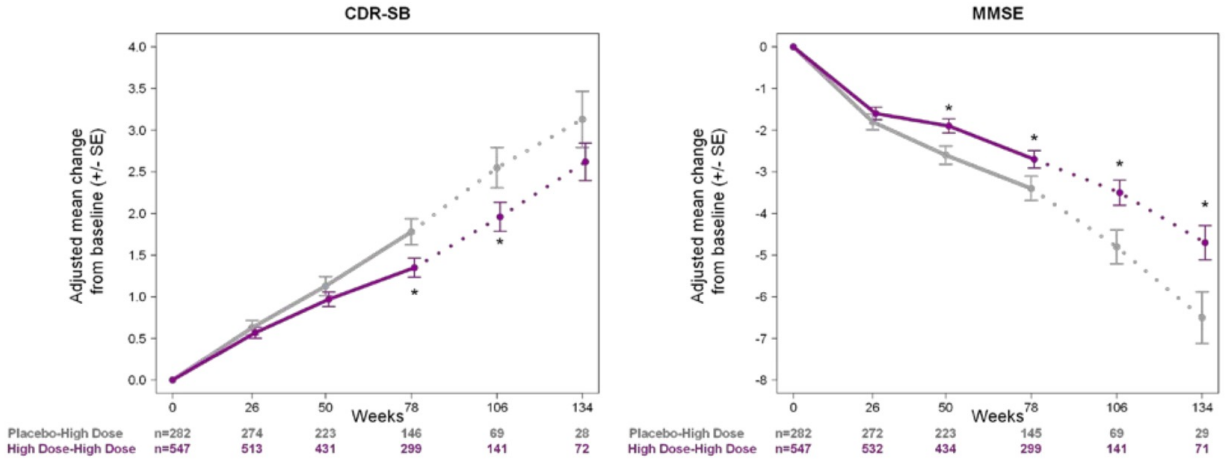
10 mg/kg is appropriate dose for comparison to high dose in Study 302

	Study 302 High Dose	Study 103 10 mg/kg
# 10 mg/kg doses	14	14
SUVR Difference from placebo	-0.278 (Week 78)	-0.277 (Week 54)

(full slide deck available to the public at https://www.fda.gov/media/143504/download)

Aducanumab: Clinical Results

Figure 6: Study 302 Longitudinal Change from Baseline for Clinical Endpoints in High-Dose Aducanumab Treatment Arm through Week 134



FDA Center for Drug Evaluation and Research (2021). Clinical Reviews for application 761178Orig1s000**.** Accessed online at https://www.accessdata.fda.gov/drugsatfda_docs/nda/2021/761178Orig1s000MedR_Redacted.pdf

Controversy

GeriPal podcast, April 15, 2021

- "All Things Amyloid"
- Guest: Gil Rabinovici

FDA Advisory Committee

- Overview in BMJ https://doi.org/10.1136/bmj.n1503

Medicare Payment Advisory Committee

American Geriatrics Society

Alzheimer's Association, UsAgainstAlzheimer

Nothing has been approved for years

People need options and are willing to take risks

Medicare pays for other 'accelerated approved' drugs that are high-risk (oncology)

- This is ageism

Available Amyloid Targeted Therapies

Monoclonal antibody	Target antigen	Efficacy data	Status
Donanemab	Form of Aβ found in aggregated plaques	Significantly slowed iADRS score decline over 76 weeks in the phase 2 TRAILBLAZER-ALZ trial	FDA approved (January 2024)
~~Gantenerumab~~	Conformational epitope on Aβ fibrils, leading to preferential interaction with Aβ aggregates	Failed to slow decline on the CDR-SB and cleared less Aβ plaques than expected in the phase 3 GRADUATE trials	Failed Trial
Lecanemab	Large, soluble Aβ fibrils that may contribute to AD neurotoxicity	Significantly slowed CDR-SB score decline by 27% over 18 months in topline results from the phase 3 CLARITY trial	FDA Approved (July 2023)

Donanemab. Accessed November 28, 2022. https://www.alzforum.org/therapeutics/donanemab. ; Gantenerumab falls short in phase 3. November 14, 2022. Accessed November 28, 2022. https://www.alzforum.org/news/research-news/gantenerumab-falls-short-phase-3-0.; Meglio M. FDA accepts BLA for lecanemab in Alzheimer disease. July 7, 2022. Accessed November 28, 2022. https://www.neurologylive.com/view/fda-accepts-bla-for-lecanemab-alzheimer-disease-sets-pdufa-date-january-2023. Mintun MA et al. *N Engl J Med.* 2021;384(18):1691-1704.

Monoclonal Antibodies

DETERMINE ELIGIBILITY

Does the person have Amyloid? Tau?

What is the extent of neurodegeneration?

- -Brain scan
- -Blood test
- -Neuropsychological tests

What is the genetic (APOE4) status?

Are there previous bleeds or strokes?

What is the risk tolerance?

THERAPY

Initial MRI

Titrating infusions

Care navigation

Monitoring for ARIA-E and ARIA-H

Determination of stop point

Ongoing monitoring

What are We Getting?

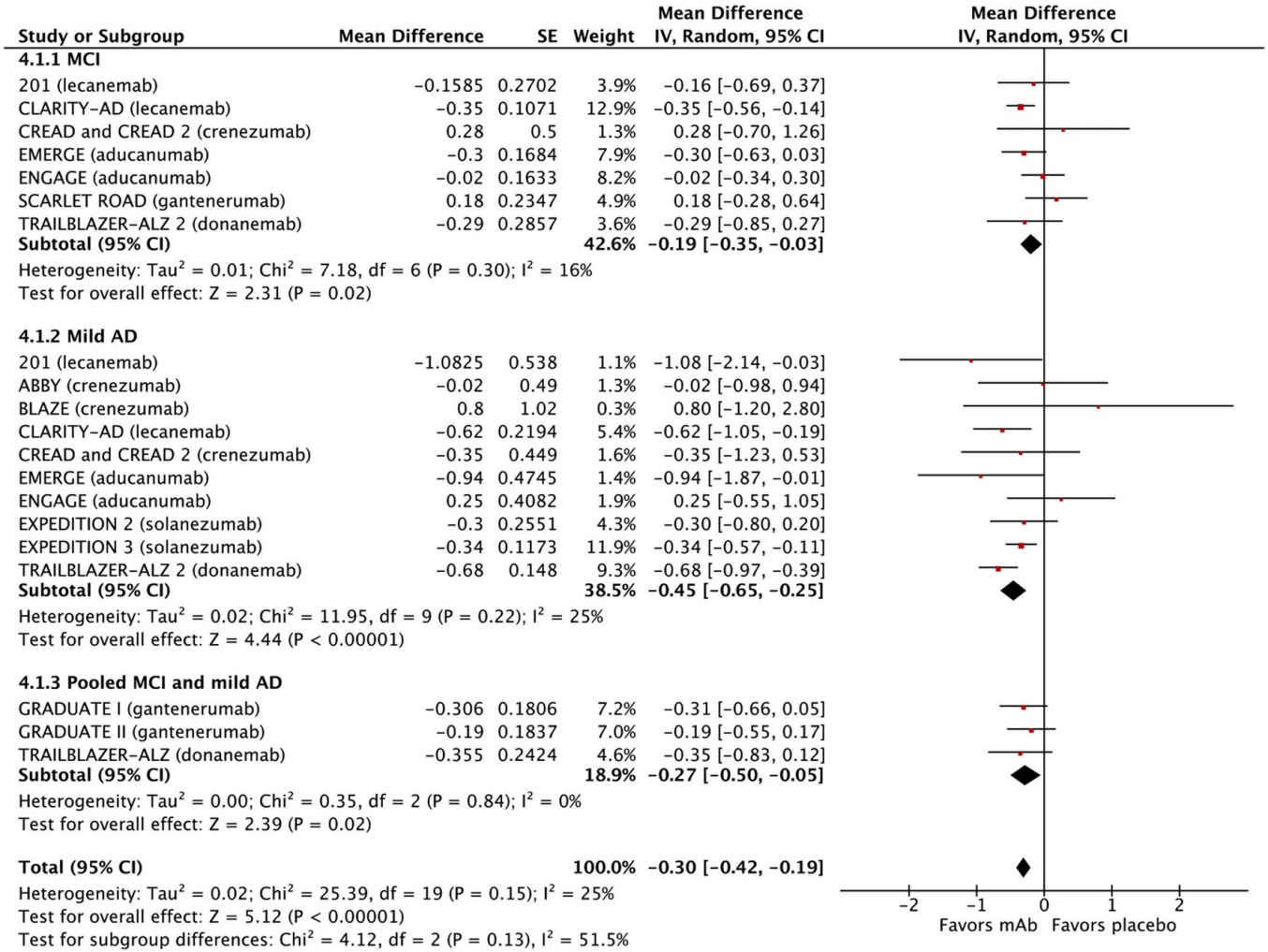
CONCLUSION

“Anti-amyloid-ß mAbs attenuate cognitive and functional decline compared with placebo in early AD;

whether the magnitude of this effect is clinically important remains uncertain, especially relative to the safety profile of these medications.

Starting immunotherapy in patients with MCI was not significantly different than starting in the mild dementia stage.”

Dantas, J.M., Mutarelli, A., Navalha, D.D.P. et al. Efficacy of anti-amyloid-ß monoclonal antibody therapy in early Alzheimer’s disease: a systematic review and meta-analysis. Neurol Sci 45, 2461–2469 (2024). https://doi.org/10.1007/s10072-023-07194-w

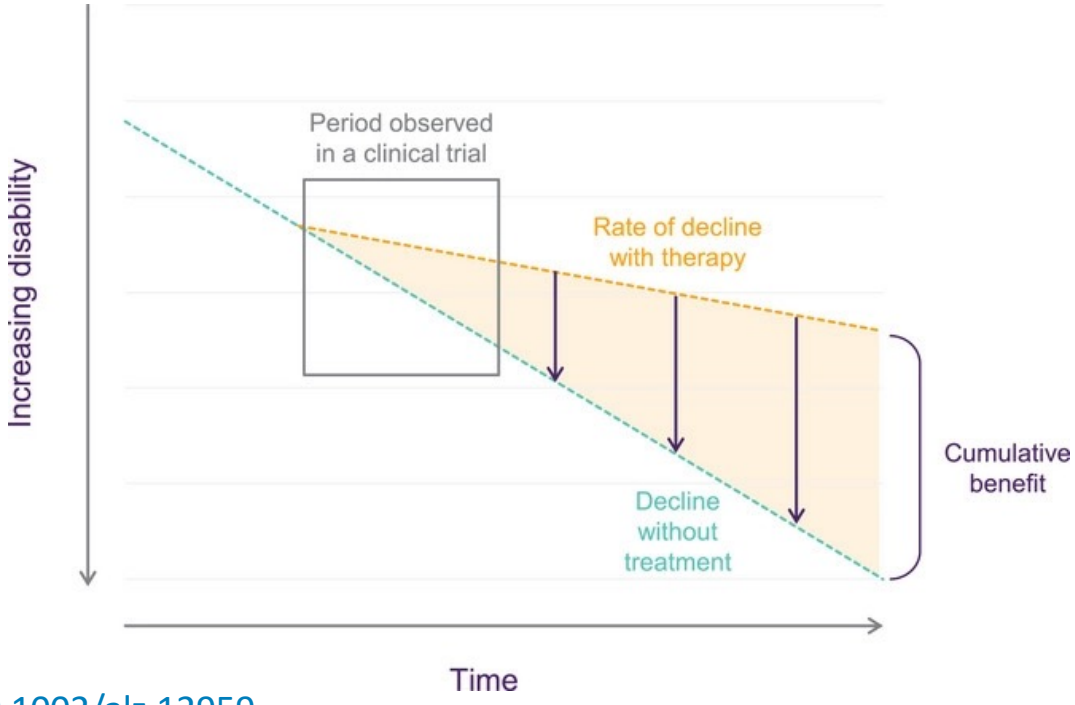

Study or Subgroup
Mean Difference
SE
Weight
Mean Difference IV, Random, 95% CI
Mean Difference IV, Random, 95% CI
4.1.1 MCI
201 (lecanemab) −0.1585 0.2702 3.9% −0.16 [−0.69, 0.37]
CLARITY-AD (lecanemab) −0.35 0.1071 12.9% −0.35 [−0.56, −0.14]
CREAD and CREAD 2 (crenezumab) 0.28 0.5 1.3% 0.28 [−0.70, 1.26]
EMERGE (aducanumab) −0.3 0.1684 7.9% −0.30 [−0.63, 0.03]
ENGAGE (aducanumab) −0.02 0.1633 8.2% −0.02 [−0.34, 0.30]
SCARLET ROAD (gantenerumab) 0.18 0.2347 4.9% 0.18 [−0.28, 0.64]
TRAILBLAZER-ALZ 2 (donanemab) −0.29 0.2857 3.6% −0.29 [−0.85, 0.27]
Subtotal (95% CI) 42.6% −0.19 [−0.35, −0.03]
Heterogeneity: Tau² = 0.01; Chi² = 7.18, df = 6 (P = 0.30); I² = 16%
Test for overall effect: Z = 2.31 (P = 0.02)
4.1.2 Mild AD
201 (lecanemab) −1.0825 0.538 1.1% −1.08 [−2.14, −0.03]
ABBY (crenezumab) −0.02 0.49 1.3% −0.02 [−0.98, 0.94]
BLAZE (crenezumab) 0.8 1.02 0.3% 0.80 [−1.20, 2.80]
CLARITY-AD (lecanemab) −0.62 0.2194 5.4% −0.62 [−1.05, −0.19]
CREAD and CREAD 2 (crenezumab) −0.35 0.449 1.6% −0.35 [−1.23, 0.53]
EMERGE (aducanumab) −0.94 0.4745 1.4% −0.94 [−1.87, −0.01]
ENGAGE (aducanumab) 0.25 0.4082 1.9% 0.25 [−0.55, 1.05]
EXPEDITION 2 (solanezumab) −0.3 0.2551 4.3% −0.30 [−0.80, 0.20]
EXPEDITION 3 (solanezumab) −0.34 0.1173 11.9% −0.34 [−0.57, −0.11]
TRAILBLAZER-ALZ 2 (donanemab) −0.68 0.148 9.3% −0.68 [−0.97, −0.39]
Subtotal (95% CI) 38.5% −0.45 [−0.65, −0.25]
Heterogeneity: Tau² = 0.02; Chi² = 11.95, df = 9 (P = 0.22); I² = 25%
Test for overall effect: Z = 4.44 (P < 0.00001)
4.1.3 Pooled MCI and mild AD
GRADUATE I (gantenerumab) −0.306 0.1806 7.2% −0.31 [−0.66, 0.05]
GRADUATE II (gantenerumab) −0.19 0.1837 7.0% −0.19 [−0.55, 0.17]
TRAILBLAZER-ALZ (donanemab) −0.355 0.2424 4.6% −0.35 [−0.83, 0.12]
Subtotal (95% CI) 18.9% −0.27 [−0.50, −0.05]
Heterogeneity: Tau² = 0.00; Chi² = 0.35, df = 2 (P = 0.84); I² = 0%
Test for overall effect: Z = 2.39 (P = 0.02)
Total (95% CI) 100.0% −0.30 [−0.42, −0.19]
Heterogeneity: Tau² = 0.02; Chi² = 25.39, df = 19 (P = 0.15); I² = 25%
Test for overall effect: Z = 5.12 (P < 0.00001)
Test for subgroup differences: Chi² = 4.12, df = 2 (P = 0.13), I² = 51.5%
−2
−1
0
1
2
Favors mAb
Favors placebo

What Benefit Can We Reasonably Expect?

What is clinically meaningful?

When is meaningful change expected?

"Our expectations of outcomes from therapeutic interventions in AD may need to be modified"



https://alz-journals.onlinelibrary.wiley.com/doi/10.1002/alz.12959

Everything is Relative

CURRENT MEDICATION BENEFITS

Donepezil RCT: Results

Donepezil (both doses) maintained score at 24-weeks

Some residual benefit even after washout (not SS)

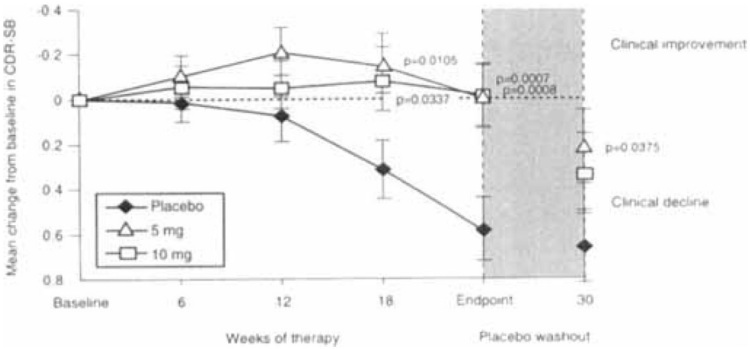


Figure 5. Mean (± SEM) change from baseline in CDR-SB score for 5- and 10-mg/d-donepezil- and placebo-treated patients with mild to moderate Alzheimer's disease.

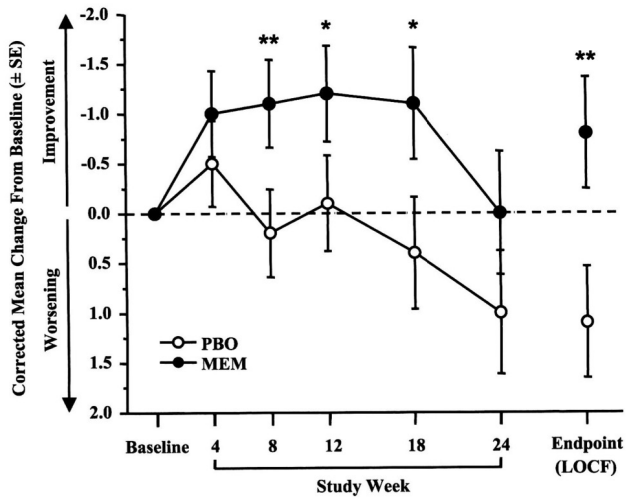
Rogers, S. L., Farlow, M. R., Doody, R. S., Mohs, R., Friedhoff, L. T. (1998). A 24-week, double-blind, placebo-controlled trial of donepezil in patients with Alzheimer's disease. donepezil study group. Neurology, 50, 136-45.

Memantine RCT: Results

Alzheimer's Disease Assessment Scale-Cog

- 11-items
- 70-points (higher = worse)
- Global cognitive performance (language, memory, executive function, etc)

FIGURE 2. Mean Change From Baseline Over Time in Alzheimer's Disease Assessment Scale–Cognitive Subscale Score



No. of Patients							
Memantine	195	191	187	177	163	160	195
Placebo	198	195	188	177	172	162	198
Corr. Mean Difference		-0.5	-1.4	-1.1	-1.5	-1.1	-1.9
P Value		0.29	0.006	0.04	0.02	0.13	0.003
SDs							
Memantine		5.94	6.02	6.39	7.15	7.84	7.82
Placebo		6.00	6.03	6.39	7.34	7.89	7.88

*p < 0.05
**p < 0.01

LOCF: last observation carried forward; SDs: standard deviations.

Kueper JK, Speechley M, Montero-odasso M. The Alzheimer's Disease Assessment Scale-Cognitive Subscale (ADAS-Cog): modifications and responsiveness in pre-dementia populations. a narrative review. J Alzheimers Dis. 2018;63(2):423-444. doi:10.3233/JAD-170991

Peskind, E. R., Potkin, S. G., Pomara, N., Ott, B. R., Graham, S. M., Olin, J. T., McDonald, S. (2006). Memantine treatment in mild to

What are the Immediate Risks?

Amyloid-Related Imaging Abnormalities

Edema (swelling)

Hemorrhage (bleeding)

Withington CG, Turner RS. Amyloid-Related Imaging Abnormalities With Anti-amyloid Antibodies for the Treatment of Dementia Due to Alzheimer's Disease. *Front Neurol*. 2022;13:862369. Published 2022 Mar 23. doi:10.3389/fneur.2022.862369

	Aducanumab (2)	Lecanemab (3, 4)	Donanemab (5)	Gantenerumab (6)
CNS amyloid clearance	+	+	+	+
Clinical stabilization	+/–	+	+	?
Tau reduction	+	+	?	+
Estimated ARIA-E incidence at the highest dose (%)	42	9.9	27.5	13.5
Estimated ARIA-H incidence at the highest dose (%)	Same as placebo	5.6	30.5	16.2

ARIA (–E/–H), Amyloid-related imaging abnormalities (–edema/–hemorrhagic); [+] indicates that the drug is associated with the variable in the row; ? not known.

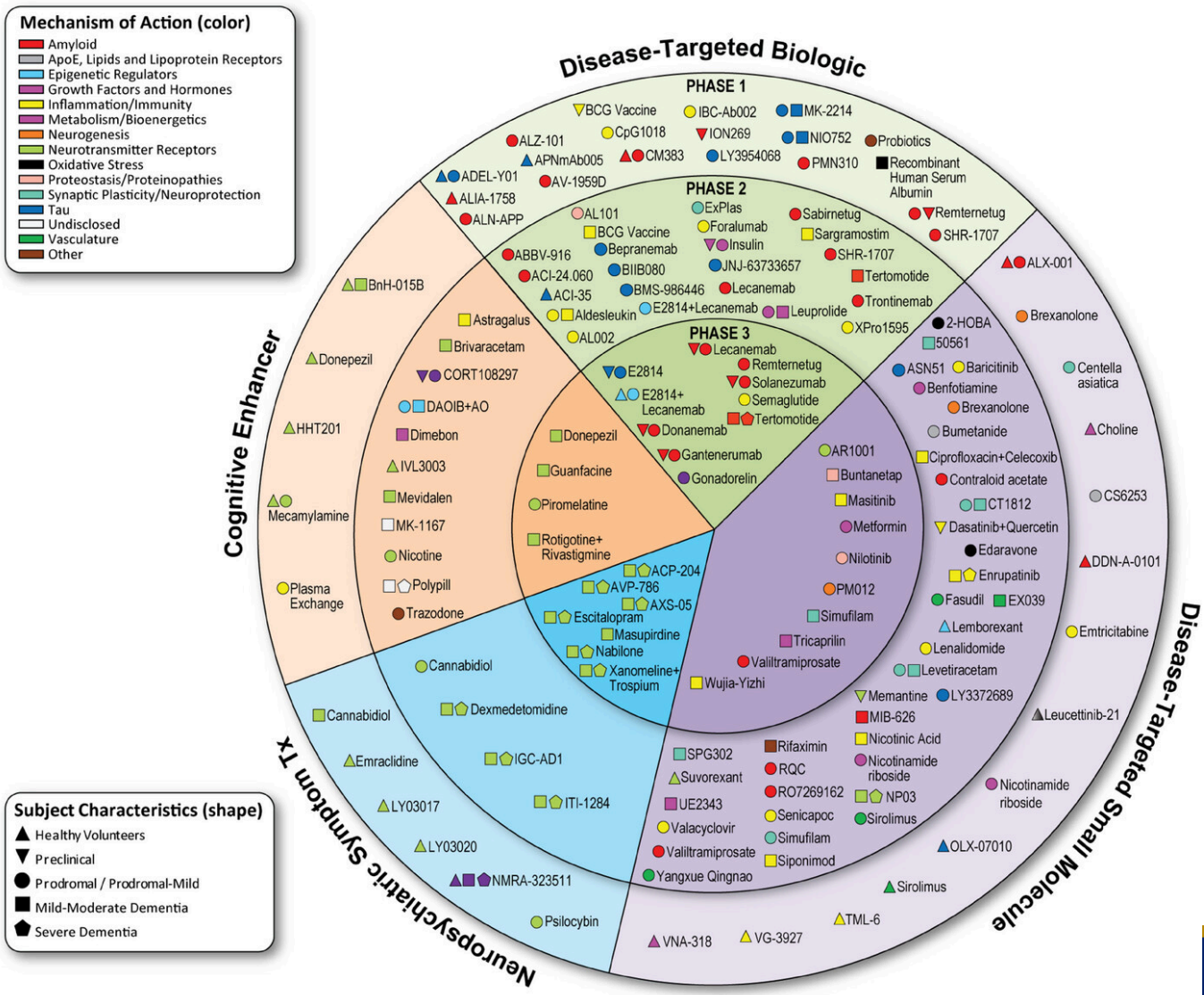
Long-Term Effect Modeling

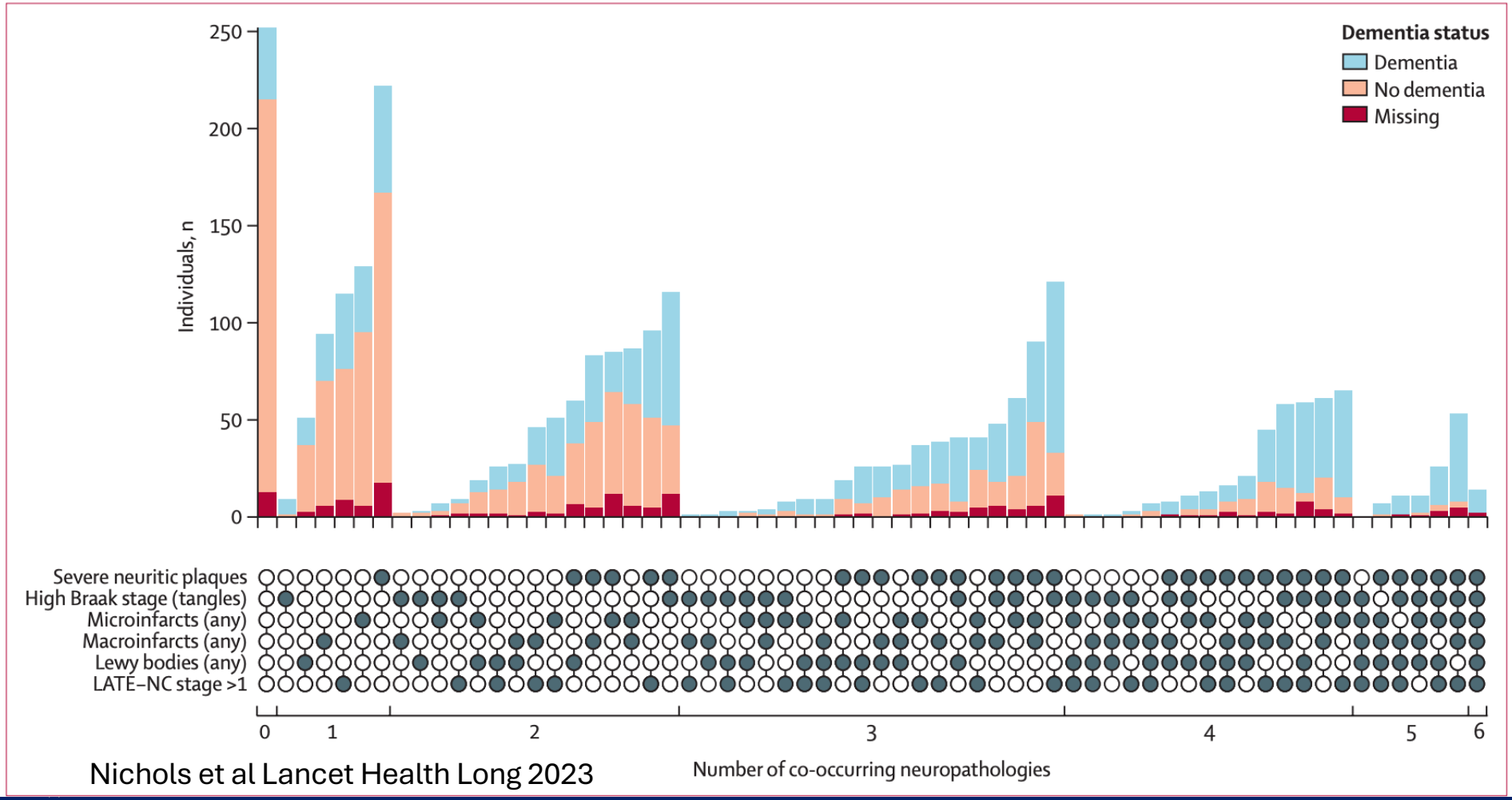
Potentially cause

- Whole brain atrophy
- Ventricle enlargement
- Especially for those who experience ARIA*
- ~8 months faster decline
- *APOe4/4 most likely to experience ARIA

More to Come

2025 Alzheimer's Drug Development Pipeline





Nichols et al Lancet Health Long 2023

Timely, Accurate Diagnosis Matters

Medical

- Slow disease progression
- Manage symptoms
- New medications, depending on disease type

Personal

- Safety (see later slide)
- Legal matters

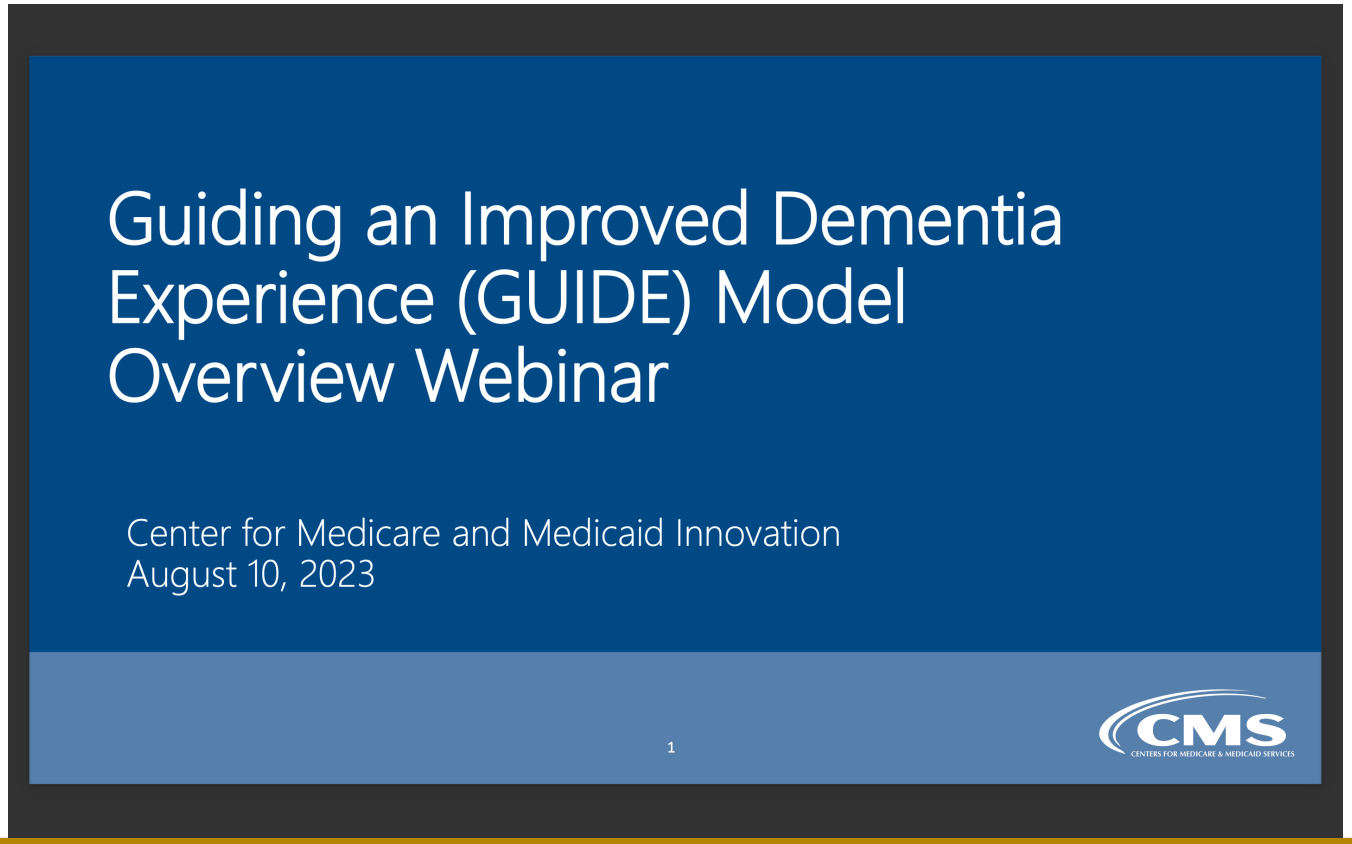
Helpful and not Harmful Care

- Access to **specialized programs** for person and family
- Home environment setup
- Avoidance of unnecessary hospital

Dementia Care

FOR THE 90%

imc



Coming to a practice near you

July 2024 and July 2025

What is GUIDE?

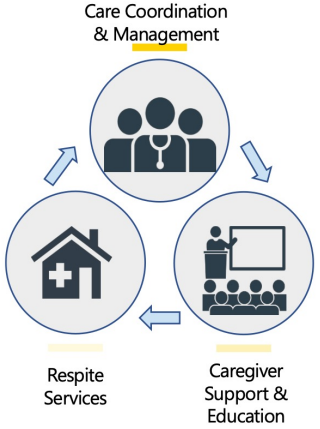
The Centers for Medicare & Medicaid announced the Guiding an Improved Dementia Experience (GUIDE) model on July 31st, 2023.

- Overall Goal
  - Test whether providing an alternative payment methodology for participating dementia care programs reduces expenditures while preserving or enhancing the quality of care.

- Measures
  - Improved quality of life for PLWD and caregivers
  - Deprescribing low-value treatments
  - Reduced total cost of care
  - Delayed nursing home placement

Model Purpose and Overview

The GUIDE Model will test whether a comprehensive package of care coordination and management, caregiver support and education, and respite services can **improve quality of life for people with dementia and their caregivers** while **delaying avoidable long-term nursing home care** and **enabling more people to remain at home** through end of life.



Care Coordination & Management

Beneficiaries will receive care from an **interdisciplinary team** that will develop and implement a comprehensive, person-centered care plan for **managing the beneficiary's dementia and co-occurring conditions** and provide **ongoing monitoring and support.**

Caregiver Support & Education

GUIDE participants will provide a **caregiver support program**, which must include caregiver skills training, dementia diagnosis education, support groups, and access to a personal care navigator who can help problem solve and connect the caregiver to services and supports.

Respite Services

A subset of beneficiaries in the model will be eligible to receive payment for respite services with no cost sharing, up to a cap of **$2,500 per year**. These services may be provided to beneficiaries in a variety of settings, including **their personal home, an adult day center, and facilities that can provide 24-hour care** to give the caregiver a break from caring for the beneficiary.

CMS CENTERS FOR MEDICARE & MEDICAID SERVICES
CENTER FOR MEDICARE & MEDICAID INNOVATION

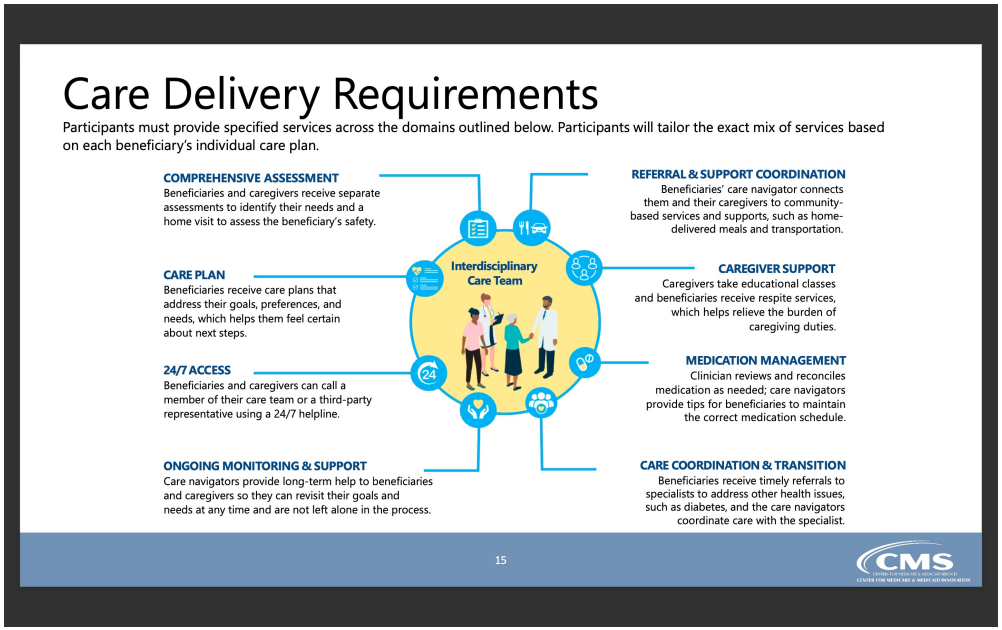
GUIDE Model Components

Team
- Dementia proficient clinician
- Dementia care navigator

Care Coordination and Management
- Dementia and co-occurring conditions
- Ongoing monitoring and support

Caregiver Education and Support
- Skills training
- Diagnosis education
- Support groups

Respite
- Up to $2500/year cap
- At home, adult day, or facility-based



Core Elements

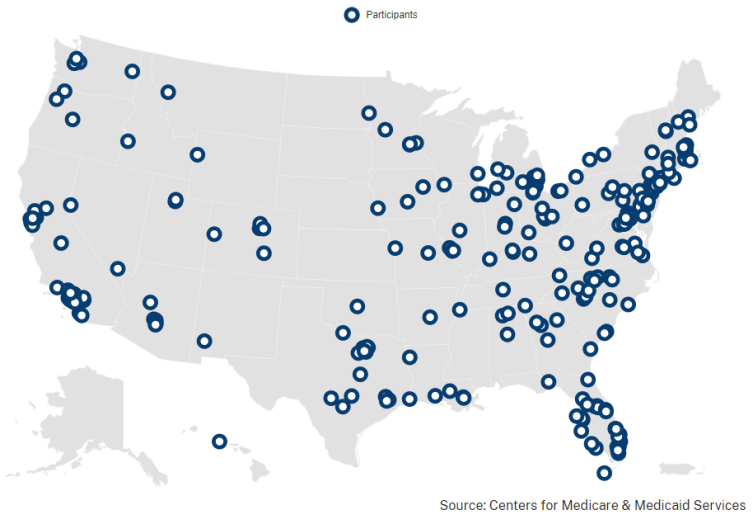
- Comprehensive Assessment
- Written Care Plan
- 24/7 Access to the Clinical Team
- Ongoing Monitoring and Support
- Care Coordination and Transitional Care Management
- Referral and Coordination of Services and Supports
- Medication Management and Reconciliation
- Caregiver Education and Support

IMC Model Components

Primary Care	Dementia Care	Caregiver Services
Annual wellness visits	Disease and goals of care priorities	Caregiver-only visits with practitioners
Screening and preventive care that is context-appropriate	Cognitive, behavioral, and functional assessments	Psychoeducation classes (Savvy Caregiver)
Chronic disease management	Disclosure/clarification of diagnosis and stage	Stage-based support groups
Deprescribing and Rx management	Anticipatory guidance, advance care planning	Ad hoc dementia education
Minor acute illness management	Coordination with adult day, assisted living, home care	Psychotherapy and coaching
Post-hospital follow-up	Customized companion care	Facilitated family therapy/meetings
Coordination with home health, specialists	After hours direct access	

Timeline

- **Established Track**
  - July 2024
  - <100 participants
- **Developing/New Tracks**
  - July 2025
  - ~300 participants
- **End Date**
  - June 2032



Source: Centers for Medicare & Medicaid Services

Defining GUIDE "Success"

1. Quality of life
2. Caregiver burden
3. Deprescribing
4. Cost of care (total)
5. Long-term care use

Best Performers + 10%

Worst Performers – 3.5%

“Demonstration Project” = Clinical Trial

Data will be monitored against similar patients not enrolled in GUIDE

Determine whether the program will be permanent

APPENDIX

Keeping Someone with Dementia Safe

1. Medications: appropriate prescribing, organization and oversight

2. Driving: sensory issues, CarFit, tracking, driving evaluation, rehabilitation or retirement

3. Finances: care is out of pocket; daily banking, billpay services, major financial decisions and documents

4. Firearms: what matters and how to minimize risk

Lowering Risk for Dementia Symptoms

Sleep—diagnose and treat apnea

Chronic conditions—thyroid, diabetes, blood pressure

Metabolic syndrome—insulin resistance, high cortisol, abdominal obesity

Diet—plant-based, MIND diet

Exercise—aerobic

Social engagement—increases brain hormones, ideal to do the above with a friend