DON'T *MIND* THIS OLD THING: THE AGING BRAIN

Dementia and Alzheimer's disease

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Lecture 3

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1

SECOND CHILDISHNESS

Function

"Second childishness and mere oblivion, sans teeth, sans eyes, sans taste, sans everything."

-William Shakespeare in The Seven Stages of Man

- Historically refers to a natural and inevitable deterioration of the mind
- Common euphemism for "senility"
- Senility based on Latin term for "old man"

2

SECOND CHILDISHNESS

- State was recognized by early Greek philosophers (Pythagoras, Aristotle, Hippocrates)

Normal Aging ≠ Severe mental deterioration

Senility ≠ Dementia

- Today, we recognize that aging can come with some mental decline, but severe decline is usually related to a mental disease.

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DEMENTIA

- Loss in mental abilities severe enough to consistently interfere with daily activates
- Not present since birth
- No alterations in consciousness
- Last more then 6 months

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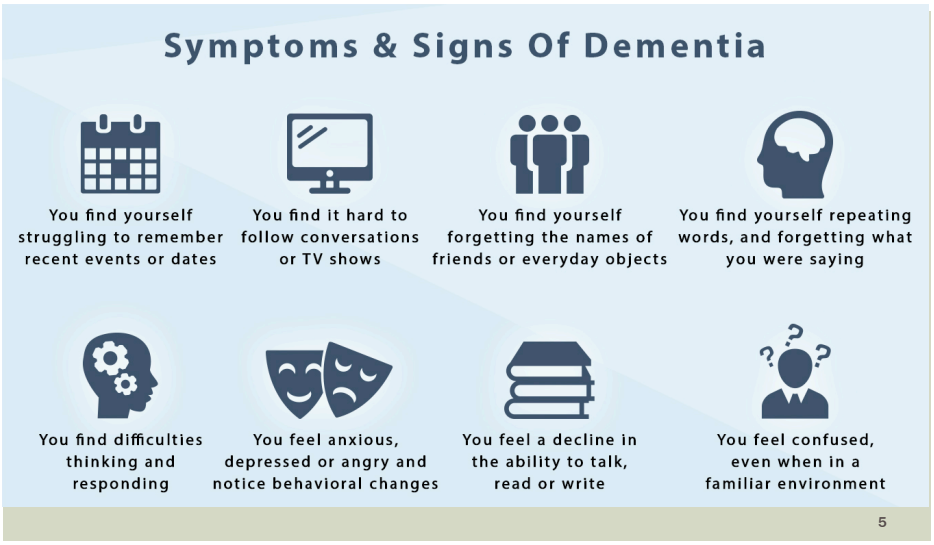
SYMPTOMS OF DEMENTIA



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SYMPTOMS OF DEMENTIA

- First sign is <u>memory loss</u>

Dementia ≠ Occasional Memory loss

- Asking for the same information repeatedly
- Forgetting names of friends and family
- Confusion about directions
- **Trouble remembering what a toothbrush is for**

- Misplacing your keys
- Forgetting where you parked
- Trouble recalling name of an acquaintance
- **Trouble remembering the word for toothbrush**

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WARNING SIGNS OF DEMENTIA

1. Asking for information repeatedly
2. Finding it hard to complete mental tasks that used to be easy
3. Forgetting what season it is
4. Getting lost in a familiar place
5. Difficulty carrying on a conversation
6. Failing to take care of basic hygiene
7. Mood and personality changes

If you or a loved one experiences these symptoms, it is worth consulting a doctor.

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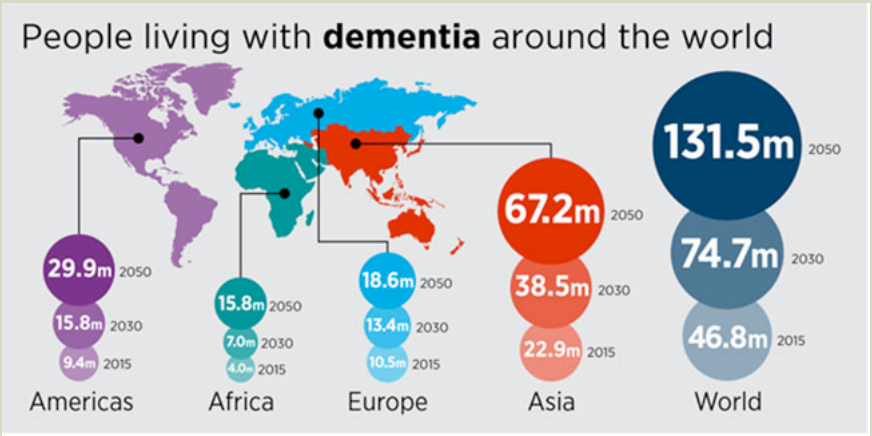
DEMENTIA AS A GLOBAL CONCERN



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TYPES OF DEMENTIA

1. **Temporary (Acute)**: symptoms of dementia can improve or disappear when treated
   - Treatable condition
   - Medication side-effect

2. **Chronic**: dementia occurs due to permanent changes in the brain
   - Progressive
   - Lasts the remaining life time

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TEMPORARY DEMENTIA CAUSES

Some disorders, conditions, and medications can cause dementia-like symptoms. Because these are treatable, symptoms can be reversed.

Conditions

- Depression
- B-12 Deficiency
- Lyme Disease

Medications

- Benzodiazepines
  - Xanax, Ativan, Valium
- Opioid pain killers
  - Vicodin, Percocet
- Beta-blockers
  - Given for heart disease or high blood pressure

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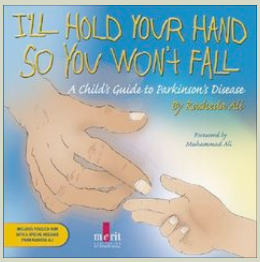
DEMENTIA CAUSED BY NEUROLOGICAL DISEASE

- **Parkinson's disease**
  - Dementia as a non-motor symptom
  - Onset as disease progresses
  - ~50% of idiopathic PD patients develop symptoms of dementia
  - Onset generally 10 years after initial diagnosis

Motor Symptoms Lecture 2



I'll Hold Your Hand So You Won't Fall Rasheda Ali

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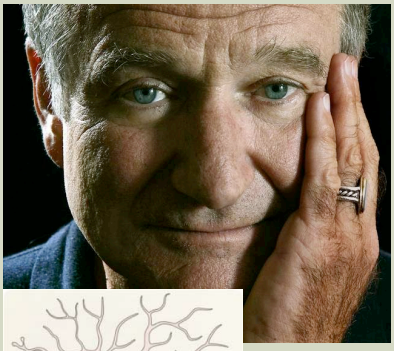
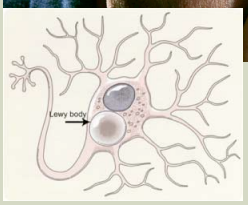
DEMENTIA CAUSED BY NEUROLOGICAL DISEASE

- **Lewy Body Dementia**
  - Lewy bodies (protein clumps of alpha-synuclein) form in neurons in the cortex
  - Early symptoms of sleep disturbances, hallucination, slowness and balance problems (PD)
  - Can co-exist with Alzheimer's and vascular dementia pathology
  - ~15% of dementia cases





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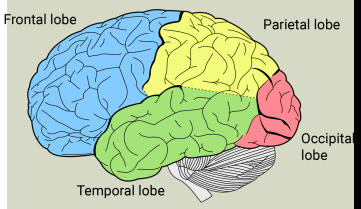
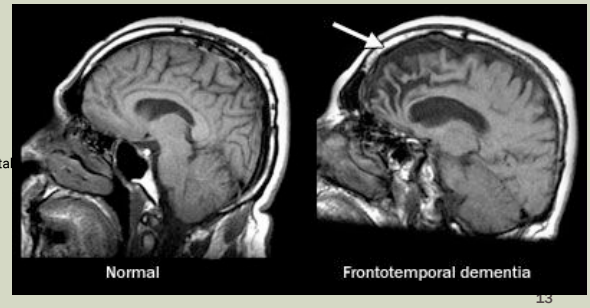
DEMENTIA CAUSED BY NEUROLOGICAL DISEASE

- **Pick's disease or Frontotemporal Dementia**
  - Loss of neurons in the frontal cortex and temporal lobes
  - Significant personality problems early in disease
  - Memory problems at later stages
  - Problems reading and understanding language





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DEMENTIA CAUSED BY NEUROLOGICAL DISEASE

- **Frontotemporal dementia**
  - Diagnosis between ages of 45-65 (21 early, 80 late)
  - Average 6-8 year survival
  - https://www.youtube.com/watch?v=7l4f9nGvmF4
  - https://www.youtube.com/watch?v=gcpzWJgDsKo

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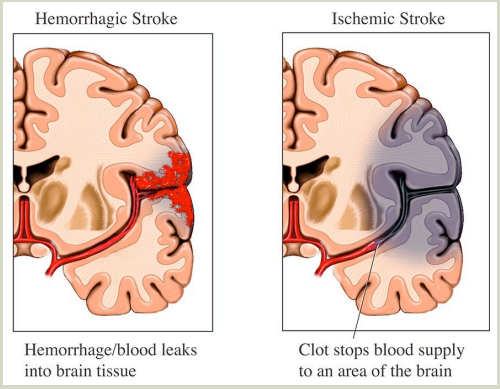
DEMENTIA CAUSED BY NEUROLOGICAL DISEASE

- **Vascular dementia or multi-infarct dementia (MID)**
  - Infarct: Localized area of tissue that dies because of lack of oxygen



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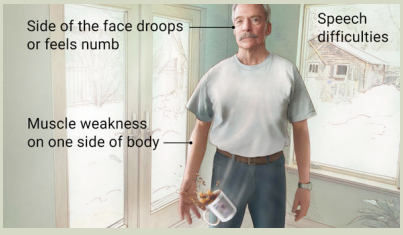
DEMENTIA CAUSED BY NEUROLOGICAL DISEASE

- **Major stroke**: outward signs like trouble moving, face drooping, trouble speaking



- **Minor stroke**: only impacts small part of brain, no outward symptoms
  - "Silent Strokes"
  - Victim may be unaware
  - More common then major strokes
  - Multiple silent strokes → multiple infarcts → accumulation of infarcts → dementia

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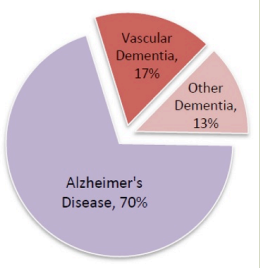
DEMENTIA CAUSED BY NEUROLOGICAL DISEASE

- **Vascular Dementia**
  - Symptoms depend on area of brain affected
  - Risk factors include high blood pressure, high cholesterol, heart disease, diabetes
  - Reduce risk through exercise, not smoking, healthy diet, reducing stress
  - Second most common dementia to Alzheimer's dementia



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PREVALENCE OF DIFFERENT TYPES OF DEMENTIA

Table 1
Characteristics of dementia subtypes

Dementia subtype	Early, characteristic symptoms	Neuropathology	Proportion of dementia cases
Alzheimer's disease (AD) *	Impaired memory, apathy and depression Gradual onset	Cortical amyloid plaques and neurofibrillary tangles	50-75%
Vascular dementia (VaD) *	Similar to AD, but memory less affected, and mood fluctuations more prominent Physical frailty Stepwise onset	Cerebrovascular disease Single infarcts in critical regions, or more diffuse multi-infarct disease	20-30%
Dementia with Lewy Bodies (DLB)	Marked fluctuation in cognitive ability Visual hallucinations Parkinsonism (tremor and rigidity)	Cortical Lewy bodies (alpha-synuclein)	<5%
Frontotemporal dementia (FTD)	Personality changes Mood changes Disinhibition Language difficulties	No single pathology – damage limited to frontal and temporal lobes	5-10%

* Post mortem studies suggest that many people with dementia have mixed Alzheimer's disease and vascular dementia pathology, and that this 'mixed dementia' is underdiagnosed

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ALZHEIMER'S DISEASE (AD)

A. Discovery of AD
B. Progression of the disease
C. Steps you can take to reduce your risk
D. Research for therapies
E. Caregiving

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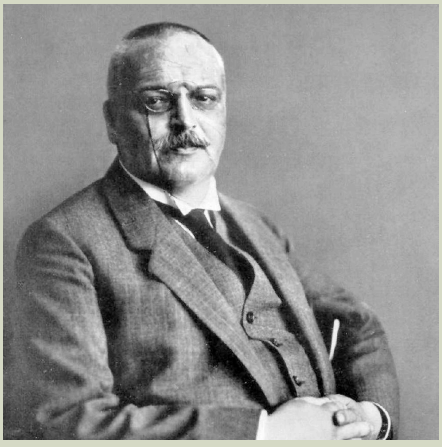
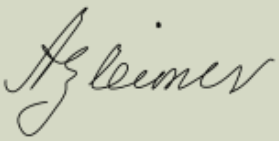
ALOIS ALZHEIMER



- 1864 - 1915
- German Psychiatrist
- Specialized in neuropathology
- Best known for the first published case of "presenile dementia"



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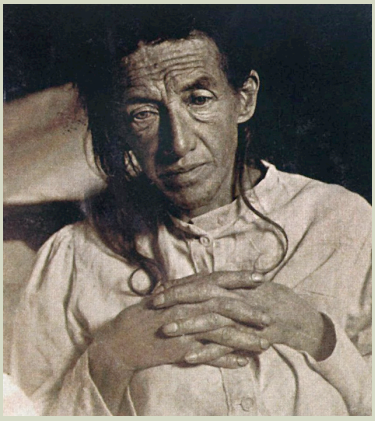
ALZHEIMER AND AUGUSTE DETER

- Nov. 25, 1901 Karl Deter admitted his wife, Auguste Deter to Frankfurt Hospital
- Symptoms
  - Reduced memory and comprehension
  - Disorientation
  - Hallucinations
  - Psychological impairment
  - "I've lost myself"
- Auguste was only 51 years old (she did not have the dementia associated with old age)

Auguste Dieter – Nov. 1902



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ALZHEIMER AND AUGUSTE DETER

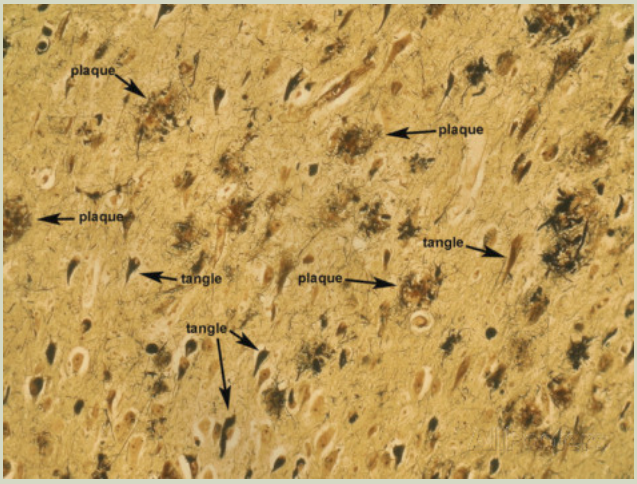
Cover from Auguste Dieter's file

Excerpt from Auguste's file on Nov. 29, 1901

Question	August's Answer
What year is it?	1800.
Are you ill?	Second month.
What month is it now?	The 11th.
What is the name of the 11th month?	The last one, if not the last one.
Which one?	I don't know.
What color is snow?	White.
What color is soot?	Black.
The sky?	Blue.
How many fingers so you have?	Five.
Eyes?	Two.

ALZHEIMER AND AUGUSTE DETER

- (1906) Alzheimer examined her brain
- Histopathology
  1. **Plaques**
  2. **Tangles**



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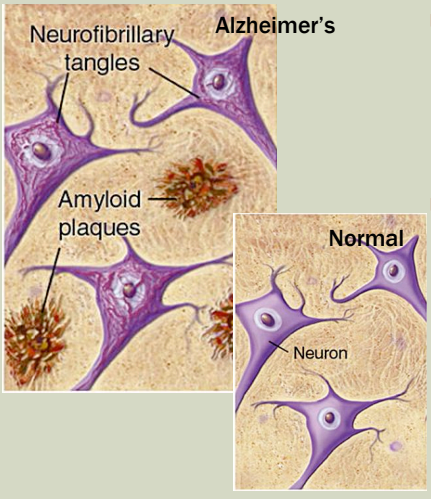
HALLMARKS OF AD



- **Amyloid Plaques**
  - Clumps of protein fragments
  - Between neurons
  - Amyloid Beta protein
- **Neurofibrillary Tangles**
  - Inside of neurons
  - Tau protein
  - Other tauopathies

Kill Neurons and Damages Connections

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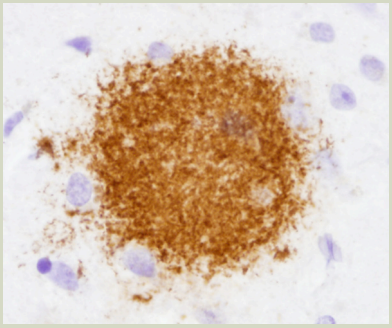
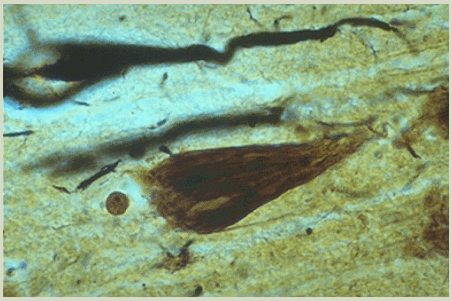
PLAQUES AND TANGLES

- Finding plaques and tangles in the brain is the ONLY way AD can be definitively diagnosed
- "Probable Alzheimer's" or "Dementia of the Alzheimer's type"





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PROGRESSION OF AD

- Progresses at different rates in different individuals
- No treatments that can slow or stop disease
- Average lifespan is 4-8 years after initial diagnosis (some patients live significantly longer)
- AD is the most common cause of dementias

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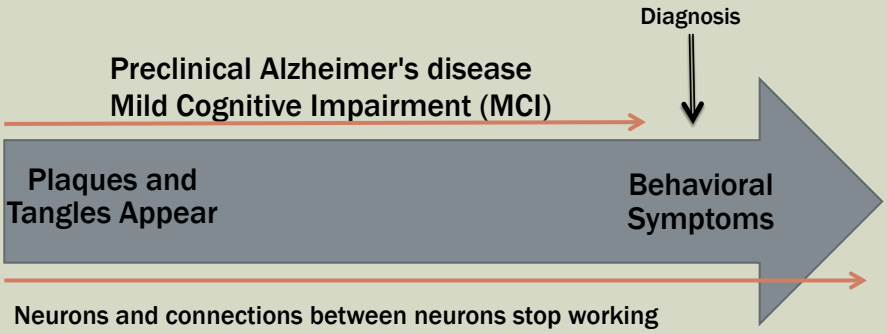
PROGRESSION OF AD

- Symptoms appear many years after plaques and tangles appear in the brain



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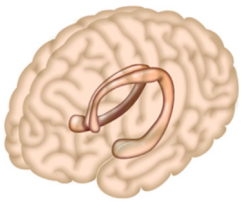
HIPPOCAMPUS FIRST AFFECTED IN AD

- *Lecture 1 – hippocampus shrinks in healthy aging*
  - *Hippocampus crucial role in episodic memory*



Natural shrinking + Accumulation of plaques and tangles

Significant memory problems
First signs of AD are substantial memory deficits

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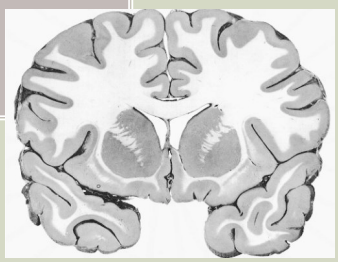
PROGRESSION IN THE BRAIN

Hippocampus plaques and tangles

Memory Deficits

Cerebral cortex plaques and tangles

Behavioral Problems



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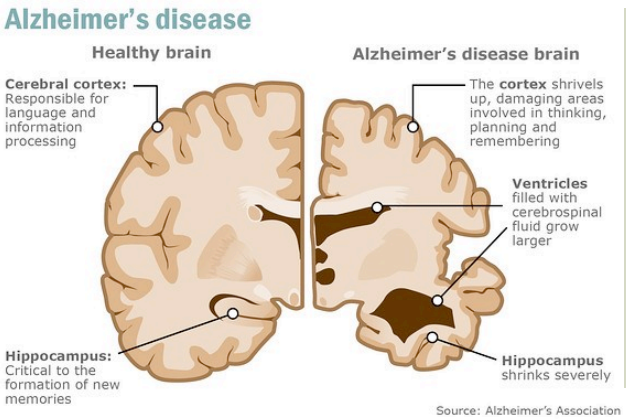
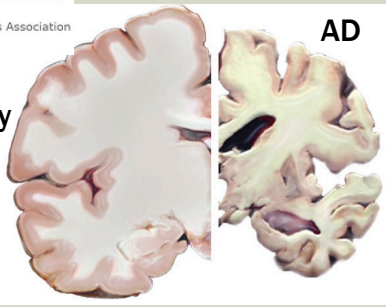
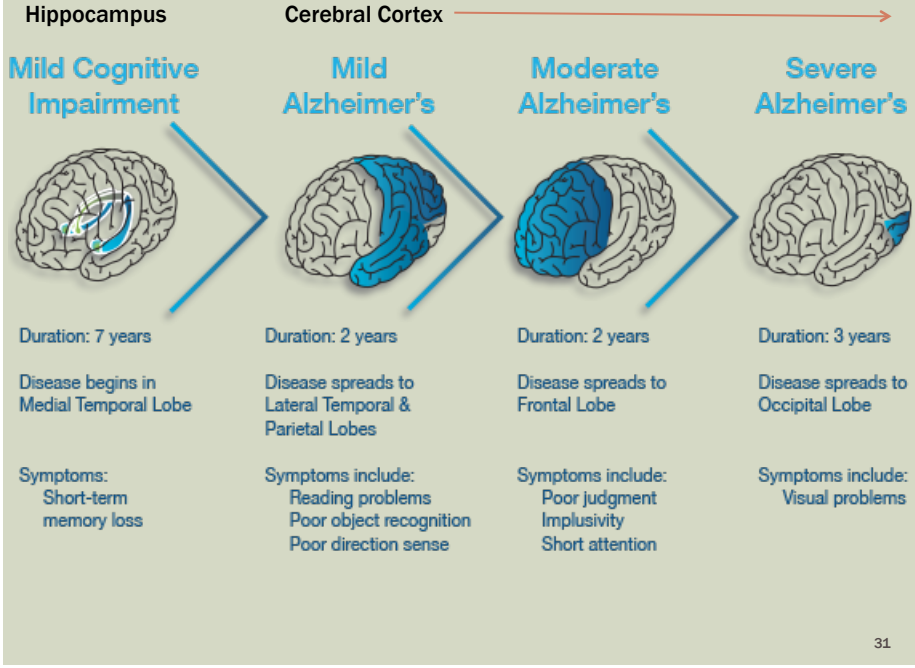


AD

Healthy




Hippocampus
Cerebral Cortex
Mild Cognitive Impairment
Mild Alzheimer's
Moderate Alzheimer's
Severe Alzheimer's
Duration: 7 years
Disease begins in Medial Temporal Lobe
Symptoms:
Short-term memory loss
Duration: 2 years
Disease spreads to Lateral Temporal & Parietal Lobes
Symptoms include:
Reading problems
Poor object recognition
Poor direction sense
Duration: 2 years
Disease spreads to Frontal Lobe
Symptoms include:
Poor judgment
Implusivity
Short attention
Duration: 3 years
Disease spreads to Occipital Lobe
Symptoms include:
Visual problems
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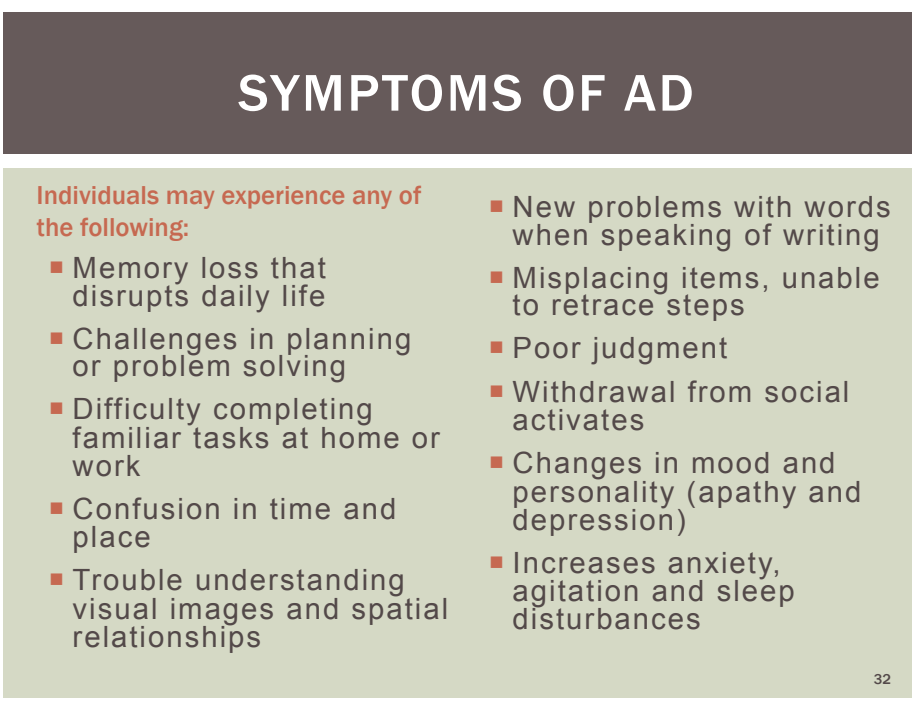
SYMPTOMS OF AD
Individuals may experience any of the following:
Memory loss that disrupts daily life
Challenges in planning or problem solving
Difficulty completing familiar tasks at home or work
Confusion in time and place
Trouble understanding visual images and spatial relationships
New problems with words when speaking of writing
Misplacing items, unable to retrace steps
Poor judgment
Withdrawal from social activates
Changes in mood and personality (apathy and depression)
Increases anxiety, agitation and sleep disturbances
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NON-MODIFABLE RISK FACTORS FOR AD

Genetic + Environmental

Non-modifiable risk factors:

- Age - Age is greatest risk factor, but age alone is not sufficient to cause the disease.
- Family history – A relative with AD increases your chance of developing AD.
- Genetic mutations - mutation in the amyloid precursor protein (APP) gene, presenilin 1 or presenilin 2 genes, apolipoprotein E gene

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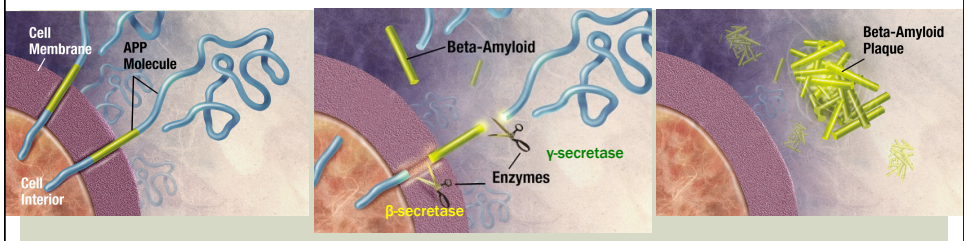
AMYLOID PRECURSOR PROTEIN (APP)



- Normal function not understood
- Implicated as a regulator of synapses formation and neural plasticity
- Enzymes cut APP → Aβ protein fragment found in plaques
- Mutations are guaranteed to develop AD early in life

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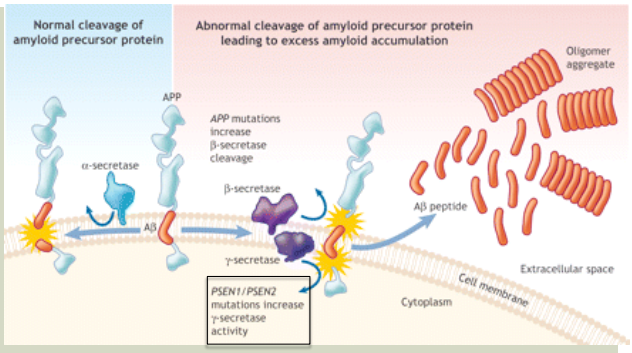
PRESENILIN 1 AND 2

- PSEN1 and PSEN2
- Normally, presenilin proteins regulate APP processing through regulating γ-secretase action



- Mutations allow γ-secretase to be more active then usual
- Cleavage of APP protein and increased Aβ
- Risk of developing AD increased by 95%
- Early diagnosis

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APOPLIPOPROTEIN E (APOE)

- APOE influences how likely it is for amyloid plaques to form because it transports Aβ fragments out of neurons
- 3 forms – e2, e3, and e4
- *e4 is the worst* at transporting Aβ out of neurons

TABLE 2 Estimated Percentages of the U.S. Population with the e2, e3 and e4 Forms of the Apolipoprotein E (APOE) Gene*

APOE Form	Percentage
e2/e2	0.5
e2/e3	11
e2/e4	2
e3/e3	61
e3/e4	23
e4/e4	2

- Greatest genetic risk factor for late-onset AD
- 1 copy of e4 gives 3x higher risk
- 2 copies of e4 gives 8-12x higher risk
- Develop AD at a younger age
- Does not guarantee AD development

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GENETIC TESTING FOR AD

- Blood test can determine which version of APOE a person has
- CAN NOT determine if a person will get AD
- Unlikely that a genetic test will ever predict with 100% accuracy because many factors are at play
- Genetic testing primarily used in research studies
- Genetic testing often not recommended
- Ethical and moral concerns

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THE HUNT FOR GENETIC PREDICTORS

Major Alzheimer's Genetics Research Efforts Underway

The National Institute on Aging supports several major genetics research programs.

- The **Alzheimer's Disease Sequencing Project** (ADSP) is an innovative collaboration between NIA and the National Human Genome Research Institute, both part of NIH. The first phase of the project determined the order of all 3 billion letters in the individual genomes of 580 participants. It also generated whole exome sequencing data for an additional 11,000 volunteers.
- The **Alzheimer's Disease Genetics Consortium** is a collaborative effort to collect and analyze genetic data from thousands of families around the world to identify genes associated with an increased risk of developing late-onset Alzheimer's.
- The **Late-Onset Alzheimer's Disease Genetics Study** is gathering and analyzing genetic and other information from 1,500 or more families in the United States with two or more members who have late-onset Alzheimer's.
- The **International Genomic Alzheimer's Project** (IGAP) is comprised of four consortia in the United States and Europe that have been working together since 2011 on genome-wide association studies (GWAS) involving thousands of DNA samples and shared data sets. In a study of more than 74,000 individuals, IGAP recently reported the identification of 19 novel regions of interest that are associated with the disease.
- The **Genetics of Alzheimer's Disease Data Storage Site** (NIAGADS) is a national genetics data repository that gives investigators access to data to study the genetics of late-onset Alzheimer's disease.
- The **National Cell Repository for Alzheimer's Disease** (NCRAD) is a national resource that helps researchers find genes that increase the risk of Alzheimer's by providing biological samples and data.

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MODIFIABLE RISK FACTORS FOR AD

Modifiable risk factors:

- Cardiovascular disease risk – health of the brain closely linked to health of heart and blood vessels
  - Smoking
  - Midlife obesity
  - Diabetes
  - Hypertension
  - High Cholesterol

REGULAR EXERCISE

HEALTHY DIET

PHYSICAL ACTIVITY AND AD

- Kivipelto and Rovio, Karolinska Institute
- Does physical activity during middle age have any impact on the development of AD?

Active Group

Physical activity: at least twice weekly activity for 20 minutes or more that causes breathlessness and sweating (n=515)

Sedentary Group

Did no or less then 2x week physical activity (n=736)

Evaluated for AD at age 65 (or 20 years later)

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PHYSICAL ACTIVITY AND AD

Table 1.
Sociodemographic and clinical characteristics of the participants according to the midlife leisure time physical activity

	Active (n=515)	Sedentary (n=736)	p
Demographics			
Age at midlife (years)	50·8 (6·1)	49·5 (5·8)	<0·001
Age at re-examination (years)	71·5 (4·0)	70·9 (3·9)	0·08
Follow-up time (years)	20·7 (5·0)	21·3 (4·7)	0·02
Education (years)	8·7 (3·6)	8·7 (3·4)	0·96
Men : women	228 (44·3%) : 287 (55·7%)	265 (36·0%) : 471 (64·0%)	0·003
APOE ε4 carriers	171 (33·2%)	267 (36·3%)	0·26
Midlife measurements			
Systolic blood pressure (mm Hg)	144·0 (19·2)	143·8 (20·3)	0·42
Diastolic blood pressure (mm Hg)	89·3 (10·3)	89·2 (11·3)	0·93
Body-mass index (kg/m^2)	26·5 (3·7)	26·4 (3·6)	0·75
Total serum cholesterol (mmol/L)	6·7 (1·2)	6·7 (1·2)	0·98
History of locomotor disorders	150 (29·1%)	215 (29·2%)	0·97
Re-examination measurements (late-life)			
Dementia	15 (2·9%)	38 (5·2%)	0·05
Alzheimer's disease*	10/510 (2·0%)	31/729 (4·3%)	0·026
History of diabetes mellitus	40 (7·8%)	37 (5·0%)	0·047
History of stroke	32 (6·2%)	61 (8·3%)	0·17
History of myocardial infarction	79 (15·3%)	98 (13·3%)	0·31
Smokers	234 (45·4%)	325 (44·2%)	0·65
Alcohol drinkers	380 (73·8%)	532 (72·3%)	0·56

- Investigated and controlled for other risk factors
- 4-5% of sedentary individuals developed AD
- 2% of active individuals developed AD
- Sedentary people are more then 2x likely to develop AD compared to active people

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MODIFIABLE RISK FACTORS FOR AD

- Education – people with fewer years of formal education are at higher risk of developing AD compared to those with more years of formal education.

- *Cognitive reserve hypothesis* – having more education enables individuals to best compensate for brain changes
  - Connectivity between neurons better
  - Use alternate routes of neuron-to-neuron communication
- Higher educated likely to have occupations that are more mentally stimulating
- Nutrition and other resources

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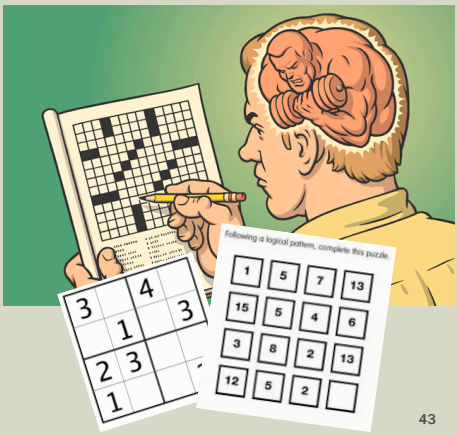
MODIFIABLE RISK FACTORS FOR AD

- Social and Cognitive Engagement- research suggests that remaining mentally active through life may support brain health



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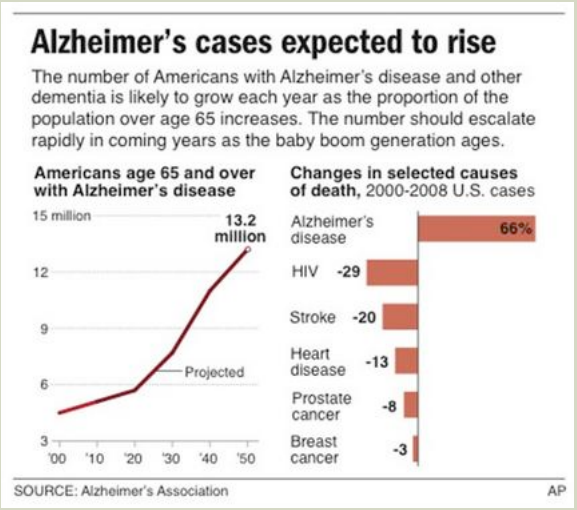
ALZHEIMER'S IN THE U.S.



- Numbers of individuals with AD is expected to rise as our population ages
- Currently 5.4 million American's living with AD

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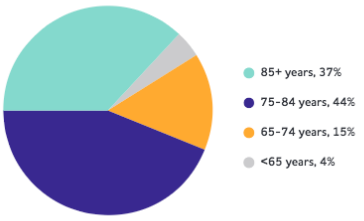
ALZHEIMER'S IN THE U.S.

- 6th leading cause of death in the United States
- 1 in 9 people 65+ has AD
- About one-third of people 85+ have AD
- Affects women more then men
- Highest in people of Hispanic origin, then African-Americans, then Caucasians

FIGURE 1 Ages of People with Alzheimer's Disease in the United States, 2016



Created from data from Hebert et al.[33,44]

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ALZHEIMER'S IN THE U.S.

TABLE 3 Projections of Total Numbers of Americans Age 65 and Older with Alzheimer's by State

State	Projected Number with Alzheimer's (in thousands) 2016	2025	Percentage Change 2016-2025	State	Projected Number with Alzheimer's (in thousands) 2016	2025	Percentage Change 2016-2025
Alabama	89	110	23.6	Montana	19	27	42.1
Alaska	6.8	11	61.8	Nebraska	33	40	21.2
Arizona	130	200	53.8	Nevada	41	64	56.1
Arkansas	54	67	24.1	New Hampshire	23	32	39.1
California	610	840	37.7	New Jersey	170	210	23.5
Colorado	67	92	37.3	New Mexico	37	53	43.2
Connecticut	74	91	23.0	New York	390	460	17.9
Delaware	17	23	35.3	North Carolina	160	210	31.3
District of Columbia	9.0	9.0	0.0	North Dakota	14	16	14.3
Florida	510	720	41.2	Ohio	210	250	19.0
Georgia	130	190	46.2	Oklahoma	62	76	22.6
Hawaii	26	35	34.6	Oregon	62	84	35.5
Idaho	23	33	43.5	Pennsylvania	270	320	18.5

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NATIONAL PLAN TO ADDRESS ALZHEIMER'S DISEASE:

- On January 4, 2011, President Barack Obama signed into law the **National Alzheimer's Project Act** (NAPA), requiring the Secretary of the U.S. Department of Health and Human Services to establish the National Alzheimer's Project to:
  - Create and maintain an integrated National Plan to overcome Alzheimer's disease.
  - Coordinate Alzheimer's disease research and services across all federal agencies.
  - Accelerate the development of treatments that would prevent, halt, or reverse the course of Alzheimer's disease.
  - Improve early diagnosis and coordination of care and treatment of Alzheimer's disease.
  - Decrease disparities in Alzheimer's disease for ethnic and racial minority populations that are at higher risk for Alzheimer's disease.
  - Coordinate with international bodies to fight Alzheimer's disease globally.

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TREATMENTS FOR AD

- No cure
- No treatments to slow or stop disease progression
- 6 pharmacological treatments (FDA approved) for temporarily improving the symptoms of AD
- All work by increasing the amount of neurotransmitters – chemical messengers- in the brain
- **These drugs do not inhibit progression of underlying disease**

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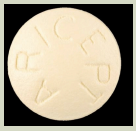
PALLIATIVE TREATMENTS FOR AD

Donepezil

- Early and middle stages
- AD acetylcholine levels drop in early dementia
- Acetylcholine has a role in cognition and memory formation
- Inhibits the breakdown of acetylcholine
- Increases acetylcholine in the brain
- Temporarily improve memory, attention, sleep and mood
- Aricept, similar drugs Galantamine and Rivastigmine



PALLIATIVE TREATMENTS FOR AD

Memantine

- Moderate-Severe stages
- Reduces activity of glutamate in the brain
- Glutamate is an excitatory neurotransmitter
- Too much glutamate can kill already compromised neurons
- Stops excitotoxicity by blocking glutamate
- Improve cognitive function

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AD RESEARCH

- AD was identified more then 100 years ago
- 1970's AD was recognized as the most common cause of dementia and major cause of death
- AD has become a major area of research
- A great deal of research has revealed much about AD
- Much is yet to be done about the precise biological changes in the brain that lead to the disease and how the disease can be slowed and stopped
- Focus in early detection – key to preventing, slowing and stopping AD

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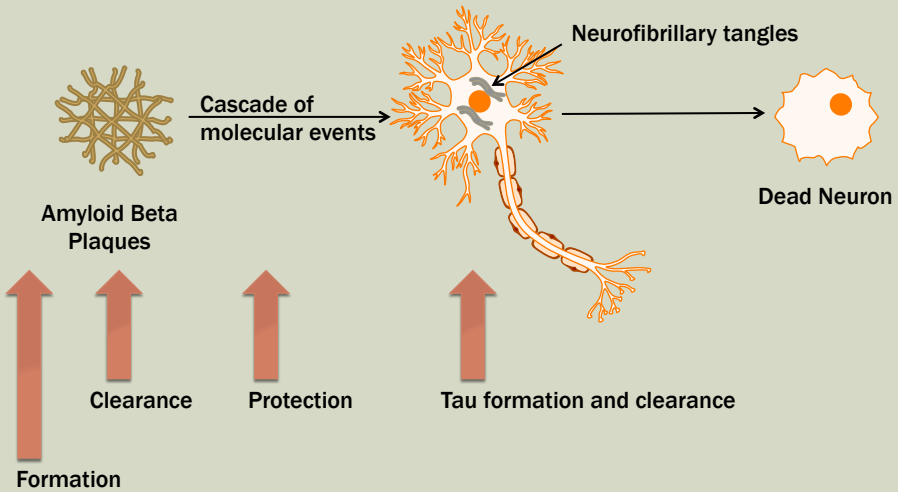
AMYLOID CASCADE HYPOTHESIS



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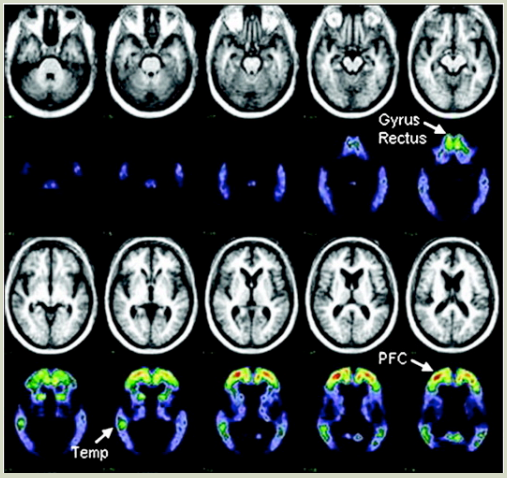
VISUALIZING AMYLOID BETA PLAQUES



- PET scans with radiolabelled molecules that bind to amyloid beta
- Brighter signals correlate to higher amyloid beta accumulation

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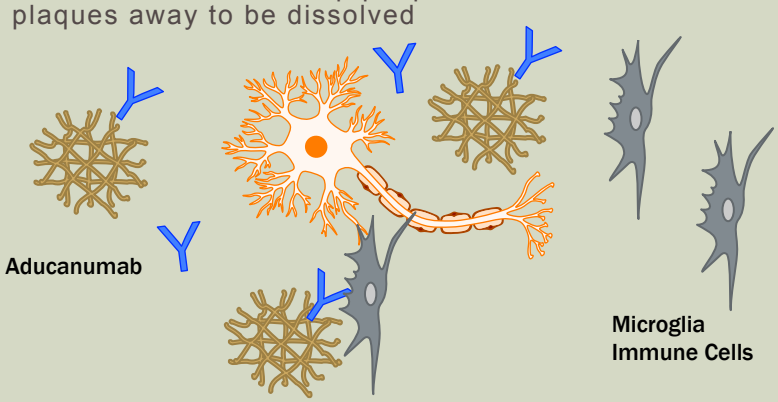
CLEARING AMYLOID BETA PLAQUES

- **Aducanumab** – human antibody to Aβ fragments
- Immunotherapy
- Aducanumab binds to Aβ plaques and immune cells carry plaques away to be dissolved



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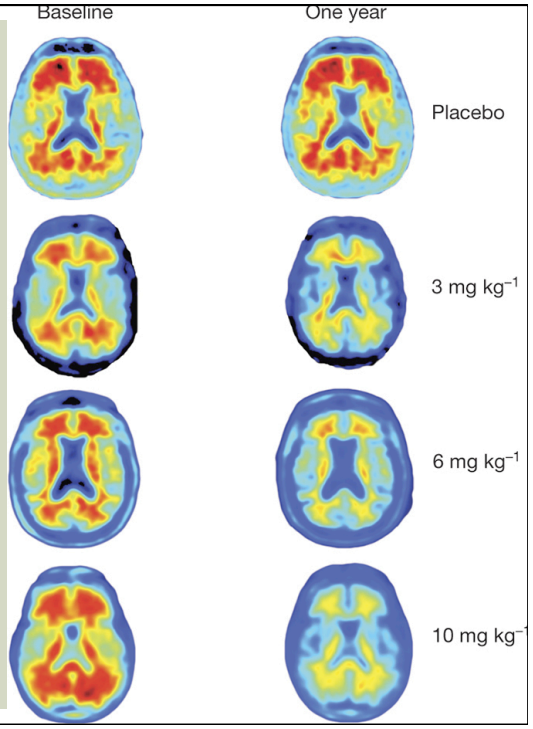
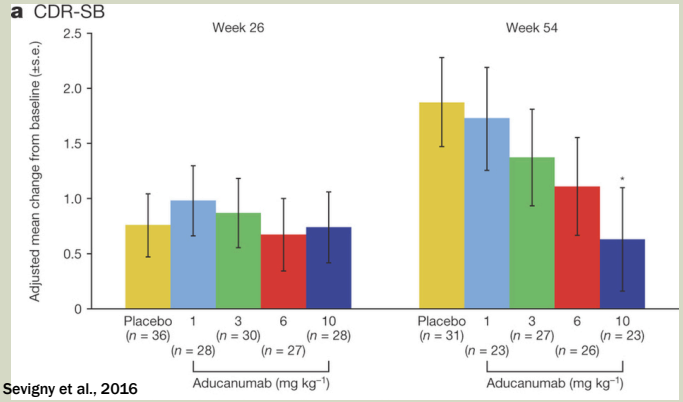
- Aducanumab given to <u>early-stage AD patients in clinical trial</u>
- Plaques form 10-15 years prior to symptom onset
- <u>Clear plaques already present and stop new ones from forming</u>
- Treatment and placebo group
- After 1 year treatment, there was <u>almost a complete clearance of amyloid plaques</u> in the brain

Sevigny et al., 2016



CLEARING AMYLOID BETA PLAQUES

- AD participants treated with Aducanumab experienced **less cognitive decline** over time compared to the placebo group



Two Phase 3 clinical trials ongoing

Sevigny et al., 2016

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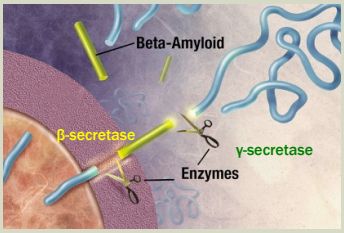
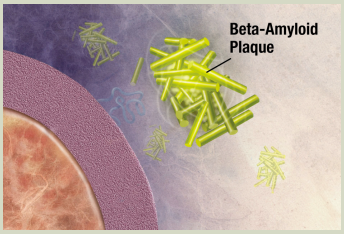
STOPPING BETA AMYLOID FROM FORMING

- **AZD3293**- oral β-secretase inhibitor
- Overactive β-secretase cleaves amyloid precursor protein to form Aβ fragments
- Inhibit Aβ fragment formation, inhibit plaque formation
- AZD3239 reduces Aβ in AD and healthy people
- Moving onto Phase 3 trial
- Fast-tracked by the FDA for faster approval





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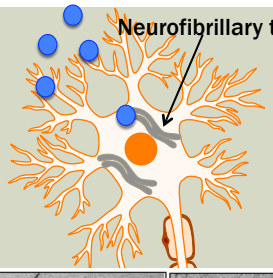
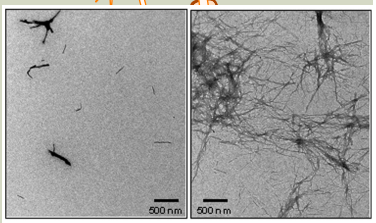
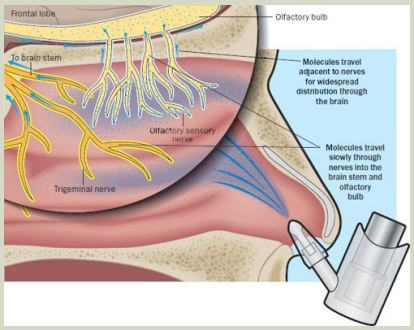
DISSOLVING TAU AGGREGATES

- **LMTX** – Tau protein aggregation inhibitor
- Methylene Blue (methylthioninium chloride) technology
- Tested for AD and FTD
- Reduced Tau aggregation
- Dissolves existing aggregates
- Mild-Moderate AD
- Results of 2 large Phase 3 clinical trials expected this month





STUDY OF NASAL INSULIN TO FIGHT FORGETFULNESS (SNIFF)



- Insulin carries out multiple functions in the brain
- Hormone with receptors in the brain
- Insulin dysregulation may contribute to the development of AD
- Measure effects on cognition and neuron atrophy
- Patients will be given therapy and followed for 12 months (Phase II/III)

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CAREGIVERS FOR AD PATIENTS

- 15 million Americans provide unpaid care for people with AD and other dementias
- 2015- caregivers provided an estimated 18.1 billion hours of care
- Family members, friends, unpaid caregivers
- 1/3 caregivers are 65 and older
- 2/3 of caregivers are married, living with a partner or in a long-term relationship
- ~250,000 of children and young adults between 8 and 18 provide someone with AD
- 23% of caregivers care for an aging parent and children under 18

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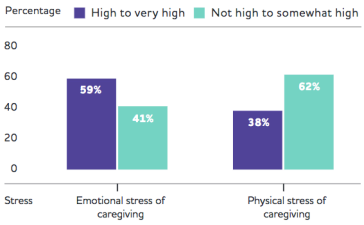
CAREGIVERS FOR AD PATIENTS

- For some caregivers, the demands of caregiving can cause declines in their own health
- Caregiver suffer emotional and physical difficulties
- 95% report the emotional stress high to very high
- 40% suffer from depression
- 74% report concern about maintaining your own heath
- As AD progresses, caregivers become on duty 24 hours everyday
- 2014- poll women reported caring for AD patients is more difficult then caring for children

FIGURE 8 Proportion of Alzheimer's and Dementia Caregivers Who Report High or Very High Emotional and Physical Stress Due to Caregiving



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Interventions to assist caregivers – there is help!

TABLE 8 Type and Focus of Caregiver Interventions

Type of Intervention	Description
Case management	Provides assessment, information, planning, referral, care coordination and/or advocacy for family caregivers.
Psychoeducational	Includes a structured program that provides information about the disease, resources and services, and about how to expand skills to effectively respond to symptoms of the disease (that is, cognitive impairment, behavioral symptoms and care-related needs). Includes lectures, discussions and written materials and is led by professionals with specialized training.
Counseling	Aims to resolve pre-existing personal problems that complicate caregiving to reduce conflicts between caregivers and care recipients and/or improve family functioning.
Support groups	Less structured than psychoeducational or therapeutic interventions, support groups provide caregivers the opportunity to share personal feelings and concerns to overcome feelings of social isolation.
Respite	Provides planned, temporary relief for the caregiver through the provision of substitute care; examples include adult day services and in-home or institutional respite for a certain number of weekly hours.
Psychotherapeutic approaches	Involve the establishment of a therapeutic relationship between the caregiver and a professional therapist (for example, cognitive-behavioral therapy for caregivers to focus on identifying and modifying beliefs related to emotional distress, developing new behaviors to deal with caregiving demands, and fostering activities that can promote caregiver well-being).
Multicomponent approaches	Are characterized by intensive support strategies that combine multiple forms of interventions, such as education, support and respite into a single, long-term service (often provided for 12 months or more).

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CAREGIVERS BILL OF RIGHTS

A Caregiver's Bill of Rights
By Jo Horne

I have the right:

Suggested addition by audience member

- To take care of myself. This is not an act of selfishness. It will give me the capability of taking better care of my loved one.
- To seek help from others even though my loved ones may object. I recognize the limits of my own endurance and strength.
- To maintain facets of my own life that do not include the person I care for, just as I would if he or she were healthy. I know that I do everything that I reasonably can for this person, and I have the right to do some things just for myself.
- To get angry, be depressed, and express other difficult feelings occasionally.
- To reject any attempts by my loved one (either conscious or unconscious) to manipulate me through guilt, and/or depression.
- To receive consideration, affection, forgiveness, and acceptance for what I do, from my loved ones, for as long as I offer these qualities in return.
- To take pride in what I am accomplishing and to applaud the courage it has sometimes taken to meet the needs of my loved ones
- To protect my individuality and my right to make a life for myself that will sustain me in the time when my loved one no longer need my full-time help.
- To expect and demand that as new strides are made in finding resources to aid physically and mentally impaired persons in our country, similar strides will be made towards aiding and supporting caregivers.

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AD RESOURCES

- Alzheimer's Association www.alz.org
- National Institute of Aging www.nia.nih.gov
- Alzheimer's Foundation of America www.alzfdn.org
- OSU https://wexnermedical.osu.edu/brain-spine-neuro/memory-disorders/alzheimers

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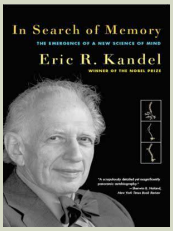
AD RESOURCES

In Search of Memory. Eric Kandel
Description of the search for the biological basis of memory from the neuroscientist Dr. Eric Kandel.



The Man Who Mistook His Wife for a Hat. Oliver Sacks
The stories of individuals afflicted with fantastic perceptual and intellectual aberrations: patients who have lost their memories and with them the greater part of their pasts; who are no longer able to recognize people and common objects; who are stricken with violent tics and grimaces or who shout involuntary obscenities; whose limbs have become alien; who have been dismissed as retarded yet are gifted with uncanny artistic or mathematical talents.