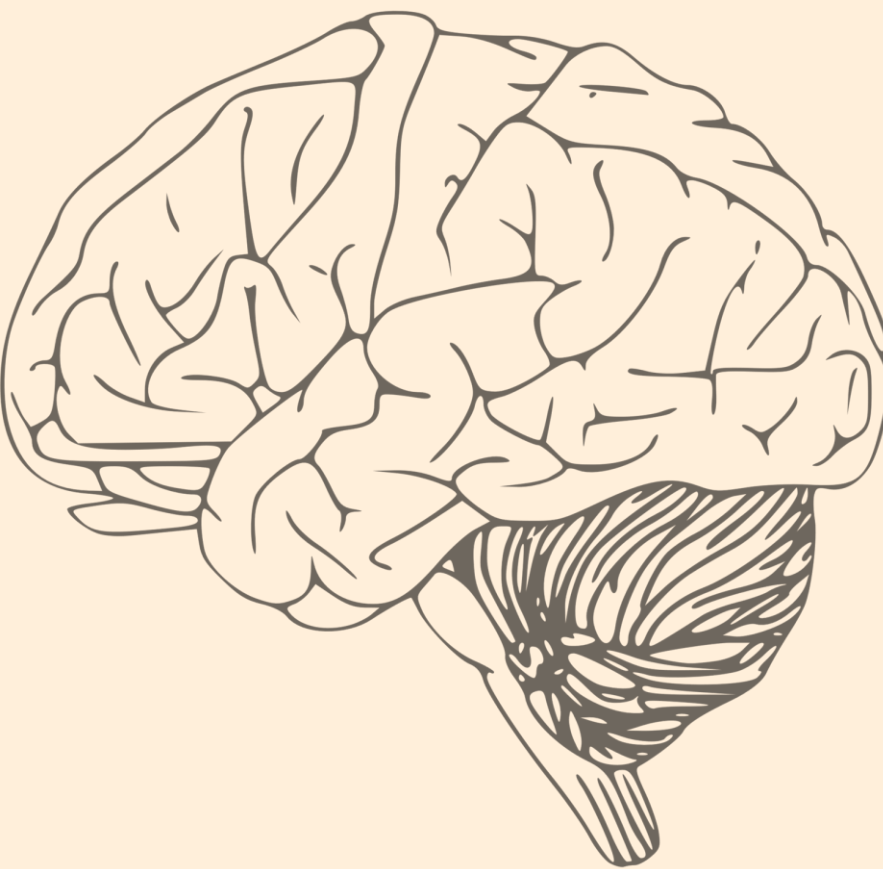# ALZHEIMER'S DISEASE PRESENTATION

Presented by: mohamed nikshara

Supervisor Dr / Elsaeid El-sherbiny

CONTENT

symptoms of alzheimer's
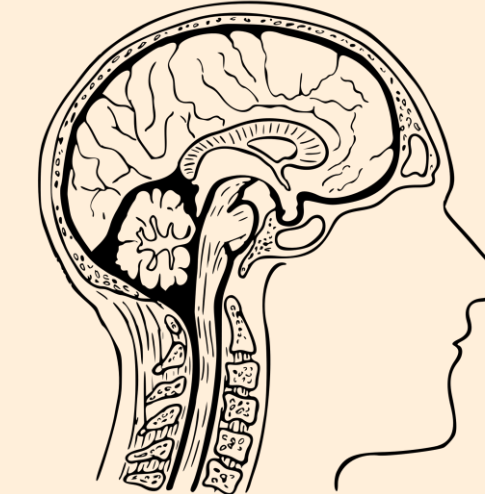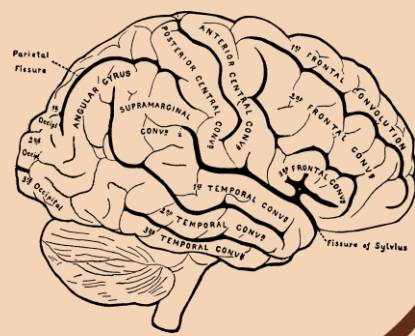
Hypotheses

introduction

conclusion

treatment option

causes and risk factor

Auguste D and Alzheimer's disease
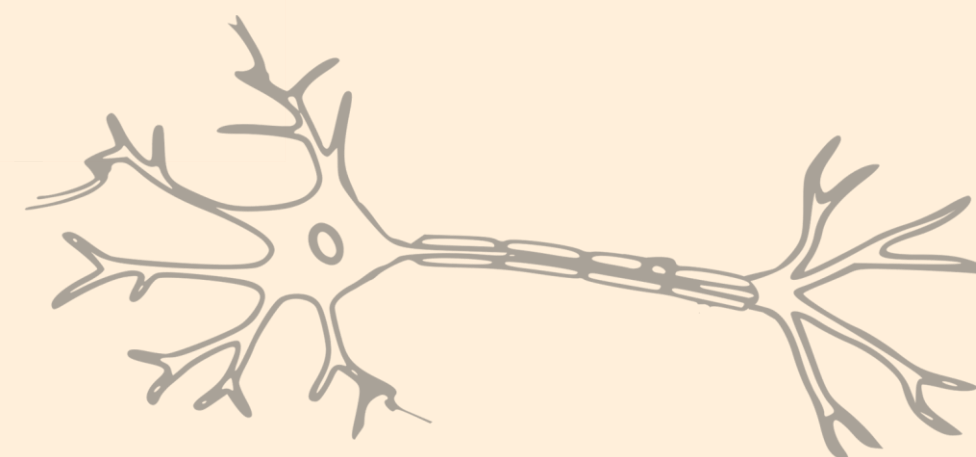
What's is your name?

family name?

what's your husband's name

your husband?

are you married?

what is this? (I show her a pencil)
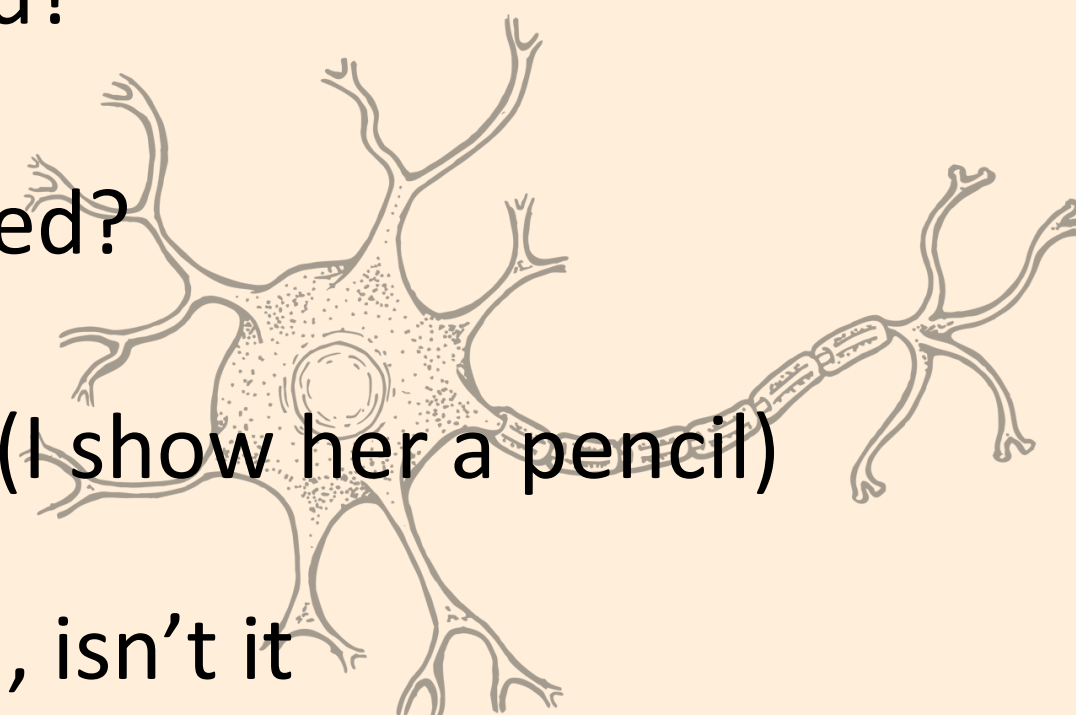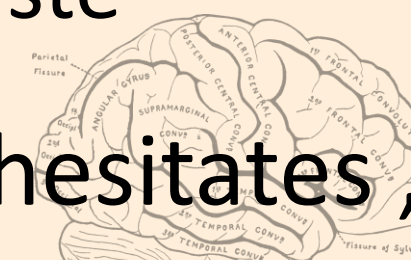
it's a difficult , isn't it

Auguste

Auguste

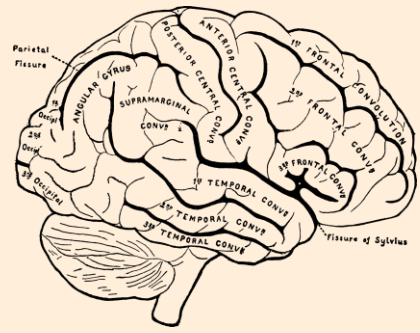she hesitates ,, finally answers I believe .... Auguste

oh ... my husband
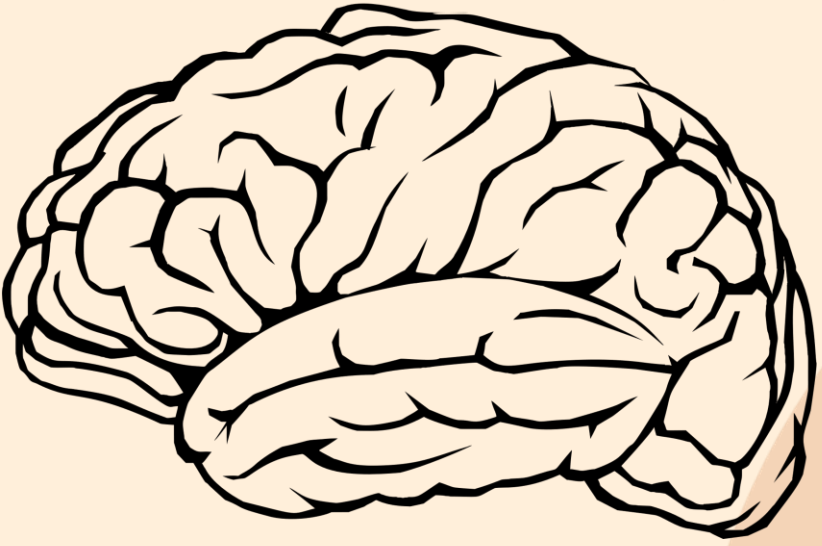
to Auguste

I don't know .... I don't know

so anxious ... so anxious

objects found during the autospy of Augustu's brain

INTRODUCTION

Alzheimer's disease is a progressive neurodegenerative disorder primarily affecting elderly individuals, characterized by cognitive decline, memory loss, and behavioral changes. The disease is marked by the accumulation of amyloid-beta plaques and neurofibrillary tangles in the brain, leading to neuronal damage and death. While genetic factors such as mutations in APP and PSEN genes play a significant role, acquired risk factors like diabetes, hypertension, and lifestyle habits also contribute. Current treatments aim to manage symptoms and slow progression, but ongoing research seeks to uncover effective therapies and preventive measures. Early detection and lifestyle modifications remain critical in mitigating its impact.
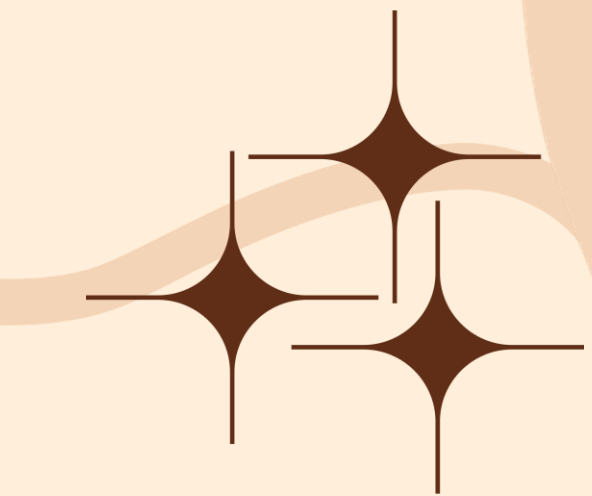
1 / Antegrade episiodic memory deficits

2 / visuospatial variant alzheimer's disease

3 / languge variant alzhiemer disease

4 / Behavooral variant alzheimer disesae

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in Late-Stage Symptoms all these symptoms come together

Hypotheses

tau propagation hypothesis

Amyloid cascad Hypothesis

amyloide bata-peptide tau proteins: It is naturally present in all of our bodies. Some have a function and some do not

beta-amyloid plaque

tau tangle

causes and risk factor

Genetic risk factors

Cerebrovascular

Hypertension

Type 2 diabetes

Obesity

Dyslipidemia

stress, depression

Smoking

Physical activity

Genetic risk factors

Alzheimer's disease (AD) is classified based on the age of symptom onset. Early-onset AD affects individuals under 65 years old, while late-onset AD affects those over 65. Genetics accounts for about 70% of the risk of developing AD. Early-onset AD is often linked to mutations in the APP, PSEN1, and PSEN2 genes, while late-onset AD is mainly associated with the APOE gene, particularly the ε4 allele. The presence of the ε4 allele increases the risk of AD, with a higher risk in homozygotes. APOE plays a role in lipid metabolism and has different effects based on its isoforms; APOE4 promotes the deposition of amyloid-beta (Aβ), while APOE2 and APOE3 help clear it from the brain, potentially protecting against AD

Cerebrovascular disease

erebrovascular diseases and Alzheimer's disease (AD) share many overlapping risk factors. Vascular changes like hemorrhagic infarcts, ischemic cortical infarcts, and white matter changes increase dementia risk. Postmortem studies show that many AD patients also have vascular disease, such as amyloid angiopathy and small vessel disease, with over 50% exhibiting hemorrhages or infarcts. Research suggests that 6-47% of individuals with dementia have concurrent cerebrovascular disease. The "double-stroke" theory of AD proposes that vascular risk factors first impair the blood-brain barrier, reducing blood flow and causing neuronal damage. This dysfunction promotes both non-amyloidogenic and amyloidogenic pathways, leading to amyloid accumulation and further neuronal damage.

Type 2 diabetes

Mechanisms for this connection include insulin resistance, insulin deficiency, hyperglycemia toxicity, and cerebrovascular damage. Animal models have shown that insulin resistance can activate β and γ-secretases, reduce Aβ clearance, and promote its accumulation in the brain. Insulin deficiency also leads to hyperphosphorylation of tau protein, contributing to neurofibrillary tangles (NFTs). Additionally, advanced glycation end products (AGEs) can cause neuronal death, promote Aβ accumulation, and enhance secretase activity, further worsening the neurodegeneration.
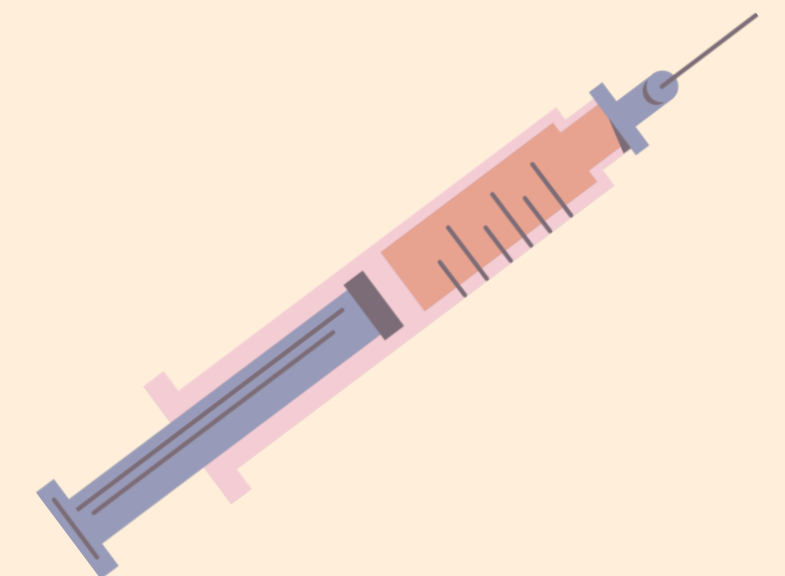
stress and depression

widowed individuals have a higher risk of developing AD, especially those carrying the APOE ε4 allele. Stress, triggered by widowhood, can increase cortisol levels, leading to increased amyloid-beta (Aβ) deposition in the brain and the accumulation of hyperphosphorylated tau, contributing to neurodegeneration. Higher cortisol levels have been linked to faster AD progression and hippocampal atrophy. Depression and sleep disorders also play a role in increasing the risk of AD, with sleep disturbances especially linked to a higher risk of developing dementia. Additionally, insomnia and sleep apnea have been associated with an elevated risk for both AD and vascular dementia.

Smoking

Smoking may increase the risk of developing Alzheimer's disease (AD) through several mechanisms, including the generation of free radicals, increased oxidative stress, and promoting inflammation in the immune system. Additionally, smoking can contribute to cerebrovascular diseases, further elevating AD risk. A meta-analysis by Cataldo et al. (2010) suggested a possible protective effect of smoking against AD, but other studies found an increased risk in smokers. Overall, the evidence supports smoking cessation as a recommendation to reduce the incidence of dementia.
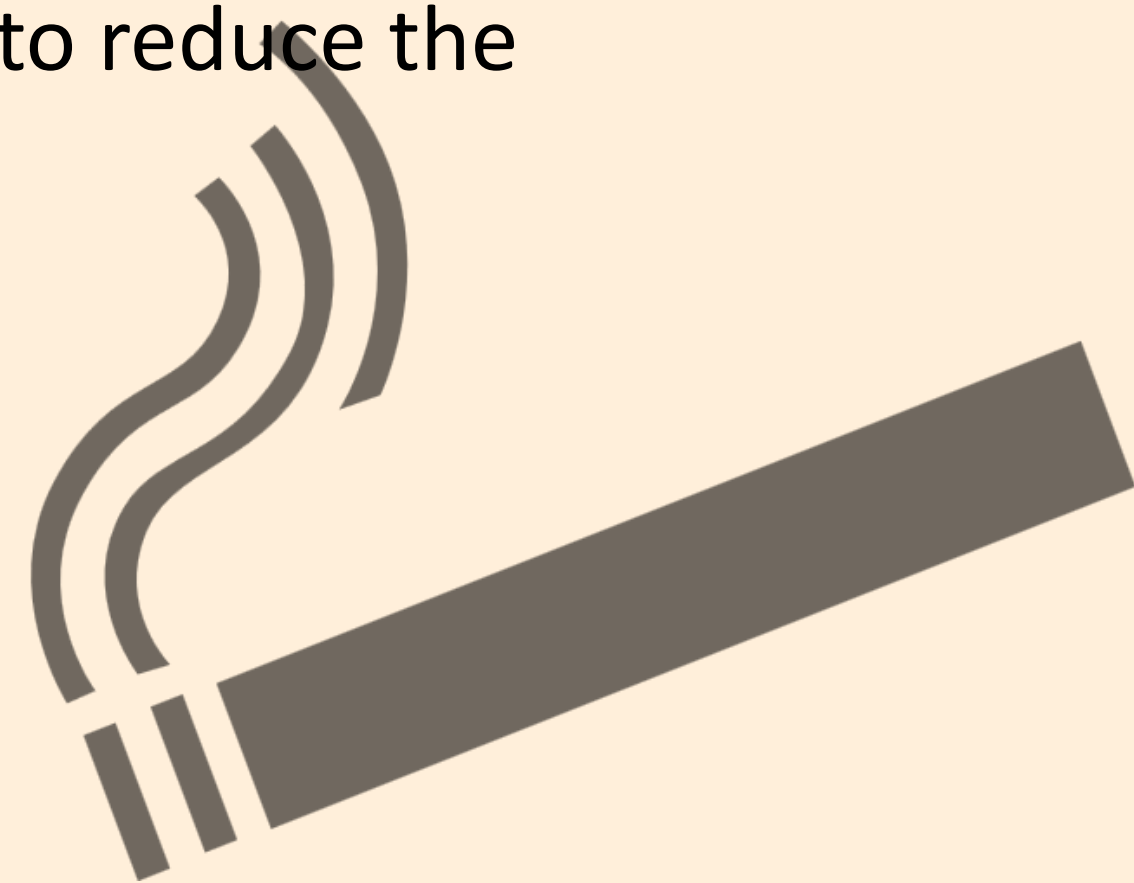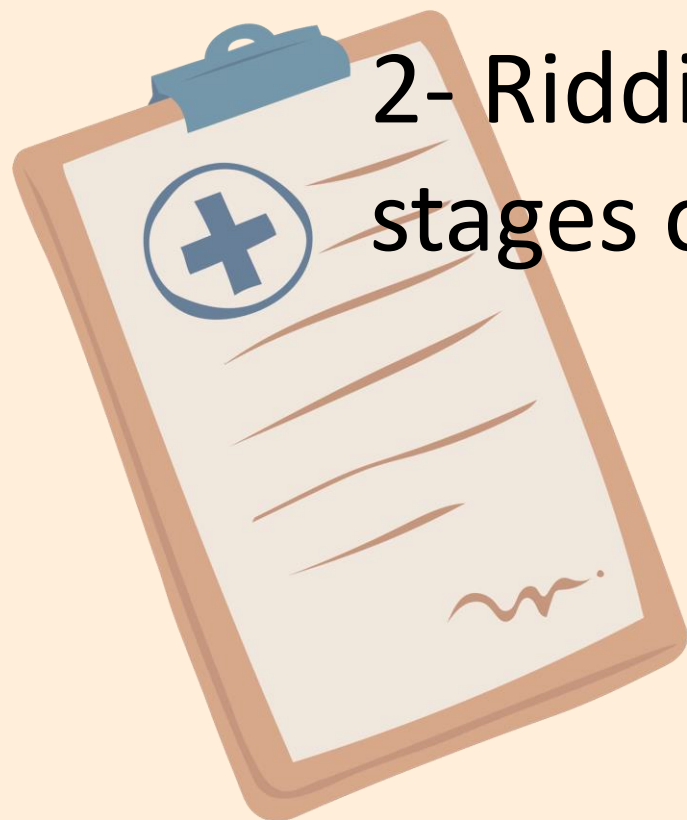
treatment option

for the treatment of Alzheimer's, there is no cure yet, but there are some medications that have proven effective in treating some symptoms

1- Improve electrical conduction between brain cells

2- Ridding the brain of deformed proteins, thus slowing down the stages of disease deterioration

THANK YOU