Support for the Amyloid Cascade Hypothesis in Guiding Further Avenues of Treatment in Alzheimer's Disease

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What is Alzheimer’s Disease?

- Alzheimer’s Disease (AD) is the most common form of dementia
- Neurodegenerative
- Currently around 50 million people affected, expected to rise to 150 million by 2050
- Deaths attributed to AD rose by 89% from 2000-2014

How does AD work?

- Characterized by tau neurofibrillary tangles and amyloid-beta (Aβ) plaques
- Mechanism historically viewed via “Amyloid Cascade Hypothesis”
- Dysregulation of protein causes harmful species of Aβ to form
- Tau protein normally promotes stability, becomes unbound

Treatments & Criticisms

• Currently no cure for the disease
• Until very recently, medications and therapies only able to alleviate symptoms
• Aduhelm was approved, showed slowing of cognitive decline
• Some scientists have criticized lack of clinical success treating Aβ plaques
• Suggested to focus on tau pathologies

Findings & Conclusions

• Completed a comprehensive literature review

• AD is now believed to operate via an amyloid-induced tau pathology

• Multiple other factors have been shown to reduce Aβ, such as
  • Hormone treatments
  • Butyrylcholinesterase inhibition
  • Induced oxidative stress

• Aduhelm, the only drug to show slowing effects, attacks Aβ plaques
Next Steps

• Amyloid Cascade Hypothesis should continue to guide research
• Identify effects of combining listed therapies and treatments
• Treatments should initially be studied in mice to examine the interactions of preemptive and corrective treatments
• Further emphasis should be placed on clinical trials
• With further research and treatment, AD can be decreased in severity and prevalence