

## ACTA SCIENTIFIC MEDICAL SCIENCES (ISSN: 2582-0931)

Volume 7 Issue 11 November 2023

**Short Communication** 

## What is Alzheimer's Disease?

## Yoheenee Jugdoyal\*

BSc Biotechnology, Johannesburg, South Africa

\*Corresponding Author: Yoheenee Jugdoyal, BSc Biotechnology, Johannesburg, South Africa.

Received: August 29, 2023
Published: October 19, 2023

© All rights are reserved by **Yoheenee** 

Jugdoyal.

#### **Abstract**

A disease which is characterised by memory loss, change in behaviour and thinking patterns. Alzheimer's disease is neurodegenerative and progressive. It is one of the most common causes of dementia and causes irreversible damage to the brain.

Keywords: Alzheimer's Disease; Brain; Cell

### Introduction

The human brain has approximately 86 billion neurons (nerve cells). These nerve cells link and form networks, which in turn helps us think, remember and learn. Cells in the brain require efficient oxygen supply and large amounts of nutrients to remain healthy throughout our lives. Alzheimer's disease disrupts the coordination of cells in the brain which in turn slows down its overall function [1].

The typical symptoms of this disease can be classified as

- Psychological: Which include signs of depression, becoming easily paranoid and hallucinating.
- Behavioural: Aggression, anger and general signs of discontent.
- **Cognitive:** Disorientation and mental decline are common symptoms.

# Alzheimer's in depth

The presence of the apolipoprotein E gene on chromosome 19 is considered a genetic risk factor. The Apolipoprotein E gene is a risk factor because it may increase the chances of developing the disease but it has been found that some individuals with this gene

have not developed Alzheimer's. There are many factors beyond genetics that may contribute to Alzheimer's disease such as lifestyle factors, health and environmental factors [2].

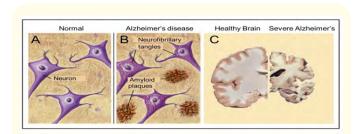


Figure 1: (Hunter, 2010) [3].

Tangles and plaques damage and kill nerve cells in the brain. Plaques are abnormal clusters of a protein called beta-amyloid that build up between nerve cells in the brain and interrupt the cell-to-cell signals at synapses. In a healthy brain beta-amyloid is breaks down and in eliminated. These abnormal clusters form when the enzyme APP does not cut beta-amyloid small enough and the large strands begin to cluster. There is currently no scientifically proven cure to this malfunction of the enzyme in the brain [3].

Tangles are twisted strands of protein. These tangles stop essential nutrients from moving to the cells, which eventually causes damage and death to the cell. A protein called tau keeps these strands straight. When it detaches from microtubules and get stuck to other tau molecules is eventually tangles inside the neurons, harming synaptic communication between neurons. The tau protein forms a "C" shape in the core of the tangle [1].

## **Bibliography**

- 1. Wang MQ., et al. "Research progress of genetics of Alzheimer's disease". Chinese Journal of Rehabilitation Theory and Practice 131.13 (2015).
- Gauthier S., et al. "Efficacy and safety of tau-aggregation inhibitor therapy in patients with mild or moderate Alzheimer's disease: A randomised, controlled, double-blind, parallel-arm, phase 3 trial". Lancet 388.10062 (2016): 2873-2884.
- 3. Hunter TA. "Replication of CLU, CR1, and PICALM associations with Alzheimer disease". *Archives of Neurology* 67.8 (2010): 961-964.