Journal of
Alzheimer's Disease & Parkinsonism

Tales, J Alzheimers Dis Parkinsonism 2021, 11:4

Editorial Open Access

Alzheimer's Pathology

Andrea Tales*

Department of Experimental Psychology, University of Bristol, UK

About the Study

Alzheimer's disease is a progressive neurodegenerative disorder involving misfolding and aggregation of proteins in conjunction with prolonged cellular stress. The neuropathology of Alzheimer's disease was discovered by Alois Alzheimer over a century ago. Alzheimer's pathology also may present in individuals without symptoms of dementia. Amyloid-β oligomer accumulation in intraneuronal compartments appears to be a significant risk factor for Alzheimer's disease. Mutant tau protein is present in inclusions. The microscopic observations of globose neurofibrillary tangles and variable neuron loss with gliosis of the globus pallidus, subthalamic nucleus, periaqueductal grey matter of pons, and substantia nigra are used to make the pathologic diagnosis.

The expression and function of enzymes involved in mitochondrial bioenergetics decreases as Alzheimer's disease progresses leading to the compromised electron transport chain complex activity and reduced ATP synthesis. Mitochondrial dysfunction plays a central role in the pathogenesis of neurodegenerative disorders, including Alzheimer's disease. Recent studies on mitochondrial functional analyses to address whether mitochondrial dysfunction precedes the development of Alzheimer's disease pathology indicated significant mitochondrial dysfunction occurs early in Alzheimer's disease pathogenesis. Endoplasmic Reticulum stress can also induce an inflammatory response via different unfolded protein response transducers leading to the activation of the inflammatory response and the evolution of pathological changes in Alzheimer's disease.

Oral administration of phenolic compounds prevented the development of Alzheimer's disease pathology by affecting different Aβ aggregation pathways in vivo. Aβ aggregation is the primary event in Alzheimer's disease pathogenesis and leading to the proposal that anti-Aβ aggregation is a strategy for Alzheimer's disease therapy.

When one aspect of the pathology is in place, it serves as a catalyst for the development of the other sections; for example, Abeta accumulation can lead to increased inflammation.

Genetics may play a key role while there is no underlying definition of Alzheimer's disease. Genetic and molecular studies have confirmed the central role of amyloid- β production and fibrillation in the pathogenesis of Alzheimer's disease. However, the pathological pathways from amyloid-β peptide oligomerization to the major pathological hallmarks of Alzheimer's disease, such as neurofibrillary tangles, inflammation and loss of cholinergic neurons, are largely unknown. The activation mechanisms of NALP inflammasomes in neurons and microglia and several downstream effects in brain demonstrating that toxic amyloid-β oligomers and fibrils can induce Alzheimer's pathology.

Neuropathological changes and neurodegeneration occur years before the onset of clinical symptoms. Understanding the nature of changes during this apparently asymptomatic state may shed light on mechanisms that forestall the progression of the disease and maintain cognitive health. Logopenic/phonological aphasia may be a "unihemispheric" presentation of Alzheimer's disease, and discuss this concept inrelation to accumulating evidence concerning language dysfunction in Alzheimer's disease. It is most commonly associated with Alzheimer's disease pathology. PPA syndromes may also be associated with Alzheimer's disease pathology.

***Corresponding author:** Andrea Tales, Department of Experimental Psychology, University of Bristol, UK, E-mail: Andrea.Tales@bristol.ac.uk

Received April 08, 2021; **Accepted** April 22, 2021; **Published** April 29, 2021

Citation: Tales A (2021) Alzheimer's Pathology. J Alzheimers Dis Parkinsonism 11: e145.