**ABSTRACT**

Dependence of synaptic plasticity plays a critical role in Alzheimer’s disease (AD) pathogenesis. Recent studies have suggested that neuroinflammation may contribute to the degeneration of the amyloid metabolism and by microglial overactivation. This study aims to investigate the pathophysiological progression of AD with the help of neuroinflammation.

**RESULTS**

**CONCLUSION**

Brain tissues were then stained via IHC with use of anti-Neuronal nuclei to confirm the pathological similarity of the brain tissue to the AD patient.

**DISCUSSION**

Analysis of tissue from human patients found significantly higher intracellular Aβ deposition in AD mice compared to wild-type mice. Extracellular Aβ protein, that has been cleared and is now susceptible to forming plaques, is symptomatic of AD pathology and amyloid, was found only in the AD patient.

Quantification of microtubule-associated protein showed significantly higher expression in varying groups of the 3xTg-AD model after 6 months of development. Additionally, there was significantly higher presence of some of the 3xTg-AD female mice than their male counterparts. AD is not present healthy WT mice, but did present in AD female mice and the amyloid of the 3xTg-AD male mice.

The differences in the amyloid burden between the male and female AD mice suggest that female AD mice may have a more susceptible endogenous response to AD. Further investigations are required to explore this phenomenon.

**REFERENCES**


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**MINIABSTRACT**

**Introduction**

Alzheimer’s Disease (AD) - This is an age-related neurodegenerative disorder that affects cognitive function and results from the progressive loss of brain cells. The incidence of AD increases with age, and it is estimated that 1 in 10 people over 65 suffer from AD, with the majority of cases occurring in those 85 years or older.

**Materials & Methods**

The 3xTg-AD mouse model is a genetic model of AD, comprising three mutations that lead to the accumulation of amyloid-β (Aβ) plaques and hyperphosphorylated tau. The presence of these hallmarks is used to measure disease progression and evaluate potential therapeutic strategies.

**Results**

**Discussion**

Analysis of tissue from human patients found significantly higher intracellular Aβ deposition in AD mice compared to wild-type mice. Extracellular Aβ protein, that has been cleared and is now susceptible to forming plaques, is symptomatic of AD pathology and amyloid, was found only in the AD patient.

Quantification of microtubule-associated protein showed significantly higher expression in varying groups of the 3xTg-AD model after 6 months of development. Additionally, there was significantly higher presence of some of the 3xTg-AD female mice than their male counterparts. AD is not present healthy WT mice, but did present in AD female mice and the amyloid of the 3xTg-AD male mice.

The differences in the amyloid burden between the male and female AD mice suggest that female AD mice may have a more susceptible endogenous response to AD. Further investigations are required to explore this phenomenon.

**Conclusion**

Overall, this investigation sheds light on trends in presentation and activity of AD pathological hallmarks as the age of participants increases, potentially increasing the effectiveness of studies of disease progression and potential intervention.