Role of a soy protein Lunasin in Aβ42 mediated neurodegeneration in Alzheimer’s Disease.

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Abstract

Alzheimer’s Disease (AD) is a neurodegenerative disease caused by a number of factors. One of the leading factors behind the onset of AD is the accumulation of abnormal proteins in the brain of affected individuals. These proteins are linked with levels of oligomeric and/or aggregated protein structures, which can lead to the formation of plaques and neurofibrillary tangles (NFTs) in the brain. Epigenetic changes, oxidative stress, and inflammation are all factors that contribute to the progression of AD. The presence of Aβ42 and Aβ40 peptides, which are derived from the amyloid precursor protein (APP), play a critical role in the pathogenesis of AD. These two peptides aggregate in the brain, forming insoluble plaques. These plaques are implicated in the progression of AD and serve as a marker for the disease.

Gain of function approach: GAL4/UAS- System

Lunasin suppresses Aβ42 mediated neurodegeneration

Alzheimer’s Disease

1. Amyloid plaques
2. Neurofibrillary tangles
3. Oxidative stress due to ROS
4. Neurofibrillary tangles (Secondary)
5. Genetic Basis of Aβ42

Drosophila eye model to study Aβ42 mediated neurodegeneration

Downregulation of crumbs increases levels of Lunasin in Aβ42 background

The Power of a soybean protein Lunasin

- Protein found within soy products
- 43 amino acids per protein
- Cancer research related to tumor suppression and Increase cancer cell proliferation
- Heart disease research links Lunasin to be anti-inflammatory

Conclusions and Future Research

Conclusions

- In our preliminary findings we have shown that Lunasin is able to rescue the neurodegenerative Aβ42 plaques in our Drosophila eye model.
- The marker protein Wg expression is similar in our GMR-Aβ42 Tg and in our GMR-Aβ42+Lunasin

Future Directions

- Discover the role Lunasin plays in regulating other protein markers (E3) and pathways involved in Aβ42 mediated neurodegeneration.
- Research the underlying effects of Lunasin that rescue the misexpression of Aβ42 in the Drosophila eye model.