Background

In mitochondrial dysfunction the respiratory chain efficacy decreases which causes electron leakage, decrease in ATP production and increase in ROS production [1]. Mitochondrial dysfunction is a known driver of cell senescence and has been associated with aging and aging related diseases, impaired mitochondrial function causes the disruption of cellular homeostasis and affects cellular cell function [1].

Aim

Build a model that portrays the role of mitochondrial dysfunction in cell senescence and aging.

Research Model

To understand the role of mitochondrial dysfunction on aging and longevity, I will use a transgenic *Drosophila* model to overexpress α-synuclein, a protein of 140 amino acids expressed in the central nervous system. α-synuclein is implicated in the induction of mitochondrial dysfunction. Overexpression of α-synuclein should result in mitochondrial dysfunction. Then the *Drosophila* will be aged by maintain them at 29°C since their lifespan is extended at this temperature. Furthermore, the *Drosophila* will be observed and their survival rate will be recorded. The *Drosophila* with overexpressed α-synuclein will be compared to *Drosophila* with normal levels of the α-synuclein protein and their survival will be recorded as well. By comparing the survival rate of the Drosophila with overexpressed α-synuclein and the Drosophila without overexpressed α-synuclein, the role of mitochondrial dysfunction can be understood. In the future this model can also be used to find treatments or medications that will target mitochondrial dysfunction. Furthermore, it can be used to study disease affected by the overexpression of α-synuclein such as Parkinson's Disease.

References

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