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Bio 320: Qin’s Research Proposal

T/Th-1p

**Summary: Qin’s Research Proposal**

Aging, and the components and factors that influence it, is a widely questioned, researched, yet still incompletely understood topic. Cellular aging is believed to be influenced by gene/protein networks. In Qin’s research proposal it is hypothesized that rate of cellular aging is proportional to gene/protein network robustness. The project and analysis of causes of aging were tested by statistical computations and analysis of statistical significance. The next part of the project was analyzing the effects of gene/protein network robustness on the rate of cellular aging. Robustness is defined as endurance to Hsp90, TOR inhibitors, and oxidative stress, which influence Reactive Oxygen Species, ROS, in cells and their rate of death. The Gompertz coefficient is a mathematical explanation of robustness and cellular aging. The Gompert coefficient **m = - = IeGt** includes the following variables: (m) mortality rate, (s) survival fraction of a population, (I) innate susceptibility to dying, and (G) gompertz coefficient, rate of aging. Another goal of the project is to identify target genes, SIR2, TOR1, and SCH9, that influence aging and network robustness. Another hypothesis in the research proposal is that individual cellular components influence the overall effect of cellular aging in cells. SIR and TOR genes are believed to share pathways that promote genomic stability during aging. Through the research it has been found that deletion of genes can either shorten or lengthen chronological and even replicative life span. A few are shown to be related to the TOR pathway. These findings prove that gene/protein networks are indeed influential in cellular aging.

Other research projects have identified gene networks as network models of aging (Xue et. al 2007). In this research project 30 post-mortem human brain frontal cortex samples were collected and a microarray analysis conducted, where 440 differentially expressed genes between old and young brains were found. The study identified a large number of genes that display age-related changes in various seemingly unrelated biological processes (Xue et. al 2007). Another research project found that diverse array of genetic mutation can result in increased life span (Bell 2009). This finding contributes to the Qin proposal’s hypothesis that not just one individual gene but the interaction of gene/protein networks and the changes that occur through them effect and influence aging. The project was carried out by mapping the protein interaction network of human homologs of proteins that effect lifespan in invertebrates. The lifespan/longevity network was composed of 175 human homologs of proteins that have been known to increase lifespan in species such as yeast, as Qin’s proposal uses, and flys. 2163 human proteins interact with the homologs. Homologs of 18 human interacting proteins that show significant changes in human aging muscles were analyzed to determine if homologs of human longevity interacting proteins can modulate lifespan in invertebrates. This study also supports Qin’s proposal that there are a broad range of genes that have an influence and effect on cellular/human aging.

Works Cited

Bell R, Hubbard A, Chettier R, Chen D, Miller JP, et al. (2009) A Human Protein Interaction Network Shows Conservation of Aging Processes between Human and Invertebrate Species. PLoS Genet 5(3)

Xue H, Xian B, Dong D, Xia K, Zhu S, Zhang Z, Hou L, Zhang Q, Zhang Y, Han JD. A modular network model of aging. Mol Syst Biol. 2007;3:147