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Testing the Effects of Increased Oxidative Stress on Aging of *Drosophila* Germline Stem Cells

Aging occurs in almost all organisms, and its associated phenotypic effects have been observed in most organ systems. Despite greater knowledge on the effects of aging on different organ systems, the cellular mechanisms that contribute to this phenomenon remain relatively unknown. In trying to understand aging, stem cells are an ideal population to study because they persist throughout the lifetime of the organism, and thus, are more susceptible to accumulated damage from the cellular environment than are somatic cells. Studies have suggested that the age-related decline in the regenerative potential of tissues, a hallmark of aging, is caused in part by defects in the corresponding stem cells.

My research explores the oxidative stress theory, which attempts to explain aging as the result of accumulated cell damage caused by reactive oxygen species (ROS) over time, using *Drosophila melanogaster* as a model organism. Primarily produced by the mitochondria as a by-product of inefficient metabolism, ROS are highly reactive molecules that damage proteins, nucleic acids, lipids, and other cellular components. While data exists for the effects of ROS on the whole organism in regards to aging, the effects of oxidative damage on specific tissues is much less clear. Previous research has demonstrated that the cell cycle activity of male *Drosophila* germline stem cells (GSCs) decreases during normal aging. I am investigating the effects of increased levels of oxidative damage on the cell cycle activity of GSCs by use of the free-radical producing chemical paraquat.