



Editorial

The Study of Aging in *Caenorhabditis elegans*

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Because humans have a long lifespan relative to other organisms, aging studies have been performed in a variety of systems. *Caenorhabditis elegans* (*C. elegans*) is one model organism that has been used to study aging due to its short life span (about 3 weeks), low cost for maintenance, its ease of cultivation and the fact that it produces large number of offspring [1-2]. The mechanisms involved in aging in *C. elegans* have also been studied in other organisms, including mammals. Among these mechanisms are oxidative damage, dietary restriction, reduction in the Insulin/Insulin-like Growth Factor (IGF) signaling pathway and decreased TOR (Target of Rapamycin) signaling [1-4].

Oxidative damage in *C. elegans* has been shown to play a prominent role in aging. The *mev-1* mutants have increased production of reactive oxygen species and have shortened life spans [1] while *daf-2* mutants that have increased antioxidant genes, including a gene for Superoxide Dismutase (SOD) and mutations in the Insulin/Insulin-like Growth Factor (IGF) receptor have longer life spans compared to wild type strains [1-3]. One study has found that when *mbk-1* is lost, the lifespan of *daf-2* is shortened. *MBK-1* has been identified as a protein kinase [4].

Previous studies have shown that dietary restriction extends the lifespan of both *C. elegans* and mammals [5-6]. Effects on the insulin-like growth factor signaling pathway and lysosomal degradation by autophagy are mechanisms involved with this life extension. *ATG18* helps to maintain lifespan in *C. elegans*. It has also been found that the mammalian homolog *WIPI* is involved with age-related human diseases [5]. Other studies have looked at the effect of diet on longevity. Recent research with *C. elegans* shows that diets high in glycogen decrease lifespan and accelerates the aging of the *daf-2* worms that normally have an extended lifespan [7].

C. elegans is also a valuable organism to use to study aging since many mutant strains are available from the Caenorhabditis Genetics Center. Specific strains of *C. elegans* can be found by searching the wormbase website but some examples can be found at this link: <http://www.wormbase.org/search/gene/aging%20mutants>. Studies have also been done with *C. elegans* to identify drugs that could delay aging both in these organisms and in mammals. Many of these drugs were found

to increase the resistance to oxidative stress and target intracellular signaling [3].

Studies on aging in *C. elegans* require synchronized populations to be used so that the animals are all of the same age. One common mechanism used is to treat the *C. elegans* with bleach so that only the eggs remain. Then, the drug 5-fluoro-2'-deoxyuridine (FUdR) is used which kills dividing cells so that the adult *C. elegans* will remain but no further offspring will be viable. It is important, though, that the FUdR is administered to adults as the drug is toxic to young *C. elegans*. In order to do the experiments, the timing of administering the *C. elegans* is critical. The drug is toxic to larvae so the *C. elegans* should be transferred to plates with the FUdR just before they start laying eggs. If it is given to the worms when they are young, the survival rate will be low. In addition, if the *C. elegans* are too old, the hatched *C. elegans* may be able to survive the drug treatment [1-2].

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