Cracking the Code: New Genetic Link Between Sleep and Aging-slo-1 in C. elegans

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Aged people are more prone to sleeping disorders, which poses a serious health concern worldwide. The underlying mechanisms of sleep and aging related diseases remain unclear following my meta-analysis that confirmed the link between sleep and these diseases. Although both slo-1(js379) and slo-1(md1745) mutations have been independently proven to slow down aging and disrupt sleep, respectively, the site that regulates sleep and aging simultaneously is unknown, hindering the development of effective gene-targeting treatments. Caenorhabditis elegans (C. elegans), a nematode with significant genetic similarity to humans, was used as a model for molecular correlation. We used the control strain N2 wild-type and mutant strains slo-1(eg142)V, slo-1(js118)III, and slo-1(js379)V to test for sleep and aging behavior. A sleep study was conducted using the Lethargus model. Four assays were used to evaluate sleep stability, body bends, nose swings, pumping, and the ability to respond to a stimulant during sleep and awake. Aging speed is assessed using Tracking model. In light of the fact that mobility is a key indicator of aging speed, we examined the worms' mobility over 7 days. Compared to the wild type, the assays showed that the slo-1(js118)III mutation decrease sleep stability ($p<0.05$) and offers 1.5 times the speed of aging ($p<0.01$) that of the wild type. These proved slo-1 a new common gene in sleep and aging and is therefore a new promising therapeutic strategy, offering insights into new common pathways in sleep and aging. This discovery can lead to precision and dynamic monitoring and treatment of various aging-related diseases.

Awards Won:

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