The Effect of Antisense Oligonucleotides on ALS Models of Drosophila melanogaster

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It has recently been discovered that the C9orf72 gene mutation causes RNA molecules to block critical pathways for protein transport. Nerve cells will die off, causing degeneration in the brain. These are all effects of ALS and FTD. The effects of the C9orf72 mutation in nerve cells were reduced after Drosophila melanogaster had undergone antisense oligonucleotide treatments. This is extremely significant because of the potential that the antisense oligonucleotides have on the recovery of nerve cells in Drosophila melanogaster models, which would be applicable on humans. In this experiment, Drosophila melanogaster with various forms of the C9orf72 gene mutation were treated with antisense oligonucleotides to see if there would be a decrease in degeneration of ALS models of Drosophila melanogaster. The antisense oligonucleotides were administered orally (through their food) to the Drosophila melanogaster. After this, the Drosophila were observed under a stereoscope for signs of degeneration in the eye, which is how the gene is expressed. Once the Drosophila were treated, the degeneration in the Drosophila's eyes had notably decreased due to the antisense oligonucleotides' effect on the elimination of blockage for nucleocytoplasmic transport.

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