# Characterization of D. Melanogaster Gene CG5734 and Relation to ALS Pathogenesis

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**Abstract:** The focus of our study was to elucidate the possible function of CG5734 by analyzing its interacting partners. We also examined the functional domains of CG5734 and predicted that CG5734 is a member of the sorting nexin family, specifically Snx17. Lastly, we traced the function of CG5734 back to ALS pathogenesis and determined how this gene could possibly control the expression of the ALS phenotype found in previous studies. (Sanhueza et al, 2015)

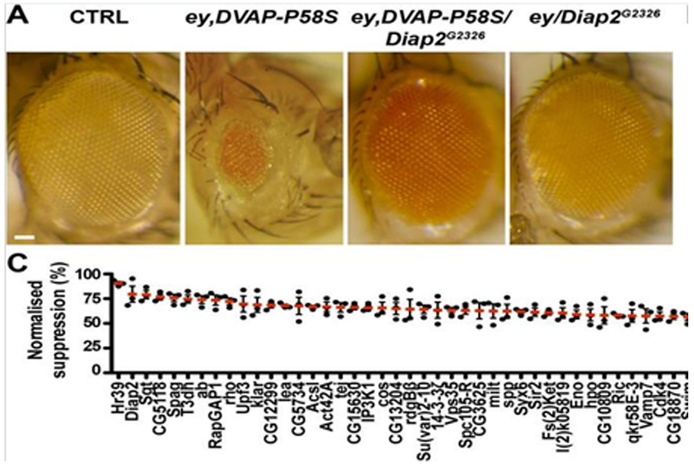


**Figure 1:** Effect of CG5734 overexpression on the Drosophila reduced eye phenotype induced by DVAP-P58. (Sanhueza et al. 2015)

| Tissue | Adult Expression Level |
|---|---|
| Head | 373.7 |
| Eye | 591.85 |
| Brain | 542.2 |
| Central Nervous System | NA |
| Thoracic-Abdominal Ganglion | 658.5 |
| Crop | 305 |
| Midgut | 218.8 |
| Hindgut | 209 |
| Malpighian Tubules | 309.6 |
| Fat Body | 341.1 |
| Salivary Gland | 204.9 |
| Heart | 310.775 |
| Trachea | NA |
| VirginFemale Spermatheca | 388.1 |
| seminatedFemale Spermatheca | 451.3 |
| Ovary | 534.9 |
| Testis | 76.8 |
| Male Accessory Gland | 219.8 |
| Carcass | 197.2 |

**Figure 2:** Expression pattern of CG5734 in D. melanogaster larva and adults. (flybase.org)

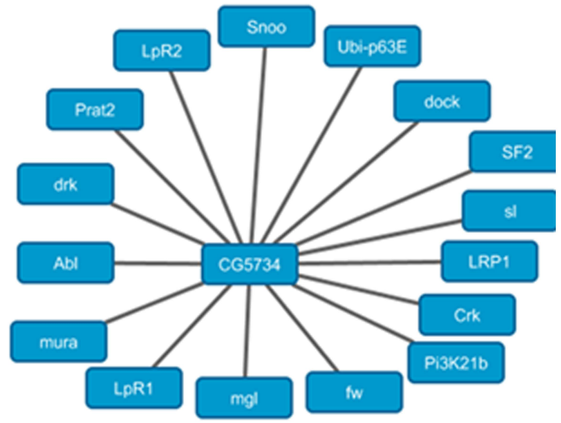


**Figure 3:** Interacting partners of CG5734.

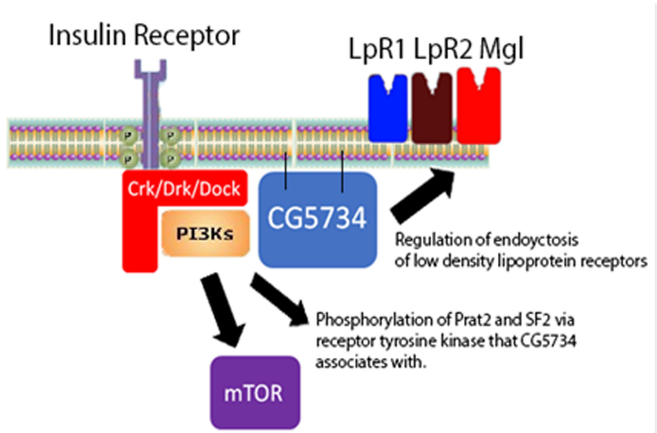


**Figure 4:** Proposed model of CG5734's role with various cellular proteins.

## Conclusion:

Statins are drugs used to reduce levels of lipids in the blood. (Colman et al. 2008)

This was an interesting link to CG5734 because it is implicated in the amount of lipid receptors on the membrane of cells. People who were taking statins had a disproportionate amount of ALS cases compared to the general population. (Colman et al. 2008)

Some patients taking statins may have a mutation in SNX17 which leads to high concentrations of lipids floating in the bloodstream as they cannot be taken up into cells lacking lipoprotein receptors at the cell membrane.

It could be possible that a mutated CG5734 would starve the brain of essential fatty acids and initiate cell death within motor neurons.

It would be interesting to further investigate the possible link between the newly characterized CG5734 to see how it effects neuronal lipid uptake in flies and how dysregulation of this uptake may have neurodegenerative effects.

In conclusion, we have elucidated a possible new link between ALS and our newly characterized Skinny gene that is implicated in fat uptake but may also modulate essential pathways such as receptor tyrosine kinase signaling and the regulation of DNA/RNA regulatory proteins.