

Póster

Suppression of the sterility in insulin signaling pathway mutants of *Caenorhabditis elegans*



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ABSTRACT

Mutants in the insulin signaling pathway are related with a low or null fertility in *Caenorhabditis elegans*. This pathway is conserved through evolution in all metazoans, and similarly to *C. elegans*, deficiencies in insulin signaling pathway activity result in a decreased fertility in other organisms including humans.

We have previously selected a strain that carries a mutation in the *age-1* gene (orthologue to human PI3K) which has a severe reduced fertility phenotype. Using this strain it was performed a screening for genes that when removed by RNAi suppressed the sterility, using for that only genes that were orthologue to human. Most of genes whose reversion was significantly different were genes involved in the initiation of the transcription (eIF). Those genes encode to non-specific non-ribosomally associated proteins involved in the initiation phase of eukaryotic translation.

The aim of this project is to better understand the relationship between fertility and the expression of eIF genes and why RNAi of only a few of them can improve fertility and not others. On the other hand, I'm searching for drugs that improve the fertility of the nematode similarly to RNAi that could be used in future human therapies.

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