YOLK PROTEIN BIOSYNTHESIS AFTER *dsc-4* KNOCKDOWN IN *CAENORHABDITIS ELEGANS*

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The *dsc-4* gene of *Caenorhabditis elegans* encodes an orthologue of the human Microsomal Triglyceride Transfer Protein (MTP). Mutations in the coding region of this gene affect worm aging processes. MTP is a member of the large lipid transfer protein (LLTP) superfamily, wich includes vitellogenins, insect apolipophorins and human apolipoprotein B (apoB). MTP mutations in man result in severe abetalipoproteinemia. It has been previously demonstrated that MTP promotes the secretion of *Xenopus laevis* vitellogenin A1 in cell cultures. Here we present an analysis of the *dsc-4* gene role in vitellogenin biosynthesis in the nematode worm *C. elegans*. In *C. elegans*, double-stranded RNA (dsRNA) provided in the diet can be absorbed from the gut and distributed troughout the body, triggering RNA interference (RNAi) in different tissues. In order to knockdown *dsc-4* expression a cDNA fragment corresponding to a partial *C. elegans dsc-4* sequence was ligated into the feeding vector L4440, between its two inverted orientation T7 promoters, and transformed into *E. coli* HT115(DE3). These dsRNA-expressing bacteria were used to fed wild-type worms (strain N2) and transgenic worms (strain DH1033) expressing YP170B::GFP. Living worms, fed on dsRNA-containing bacteria, show YP170B::GFP accumulation in the enterocytes cytoplasm, suggesting that the vitellogenin transport from the gut cells to the pseudocoelom is compromised.

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