ABSTRACT

Regulation of gene expression in response to cellular and organismal needs is essential for sustaining organisms' survival and successful competition in the evolution of life forms. This regulation is executed at multiple levels starting with regulation of gene transcription, followed by regulation at multiple posttranscriptional levels. In this thesis, I focused on posttranscriptional mechanisms that contribute to gene expression regulation in the model organism *Caenorhabditis elegans* which enables powerful genetic and genomic techniques and allows the visualization of experimental genetic manipulations *in toto*, on the level of the complete organism during its life span. For this, we analysed the function of the orthologue of mammalian transcriptional corepressor NCOR, GEI-8. We used a functionally defective mutant *gei-8(ok1671)*. I analysed the whole genome expression of homozygous *gei-8(ok1671)* mutant and its link with observed mutant phenotype that includes defective gonad development and sterility and performed experiments leading to the proposition that disbalances in 21-U RNAs of piRNA class present in the most derepressed gene, the predicted mitochondrial sulfide:quinine reductase encoded by Y9C9A.16, are associated with the gonadal phenotype. In the second part of the thesis, I focused on the function of an RNA modifying enzyme that is likely to fundamentally contribute to posttranscriptional modification of several classes of RNA, the nematode orthologue of ALKBH8, named ALKB-8. Both the nematode and vertebrate orthologues contain three functional domains, an N-terminal RNA binding motif, a 2-oxoglutarate-dependent dioxygenase module homologous to bacterial AlkB, which oxidatively demethylates DNA substrates and a methyltransferase domain homologous to yeast TRM9, which selectively modulates translation of mRNAs enriched with AGA and GAA codons under both normal and stress conditions. We show that downregulation of *alkb-8* increases the extent of lysosome-related organelles visualized by Nile red *in vivo* and reversely, forced expression of *alkb-8* strongly decreases the detection of this compartment. Overexpression of *alkb-8* applied in a pulse during the L1 larval stage projects to increased life span of *C. elegans.* Together, our results identified new regulatory pathways based on posttranscriptional mechanisms and contributed new data supporting the concept of extensive posttranscriptional mechanisms modulating gene expression to comply with organism's needs.