Public Abstract
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Analysis of the *Caenorhabditis elegans rpc-1* gene
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In eukaryotes, two large subunits form the core catalytic structure of RNA polymerase III (Pol III), which is conserved in other RNA polymerases, Pol I and Pol II. It has been found that Pol III activity is tightly associated to cell growth. TFIII B has been shown to be one of main mediators in this process. No regulation of the Pol III largest subunit gene has been found. In *Caenorhabditis elegans*, the *rpc-1* gene encodes the largest subunit of Pol III. Here, I identified two critical structural components of RPC-1, Gly644 and Gly1055, whose mutations result in larval lethal arrestment. These two amino acid residues are universally conserved in RNA polymerases, indicating their overall involvement in gene transcription mechanism. Also, I found that maternally inherited, not embryonically expressed, *rpc-1* gene products survive early development. Starvation was found to suppress *rpc-1* gene expression and re-feeding treatment enhances *rpc-1* gene expression rapidly. No similar regulation was detected in genes encoding largest subunits of Pol I and Pol II. This is the first time that *rpc-1* gene regulation has been reported. Insulin signaling may not be involved in this regulation.

Also, I found that *rpc-1* promoter is not ubiquitously active in *Caenorhabditis elegans*. Using the *rpc-1p::gfp* transgene, the *rpc-1* promoter activity is only detected in a subset of neurons in the head and the tail, and the intestine. While starvation silences the *rpc-1* promoter activity in most tissues and cells, ASK neurons still show GFP staining in the *rpc-1p::gfp* transgenic animals, indicating that *rpc-1* transcription in ASK neurons is continuously active under starvation conditions. Further studies suggest that TGF-beta signaling is involved in mediating the *rpc-1* promoter activity in ASK neurons.