D119 Induction of Metallothionein in Regenerating Rat Liver

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Metallothionein (MT) is a low molecular weight, heavy metal-binding protein that participates in the regulation of growth and development. The present study was designed to examine the role of MT in cell proliferation during liver regeneration in partial hepatectomized rats. Immunohistochemical study using anti-MT antibody revealed that there were more intense reaction products in regenerating rat liver. MT was localized predominantly in the nuclei of partial hepatectomized rat liver, whereas MT was weakly found only in the cytoplasm of sham-operated rat liver. Quantitation by silver saturation showed that MT levels were increased in the remnant liver rapidly after the hepatectomy, its concentration being several fold higher than that of the intact liver. MT was significantly induced in the liver at 6 hr after hepatectomy and peaked at 24 hr. MT induction in the remnant liver was determined in a time-dependent manner. These results suggest that MT was involved in the regulation of cell proliferation during liver regeneration.

D120 *apm-1*, a Medium Chain of Clathrin-associated Protein Complex, is a Redundant Negative Regulator in *C. elegans* Vulval Development.

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Clathrin-coated vesicles participate in intracellular trafficking of various proteins. Clathrin-associated protein complexes(AP complexes) are important in clathrin recruitment and cargo selection. The AP complexes are heterotetrameric structures composed of two large chains, one medium chain, and one small chain. *C. elegans* has two medium chain homologs of AP-1 complex : *apm-1* and *unc-101*. *unc-101* was originally cloned as a suppressor of *let-23* mutations in vulval development pathway. *apm-1* is about 74% identical to *unc-101* and mouse AP47 in amino acid sequence. It is possible that these two medium chains have similar functions in *C. elegans*. In this study, We'd like to propose redundant relationship of *apm-1* and *unc-101* in *C. elegans*. GFP and Lac-Z reporter gene expression patterns of *apm-1* and *unc-101* overlap in the vulval cells and several cells. Especially, *apm-1* RNA interference(RNA*i*) into *let-23* mutant and *unc-101* mutant animals resulted in suppression of vulvaless phenotype, and hyperinduction, respectively. Therefore, *apm-1* functions as a negative regulator in vulval development pathway like *unc-101*, and at least *apm-1* and *unc-101* are redundant in the vuval cells in *C. elegans*. The effect of *unc-101* RNA*i* was equal to that of *unc-101* mutation as we had expected. Another interesting thing is that GFP construct of *unc-101* showed dominant negative effect.