F818 Characterization of lethal mutants on chromosome I of *Caenorhabditis elegans*

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We have isolated six independent mutants (*jh1* to *jh6*) out of approximately 5,200 ethyl methanesulfonate (EMS) treated haploids. Four of the six mutants demonstrated embryonic lethal phenotypes, while the other two showed embryonic and larval lethal phenotypes. Terminal phenotypes observed in two mutants (*jh1* and *jh2*) indicated developmental defects specific to posterior part of embryos which appeared similar to the phenotypes observed in *nob* (no back end) mutants. Another mutant (*jh4*) resulted in an interesting phenotype of body-wall muscle degeneration at larval stage. These mutants were mapped by using three-factor crosses and deficiency mutants in this region. We are currently characterizing two mutants, *jh2* and *jh4*, in order to map these genes. Here we report genetic analysis and characterization of these lethal mutants.

F819 **Paralytic Behavior of a *Drosophila* Temperature-Sensitive Mutant, *shibire*, was Modified by Memory Mutants**

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The temperature-sensitive mutant fly of *Drosophila melanogaster, shibire^{ts1}* (*shi^{ts1}*), behaves normally at room temperature wherase exhibits paralytic behaviors at restrictive temperatures, above 29℃. The memory and learning mutants of *Drosophila*, *dunce* (*dnc*) and *rutabaga* (*rut*), show defect in cAMP metabolism. The *dnc* alleles exhibit an elevated intracellular cAMP level while *rut* flys show a greatly reduced level. Recent studies have suggested that changes in intracellular cAMP concentrations have been shown to be critical for regulation of growth cone motility, neurite elongation and synaptic plasticity which are implicated in neural development and differentiation of neuronal morphology. In this study, characteristics of temperature-sensitive paralytic behavior were examined in the double mutant flies constructed from two cAMP-defective mutations and a temperature-sensitive mutation. The behavioral repertoires of paralysis in the double mutants were different from those of *shi^{ts1}* single mutant flies. Furthermore, *dnc* gene in the double mutation cause a deteriorating effect in the temperature-sensitivity while *rut* does compensation in the similar settings. The results suggest that the elevated intracellular cAMP level may activate exocytosis in the double mutant while the subsequent endocytosis is still blocked by *shi^{ts1}* mutation.