Synopsis

Locomotion is essential for animals and it is regulated by crosstalk between neurons and muscles. Although defects in the crosstalk between neurons and muscles are implicated in many mobility related diseases in human, however, the underlying molecular mechanisms have not been well characterized. In an attempt to gain insights into the complex neuro-muscular crosstalk at molecular level, we have used *Drosophila melanogaster* as model system to elucidate function of a gene, *taxi*, mutations in the gene give rise to defective flight behavior resulting from a defective neuro-muscular crosstalk.

The first chapter of this thesis explains how crosstalk between neurons and muscles control different behaviors of *Drosophila melanogaster,* particularly locomotion. Apart from locomotion, this chapter also explains how flies perform vision and how they response to various stimuli. Second chapter explains materials and methods used to complete the studies.

Third chapter explains genetic and phenotypic characterization of *jumper* mutant, an allele of *taxi* gene. Further, based on experimental data, how the mutation affects the *taxi* at both transcription and translational levels have been discussed. We found that I-element insertion located at 5'UTR of the *taxi* in *jumper* is responsible for the defective flight behavior in *jumper* mutant. The I-element insertion leads to increased expression level of *taxi* in the head without affecting much of transcription and translation in other body parts.

Fourth chapter explains the role of *taxi* in flight behavior of *Drosophila melanogaster*. We found that knockdown of *taxi* in neurons gives rise to compromised flight ability. Spatio-temporal conditional knockdown experiments suggest that *taxi*'s function is critical at around 12 hours After Puparium Formation (APF). Further, from our RNA sequencing result of *taxi* null, we found that the genes important for maintaining membrane potential are differentially expressed. As a result, neuronal transmission from brain to indirect flight muscles (IFMs) through peripherally synapsing interneurons (PSI) gets altered, which might lead to reduction in wing beat duration of *taxi* mutants. We found that one of the main regulators of membrane potential is *Adar*, a gene that is crucial to many molecular and physiological activities, and it is repressed by *taxi*.

Fifth chapter of this thesis explains how *taxi* affects life span and phototaxis. We found that over-expression of *taxi* in neurons beyond threshold shorten life span of flies. It is also observed that phototaxis is abnormal with disruption in structure of ommatidia.

Six chapter of this thesis explains conclusion of the study.

Overall, in present study we have reported for the first time neuronal function of *taxi* in flight behavior, life span and phototransduction of *Drosophila melanogaster*.