Motor deficits in a *Drosophila* Atlastin mutant

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The hereditary spastic parapareses (HSPs) are a clinically and genetically heterogeneous group of disorders characterized by progressive lower limb spasticity and weakness, likely caused by the length dependent "dying back" of the terminal ends of the corticospinal tract axons. Mutations in several proteins without apparent functional similarity have been linked with HSP, most prominently Spastin, Atlastin and Spartin. Mechanistic analysis of these factors should profit from functional-genetic analysis of homologous proteins in genetically well accessible model organisms. Recent analysis in the fruit fly *Drosophila* has implicated Spastin in the regulation of axonal microtubule dynamics, resulting in synaptic dysfunction at neuromuscular terminals. Atlastin is a multimeric, integral membrane GTPase that may be involved in Golgi membrane dynamics or trafficking of Golgi-derrived vesicles. We recently produced a null mutant of the *Drosophila* Atlastin homologue. In the absence of Atlastin, few adult flies could still form, which, however, were abnormally small and sterile. Notably, flies without Atlastin showed severe motor deficits. All defects of the mutant could be rescued by transgenic re-expression of Atlastin. We know study subcellular distribution and binding partners of Atlastin. HSP diseases might have roots in impaired stress resistance of affected neurons. In fact, we could show that overexpression of Spartin could significantly prolong life span of yeast cells put under oxygen stress. Reversely, flies without Atlastin showed markedly reduced life span (average 9.5 days instead of normal 40 days). We in the moment address whether HSP related proteins converge in a neuronal stress response program.