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Evolutionary conservation of conduction velocity through NF-M microsatellite expansion

Myelination results in rapid conduction velocities due to myelin-dependent radial axonal growth and axonal insulation. As larger mammals evolved, the resulting increase in axonal length would require a compensatory mechanism to maintain rapid conduction velocity. The main cytoskeletal component of myelinated axons is neurofilaments. Additionally, neurofilaments medium (NF-M) and heavy (NF-H) are more heavily phosphorylated on serine residues of the lysine-serine-proline (KSP) microsatellite than in non-myelinated areas of the same axon. In mouse, evidence suggests that the loss of the NF-M microsatellite strongly inhibits radial-axonal growth resulting in decreased conduction velocity. My preliminary results suggest a direct relationship between the axonal length and the number of KSP repeats in the NF-M microsatellite. Using degenerate primers, I have amplified exon 3 of the NF-M gene from genomic DNA of phylogenically diverse mammals. Gel electrophoresis data indicates an increase in the length of exon 3 with an increase in species size. Through DNA sequence analysis, we are in the process of determining if the increase in length of exon 3 is due to an increase in the number of KSP repeats in the NF-M microsatellite. This evidence suggests that the expansion of the KSP repeats of the NF-M microsatellite may be a possible mechanism through which evolution increased axonal diameter as larger animals evolved. As axonal diameter is one of the key determinants of conduction velocity, larger axonal diameters may allow for conservation of conduction rates in mammals of differing sizes as is observed in mouse and humans (both conduct at ~50m/s).