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From Africa to your backyard: Evolutionary expansion of axons to maintain rapid nerve conduction in mammals

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Myelination evolved as a mechanism to allow for rapid action potential propagation along relatively small axons. Myelination results in rapid conduction velocities due to myelin-dependent radial axonal growth and insulation of the axon. Neurofilaments, the main cytoskeletal component of myelinated axons, are essential for myelin-dependent radial axonal growth. Additionally, neurofilaments medium (NF-M) and heavy (NF-H) are more heavily phosphorylated on serine residues of the lysine-serine-proline (KSP) repeats in myelinated internodes than in non-myelinated areas of the same axon. In mouse, loss of NF-M KSP repeats strongly inhibits radial-axonal growth and causes a subsequent decrease in conduction velocity. My preliminary results suggest a relationship between the axonal length (approximated by species size) and the number of KSP repeats found in NF-M. Using degenerate primers, I have amplified exon 3 of the NF-M gene from genomic DNA of phylogentically diverse mammals. Subsequent 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. As larger mammals evolved, the resulting increase in axonal length would require a compensatory mechanism to maintain rapid conduction velocity. This evidence suggests that the expansion in the number of KSP repeats in NF-M 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 diameter would, at least, allow for conservation of conduction rates in mammals of differing sizes as is observed in mouse (conduction velocity ~50m/s) and humans (conduction velocity ~50m/s).