



Arginine methylation and QUAKING as regulators of CNS myelination



Oligodendrocytes are the myelinating cells of the central nervous system (CNS). I will present two different pathways that regulate oligodendrocyte differentiation impacting CNS myelination in mice. It is known that the quaking viable mice display tremors by post-natal day 10 due to a lack in oligodendrocyte differentiation. The qkI gene encodes several spliced isoforms of the QKI RNA binding proteins. QKI-5 is exclusively nuclear, while QKI-6 is distributed throughout the cell, and QKI-7 is predominantly cytoplasmic. The QKI isoforms interact with ACUAAY-(N1-20)-UAAY (QKI Response Element). I will discuss the phenotype of the conditional deletion of QKI isoforms leading to alternative splice regulation of axoglial proteins for oligodendrocyte development and maintenance in adults. Arginines can either be ω -N G-monomethylated, asymmetrically ω -N G,N G-dimethylated arginines or symmetrically ω -N G,N' G-dimethylated arginines by protein arginine methyltransferases. I will discuss the role of PRMT5 as a key regulator of CNS myelination and define its mechanism of action

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