

Glycosylation of the Cardiac Ion Channel Protein TRPM4 in Rat and Mouse Tissues

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The monovalent cation channel Transient Potential Melastatin 4 (TRPM4) depolarizes cells upon activation and is capable of initiating diverse intracellular signaling pathways. TRPM4 mutants have been shown to affect cardiac electrical conduction; thus, the protein is expected to play an important role in cardiac electrical conduction. Recent studies have demonstrated that the protein is N-linked glycosylated at a single residue, with the modification affecting stability and regulation. Research has been done using induced expression of TRPM4 in cultured cells, but few studies on native tissues exist. The aim of this paper is to determine the extent of TRPM4 expression and presence of N-glycosylation in various native rat and mouse tissues, specifically focusing on expression in heart tissues. Western blots were performed for all samples. Heart tissue samples were additionally deglycosylated with PNGase F, an enzyme specific for cleaving N-linked glycans. TRPM4 was shown to be present in all rat and mouse tissues studied, with tissues displaying different ratios of intracellular and membrane-integrated TRPM4 as well as different ratios of fully- and core-glycosylated protein. Additionally, the core-glycosylated variant was successfully deglycosylated, confirming that N-linked glycosylation of TRPM4 exists in native tissues.

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