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The thrombin/PAR1 axis as regulator of Schwann cell functions in health and disease

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Thrombin, the key effector protease of the coagulation cascade, mediates hemostasis, thrombosis, and inflammatory responses to vascular injury predominantly acting through its main receptor, protease-activated receptor 1 (PAR1). PAR1 is a member of a family of four G-protein-coupled receptors which are activated by proteolytic cleavage of their N-terminal extracellular domains. The expression and role of PAR1 in peripheral nervous system is still poorly investigated, although high PAR1 expression was found in the dorsal root ganglia and in the non-compacted Schwann cell myelin microvilli at the nodes of Ranvier.

Our previous results indicate that rat Schwann cell plasticity can be widely modulated by thrombin acting through PAR1 (Pompili et al., Mol and Cell Neurosci 2017; Pompili et al., Eur J Histochem 2020). Here we extend those previous data showing that thrombin regulates proliferation and survival of human Schwann cells increasing the expression of factors, such as matrix metalloprotease 2 (MMP2) and macrophage migration inhibitory factor (MIF), which are key in modulating nerve regeneration.

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