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Thrombin in the peripheral nervous system as regulator of Schwann cell neurotrophic potentials

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Coagulation and inflammation are tightly and reciprocally regulated. Inflammation initiates clotting, decreases the activity of natural anticoagulant mechanisms and impairs the fibrinolytic system. Thrombin is the main effector protease in hemostasis and it also plays a role in various non-hemostatic biological and pathophysiologic processes, predominantly mediated through activation of protease-activated receptors (PARs).

PAR-1 is the main thrombin receptor in peripheral nerves and it is highly expressed at the level of non-compacted Schwann cell myelin microvilli of the nodes of Ranvier.

After nerve crush thrombin is locally generated at the site of injury [1]. Thrombin generation is generally believed to have long-term beneficial effects for tissue repair. In this connection, our previous data indicate that PAR-1 activation on Schwann cells favors their ability to promote axonal regrowth after lesion [2]. On the other hand, it has also been reported that an excessive generation of thrombin can be detrimental for nerve functions. Our present data obtained by confocal and environmental scanning electron microscopy (ESEM) show that the morphology of the nodes appears to be deeply affected by high concentrations of thrombin (0.1-1 microM). In thrombin-treated nerves we also observe a redistribution of S100b to the paranodes and to the Schmidt-Lanterman incisures.

Controlling thrombin concentration may preserve neuronal health during nerve injury and represent a novel possible target for pharmacologic therapies.

References

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Key words

Schwann cells, peripheral nervous system, regeneration, thrombin, protesase-activated receptors.