VEGF-Mimetic peptide on a Fibrin-Heparin Coating Drives Fast Endothelialization of Blood-Contacting Surfaces

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Early and late thrombosis are major causes of failure of synthetic cardiovascular grafts, underscoring the for coatings that are simultaneously pro-endothelial.[1] antithrombogenic and developed a biofunctional surface based on a controlled fibrin mesh bearing immobilized heparin and presenting the VEGFmimetic peptide KLT. The coating was formed as a thin, continuous fibrin network that efficiently retained heparin and displayed KLT at the interface, providing a hemocompatible reservoir and an endothelialization cue without the need exogenous soluble growth factors. Upon contact with heparinized fresh human blood, the coated surfaces exhibited excellent antithrombogenic behavior, consistent with reduced formation relative to uncoated controls. Human umbilical vein endothelial cells seeded on the coating adhered rapidly, spread with a cobblestone morphology, and formed a confluent monolayer within 5 days, whereas only sparse colonies developed on uncoated surfaces. Enhanced endothelialization—manifested improved by adhesion, spreading, and proliferative coveragewas driven primarily by the interfacial presentation of KLT, which mimics VEGF signaling to promote endothelial responses, while immobilized heparin contributed to hemocompatibility of the blood-facing interface. The fibrin-heparin-KLT architecture remained stable throughout cell culture and handling, indicating practical robustness of the preparation. Together, these results indicate that a heparinized fibrin mesh presenting a VEGF-mimetic peptide provides a simple, modular route to combine antithrombogenicity with rapid endothelial coverage, and they support the potential of this strategy to enable self-endothelialization of blood-contacting graft materials in vivo.

References

[1] Táborská J., Riedelová Z., Brynda E., Májek P., Riedel T., RSC Advances, 11 (2021) 5903-5913.

Figures

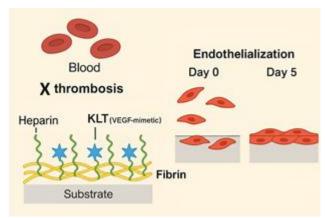


Figure 1. Scheme of the fibrin-heparin coating with immobilized KLT peptide and its effect on thrombogenicity and endothelialization.