Development of a Short Peptide to Prevent Non-Thrombogenic Clot Formation on Hydrophobic Surfaces

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Individuals with medical implants that interact with the circulatory system are at risk of developing life-threatening thrombi on the surface of these devices. The implementation of non-thrombogenic materials in medical implants is imperative in preventing this from occurring. An in silico human fibrinogen NJm model was implemented to investigate fibrinogen adsorption on a hydrophobic thermoplastic surface. Results are congruent with existing literature on hydrophobic surface binding and suggest that on a polystyrene (PS) surface, fibrinogen is positioned such that its aC domain is exposed. P-12 is a 14-peptide sequence derived from the Aa635-648 region of fibronectin (PSHISKYILRWRPK) that was shown to inhibit non-thrombogenic fibrin polymerization. A second in silico model was generated to confirm P-12 binds to all domains of fibrinogen, as experimentally observed. To evaluate the proficiency of P-12 in inhibiting non-thrombogenic fibrin polymerization on a hydrophobic surface, this study generated an in vitro bovine model of fiber formation on a PS surface. Large fibers were observed in the absence of P-12, while little to no fiber formation occurred when P-12 was added after the formation of the monolayer. This experiment confirms that P-12 blocks the ability of fibrinogen to recruit other proteins into fiber formation and that P-12 can displace fibrinogen from surfaces thus reducing efficiency. These results aligned with theoretical calculations. If medical implants are manufactured with hydrophobic surfaces, this study suggests the introduction of P-12 can be used to mitigate thrombosis.

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