Amino Acid-Based Polymers Coatings on Metallic Wires for Enhanced Nitric Oxide Delivery

Xinyue Zhang, Federico Mazur, and Rona Chandrawati*

School of Chemical Engineering and Australian Centre for Nanomedicine (ACN) The University of New South Wales Sydney, NSW, Australia

xinyue.zhang10@student.unsw.edu.au, rona.chandrawati@unsw.edu.au

Medical implants such as catheters, stents, and artificial prosthetics, are crucial for treating numerous conditions but often face complications including bacterial infections and thrombosis.¹ Localized nitric oxide (NO) delivery, leveraging NO's antibacterial, vasodilatory, and wound-healing properties, has emerged as a promising approach to address these challenges.² Coatings can be designed to deliver NO by physically carrying an NO donor molecule³ or catalytically generating it from endogenous sources.⁴ However, achieving prolonged, localized NO release while maintaining stable surface coatings on metallic implants remains a significant challenge. We have previously shown that amine-rich polymers like poly-L-lysine (PLL) can catalytically generate NO from endogenous NO donors like Snitrosoglutathione (GSNO).⁵ Here, we present a coupling-agent-based strategy for coating titanium wires with PLL to enable controlled NO release from GSNO (Figure 1). Using 3glycidyloxypropyltrimethoxysilane (GPTMS) as a coupling agent, we established robust, durable interfaces between the titanium surface and the PLL layer. This procedure minimized coating detachment even after washing, ensuring sustained NO release from NO donors, including GSNO. By optimizing GPTMS and PLL concentrations, we achieved consistent NO generation without compromising coating integrity. Our findings highlight the potential of GPTMS/PLL coatings to enhance antibacterial performance and biocompatibility in metallic implants. While these GPTMS/PLL coatings demonstrate enhanced stability, future investigations will centre on fine-tuning NO-release kinetics under physiologically relevant conditions and validating long-term performance in vivo, which are essential steps toward realizing clinically robust infection control and improved patient outcomes.



Figure 1: Schematic diagram of metallic implants demonstrating catalytic properties upon GPTMS/PLL coating towards endogenous prodrug degradation for nitric oxide generation.

References:

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