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The Impact of Immobilized Antithrombin III on the Thromboresistance of Polycarbonate Urethane

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Introduction

Thrombosis, the pathological formation of blood clots, remains one of the most significant complications associated with medical devices that come into direct contact with blood. These include a range of medical devices such as catheters, artificial heart valves, vascular grafts and blood pumps, all of which are essential in modern healthcare for treating cardiovascular and other critical conditions. However, the interaction of these devices with blood often activates the clotting cascade, leading to the formation of thrombi, which can obstruct blood flow, lead to device failure, or cause severe clinical complications such as stroke or pulmonary embolism. A key challenge in biomaterials used for blood-contacting devices is to minimize thrombosis while maintaining other essential properties such as mechanical strength, flexibility and long-term biocompatibility. Hence, the development of thromboresistant surfaces is critical to improving the safety and efficacy of such medical devices [1].

Poly Carbonate Urethane (PCU) has been widely used as a material for medical devices due to its excellent mechanical properties, such as high tensile strength, flexibility and biocompatibility. However, despite these advantages, PCU surfaces are not inherently resistant to thrombus formation when exposed to blood. This limitation stems from the polymer's surface characteristics, which can promote protein adsorption and platelet adhesion, key events that initiate clot formation. Consequently, there has been a concerted effort to develop surface modification strategies that enhance the thromboresistant properties of PCU without compromising its structural integrity [2].

Description

The preparation of Poly Carbonate Urethane (PCU) surfaces for ATIII immobilization begins with cleaning and treating the material to remove contaminants and ensure proper surface activation. Initially, PCU sheets are subjected to a plasma cleaning process to eliminate any organic contaminants, followed by surface activation techniques such as corona discharge or chemical grafting. These treatments introduce functional groups, such as amine or carboxyl groups, that are crucial for the covalent attachment of ATIII. Surface modification is critical because it not only enables the attachment of biomolecules but also influences the surface's interactions with blood proteins and cells [3].

Antithrombin III is immobilized onto the modified PCU surface through covalent bonding, using crosslinking agents like carbodiimide or glutaraldehyde. These chemical agents facilitate the formation of stable bonds between the functional groups on the PCU surface and the amino acids on the ATIII protein. The immobilization process is designed to retain the bioactivity of ATIII while ensuring it remains firmly anchored to the surface. This approach allows for a controlled density of ATIII molecules on the material surface, which is essential for maintaining effective anticoagulant activity [4].

Surface characterization is a crucial step in verifying the success of the immobilization process. Various techniques are employed to confirm the presence and stability of ATIII on the PCU surface. X-ray Photoelectron Spectroscopy (XPS) is used to analyze the surface composition and determine whether the expected chemical modifications have occurred. Fourier-Transform Infrared Spectroscopy (FTIR) is employed to detect the characteristic chemical bonds formed between the ATIII and the surface, confirming successful immobilization. Additionally, contact angle measurements are taken to assess changes in the hydrophilicity of the PCU surface, as a more hydrophilic surface typically enhances protein adsorption and reduces platelet adhesion. To evaluate the thromboresistance of ATIII-immobilized PCU, a range of in vitro blood compatibility tests are performed.

Clotting time assays are used to measure the time it takes for blood to form a clot on the surface of ATIII-immobilized PCU, compared to untreated PCU. Platelet adhesion and aggregation studies are carried out using Scanning Electron Microscopy (SEM) and confocal microscopy to visualize and quantify the number of platelets that adhere to and aggregate on the surface of both modified and unmodified PCU. Additionally, thrombin generation assays are conducted to assess how the immobilized ATIII affects thrombin activation in the presence of blood, providing insights into the anticoagulant activity of the surface modification under flow conditions. In vivo studies are also conducted to confirm the efficacy of ATIII-immobilized PCU in reducing thrombosis in a more complex physiological context. Animal models are used to implant the modified PCU materials and observe thrombus formation, inflammation and overall device performance over an extended period [5].

Conclusion

In conclusion, this study has successfully demonstrated that the immobilization of AntiThrombin III (ATIII) onto Poly Carbonate Urethane (PCU) surfaces significantly enhances their thromboresistance. The ATIII-immobilized PCU surfaces exhibited a marked reduction in clotting time, platelet adhesion and thrombin generation, suggesting that the immobilized ATIII effectively inhibits key steps in the coagulation cascade. Surface characterization confirmed the successful immobilization of ATIII and in vivo studies further validated the thromboresistant properties of the modified material. These findings contribute to the growing body of evidence that surface modifications with anticoagulant proteins like ATIII can improve the performance and safety of blood-contacting medical devices. The ability to impart thromboresistance to PCU surfaces without compromising their mechanical properties holds significant implications for the design of safer and more effective medical devices. The incorporation of ATIII into PCU materials could substantially reduce the risk of thrombus formation in devices that come into prolonged contact with blood, such as vascular grafts, heart valves and catheters. This would not only improve the clinical performance of these devices but also reduce the need for systemic anticoagulation therapy, which is associated with risks such as bleeding and hemorrhage.

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Conflict of Interest

None.

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