The side-chain chemistry of antifouling polymer brushes influences protein fouling and their hemocompatibility.

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Whenever an artificial surface comes into contact with blood, proteins are rapidly adsorbed onto its surface. This phenomenon, termed fouling, is then followed by a series of undesired reactions involving activation of complement or the coagulation cascade and adhesion of leukocytes and platelets leading to thrombus formation. Thus, considerable efforts are directed towards the preparation of fouling-resistant surfaces with the best possible hemocompatibility.[1]
We performed a comprehensive hemocompatibility study after heparinized blood contact with seven different polymer brushes prepared by surface-initiated atom transfer radical polymerization. We quantified the fouling resistance and analyzed thrombus formation and deposition of blood cellular components on the coatings. Moreover, we performed identification of the remaining fouled proteins via mass spectroscopy to elucidate their influence on the surface hemocompatibility (Figure 1). Compared with an unmodified glass surface, the grafting of polymer brushes minimizes the adhesion of platelets and leukocytes and prevents the thrombus formation. The fouling from undiluted blood plasma was reduced by up to 99%. Most of the identified proteins are connected with the initial events of foreign body reaction towards biomaterial (coagulation cascade proteins, complement component and inflammatory proteins). In addition, several proteins that were not previously linked with blood-biomaterial interaction were identified.[2]

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References

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Figures

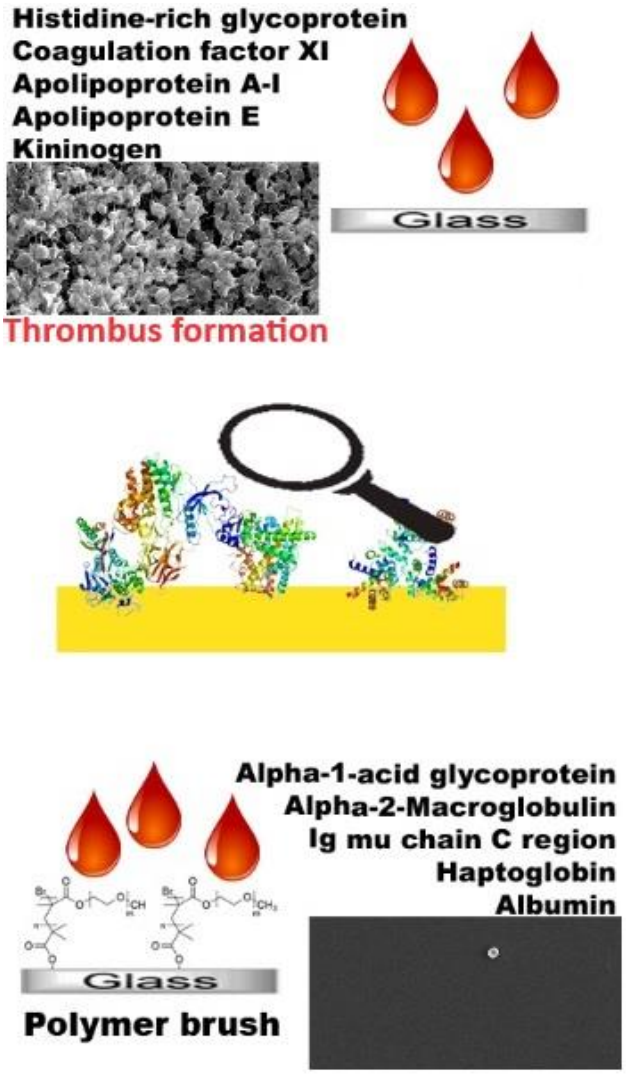

Figure 1. Hemocompatibility evaluation of polymer brushes and glass surface after heparinized blood contact and a following identification of the fouled proteins by LC-MS/MS.