Complement Activation Dramatically Accelerates Blood Plasma Fouling On Antifouling Poly(2-hydroxyethyl methacrylate) Brush Surfaces

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Non-specific protein adsorption (fouling) triggers a number of deleterious events in the application of biomaterials. Antifouling polymer brushes successfully suppress fouling,[1] however for some coatings an extremely high variability of fouling for different donors remains unexplained.[2] The authors report that in the case of poly(2-hydroxyethyl methacrylate) (poly(HEMA)) this variability is due to the complement system activation that causes massive acceleration in the fouling kinetics of blood plasma. Using plasma from various donors, the fouling kinetics on poly(HEMA) is analyzed and correlated with proteins identified in the deposits on the surface and with the biochemical compositions of the plasma. The presence of complement components in fouling deposits and concentrations of C3a in different plasmas indicate that the alternative complement pathway plays a significant role in the fouling on poly(HEMA) through the “tick-over” mechanism of spontaneous C3 activation. The generated C3b binds to the poly(HEMA) surface and amplifies complement activation locally (Figure 1). Heat-inactivated plasma prevents accelerated fouling kinetics, confirming the central role of complement activation. The results highlight the need to take into account the variability between individuals when assessing interactions between biomaterials and blood plasma, as well as the importance of the mechanistic insight that can be gained from protein identification.[3]

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References

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Figures

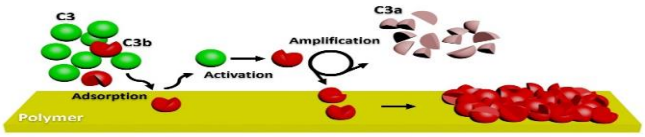

Figure 1. Proposed scheme of C3 activation on the poly(HEMA) surface. Spontaneously generated C3b molecules in the blood plasma adsorb on the surface and trigger the alternative complement pathway leading to a massive generation of C3b that fouls the surface.