

# Chemical Engineering Colloquium

September 29, 2021

12–12:50 p.m. | Goddard Hall, Room 227

## Polyelectrolyte Microgels as Self-Defensive Biomaterials

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Preventing the bacterial colonization of tissue-contacting biomedical devices is a key scientific challenge rich with potential clinical impact. Much research has concentrated on antimicrobial modification of device surfaces either by incorporating continuous elution mechanisms or by covalent surface tethering. Both strategies have shortcomings. As an alternative, we are studying a microgel-based approach to modulate the antimicrobial properties of surfaces. Notably, polyelectrolyte microgels can be electrostatically deposited to form sub-monolayer microgel coatings on complex 3-D devices. We have shown that the size and spatial distribution of antifouling PEG-based microgels critically controls the competing requirements for bacterial repulsion and tissue-cell adhesion/proliferation. Incorporating antimicrobials brings a second line of defense against bacterial colonization. To this end, we have concentrated on colloidal poly(acid) microgels such as poly(acrylic acid) (PAA) and poly(styrene sulfonate) (PSS) synthesized by suspension polymerization or membrane emulsification. After electrostatic deposition, their internal negative charge enables loading by cationic antimicrobials via a second self-assembly step driven by antimicrobial-microgel complexation. Our research concentrates on understanding and exploiting the polymer physical chemistry to identify microgel/antimicrobial combinations that can remain sequestered under physiological conditions for weeks or more, a time frame during which tissue-contacting devices are particularly prone to bacterial colonization. In addition to net electrostatic charge, aromaticity appears to play a significant role in determining the complexation strength. We have furthermore found that the sequestered antimicrobials can nevertheless be released when the loaded microgels are contacted by bacteria, a process that kills the bacteria. We speculate that this antimicrobial transfer is thermodynamically driven by the high concentration of negative charge and hydrophobicity associated with the bacterial cell envelope. Tissue cells such as osteoblasts and macrophages are, however, unable to similarly trigger release. Such bacteria-triggered release represents a new mechanism with which to create a so-called self-defensive surface - one that responds only when and where there is a bacterial challenge - with which to prevent device infection.



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