Biomaterial Interfaces
Room 321 - Session BI-TuA

Functional Materials and Biosensing

Moderators: Dr. Kenan Fears, U.S. Naval Research Laboratory, **Prof. Morgan Hawker**, California State University, Fresno

2:15pm **BI-TuA-1 Fibronectin and ECM-Mediated Regulation of Synovial Fluid Film Formation and Nanomechanics**, ***Roberto Andresen Eguiluz***, University of California Merced **INVITED**

Film formation is critical during locomotion to lubricate and protect the surface of articulated joints. The molecular components of the cartilage extracellular matrix (ECM) provide anchoring sites for lubricating molecules, thus serving as a platform for the assembly of the load/energy-dissipative films. The role of the collagen family in mediating the adsorption of lubricating and wear-protecting molecules, such as lubricin or hyaluronan, has been studied. However, the role of fibronectin (Fn) and its various conformations in regulating the adsorption of synovial fluid (SF) components and the wear-protecting properties of SF has been overlooked. During this talk, I will present our research efforts to understand the roles of various ECM components in controlling the supramolecular assembly of SF-derived films and SF components, and to gain insights into the synergies that regulate synovial joint surface nanomechanics. For this end, we employ experimental tools that include the surface forces apparatus (SFA), quartz crystal microbalance with dissipation (QCM-D), and atomic force microscopy, which I will briefly introduce.

2:45pm **BI-TuA-3 Magnetic Microparticle Biointerfaces for Fluorescence-Based Detection of C-Reactive Protein in Biological Fluids**, *Pegah Jamali, Zia Syed,* ***Umer Hassan,*** Rutgers, The State University of New Jersey

Biofunctionalized particle surfaces are useful model interfaces for studying biomolecular recognition while also enabling compact biosensing formats. In this work, we developed an antibody-presenting magnetic microparticle interface for fluorescence-based detection of C-reactive protein (CRP), an inflammatory biomarker associated with infection. The study focuses on the design and characterization of the bead–biomolecule interface, including antibody immobilization, target capture, fluorescence signal generation, and matrix-dependent assay response in biological fluids.

Carboxylated magnetic microparticles were chemically activated using EDC/NHS chemistry to create reactive surface groups for covalent antibody attachment. Anti-human CRP antibodies were then immobilized on the particle surface to form a biomolecular capture interface. The functionalized particles were incubated with FITC-labeled recombinant human CRP prepared across a concentration range of 0.5–100 µg/mL. After magnetic washing, bead-associated fluorescence was measured by optical microscopy, and image-based quantification was performed using background-corrected mean intensity analysis. This workflow links the surface density and stability of antibody-functionalized microparticles with the resulting optical response from captured protein.

The particle biointerface produced a concentration-dependent fluorescence signal in PBS buffer, human serum, and human urine. Calibration curves showed strong analytical linearity in all three matrices, with regression fits of $y = 54.10 + 1.12x$, $R^2 = 0.98$ in PBS; $y = 58.95 + 1.35x$, $R^2 = 0.98$ in serum; and $y = 13.0 + 0.32x$, $R^2 = 0.95$ in urine. The assay achieved a limit of detection of 0.5 µg/mL across all matrices. Serum produced the strongest optical response, while urine showed reduced sensitivity, highlighting the role of fluid composition, ionic environment, and nonspecific matrix effects on antibody–antigen recognition at the bead surface. Surface-conjugation stability was also evaluated over 28 days at 4°C and room temperature, with approximately 90% of the initial fluorescence signal retained, supporting the robustness of the covalent antibody–particle interface.

These results demonstrate a functional magnetic microparticle biointerface for CRP capture and fluorescence microscopy readout in complex biological environments. The platform is relevant to biomaterial-interface engineering, optical characterization of biofunctional surfaces, and particle-based biosensing, with future potential for integration into microfluidic workflows for rapid inflammatory biomarker testing.

3:00pm **BI-TuA-4 Potassium-Selective Nanoelectrode Arrays for Single-Cell Profiling of human iPSC-Derived Cardiomyocytes**, ***Dhivya Pushpa Meganathan***, *Joseph Wang,* University of California San Diego*; Zeinab Jahed Zeinab Jahed,* University of California at San Diego

Potassium ion (K^+) dynamics are central to cardiac electrophysiology, with early disruptions in K^+ flux often preceding arrhythmia and contractile dysfunction. However, current sensing technologies, such as patch-clamp, Microelectrode arrays (MEAs), and fluorescent indicators, either lack chemical specificity for K^+ or are unsuitable for long-term, single-cell analysis. Conventional ion-selective electrodes (ISEs), while more selective, are limited by bulk-phase design and poor spatial resolution. To address these limitations, we present KINESIS (K^+-Ion Nano-Electrode Selective Interface System), a nanofabricated, cell-compliant platform that enables direct, label-free potentiometric measurement of K^+ gradients with subcellular precision. KINESIS features high-aspect-ratio nanopillars coated with a valinomycin-based K^+ recognition membrane, forming a stable, non-invasive interface with human induced pluripotent stem cell-derived cardiomyocytes (hiPSC-CMs). This architecture allows localized, Nernstian sensing of K^+ efflux or depletion without disrupting cell membranes. Pharmacological validation shows distinct potential shifts in response to caffeine and ouabain. KINESIS thus offers a highly selective, spatially resolved approach for studying K^+ handling in cardiotoxicity screening and patient-specific disease modeling.

3:15pm **BI-TuA-5 Peptide Biosensor for Pathological Tau Detection in Neurodegenerative Disease**, ***Megan Pitz***, *Rebecca Mickol, Hugo Miranda Quijada, Sean Brown, Kenan Fears, Maryssa Beasley,* US Naval Research Laboratory

Tauopathies are a class of neurodegenerative diseases that are characterized by an abnormal aggregation and accumulation of the tau protein. Included in this class are Alzheimer's disease (AD), chronic traumatic encephalopathy (CTE), corticobasal degeneration (CBD), and Pick's disease (PiD), among others. Definitive diagnosis of many of these diseases is limited to post-mortem analysis of brain tissue, due to a lack of medical technologies allowing diagnosis during the patient's lifetime. However, diagnosis of these diseases ante-mortem would allow for much more targeted care and treatment for these patients. Therefore, there is a compelling need for technologies that can identify tau accumulations ante-mortem with high specificity. Here, we present a peptide-based biosensor system with the goal of detecting pathological tau aggregations in biological samples ante-mortem. One limitation in developing tau biosensors is the difficulty in producing the tau protein in laboratory settings. Consequently, in this project, we first explored recombinant expression of dGAE, a truncated form of tau which is responsible for protein aggregation. This protein segment has been historically difficult to express due to its high propensity for aggregation, interfering with efficient recombinant expression and purification. We explored three different purification tags to decrease aggregation after protein expression and compared total protein yields across expression and purification conditions. The second aim of this project explored a detector peptide system for identifying tau aggregations. A de novo peptide sequence was designed and evaluated via molecular dynamic simulations for its ability to selectively identify tau aggregates using a molecular probe. Preliminary results show the detector peptide aligning and changing conformation in the presence of tau aggregates, suggesting a peptide system capable of acting as a biomolecular probe for identifying pathological tau accumulation.

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