algorithm to three types of protein spectroscopy data: small-angle X-ray scattering, fluorescence, and electron paramagnetic resonance. In each case, the SVD algorithm returns two physically realistic basis states as well as the relative amounts of protein in the sample in each state.

Atomic Force Microscopy

876-Pos Board B676
Universal Non-Equilibrium Elastic Behavior of Macromolecules
Christian Kappel, Nicole Dölker, Mareike Zink, Ulrich Zachariae, Helmut Grubmüller.
Single molecule experiments such as atomic force microscopy, optical tweezers or force probe molecular dynamics have been used extensively to determine mechanical properties of biological macromolecules. In most cases, yielding or unbinding forces were found to depend on the loading rate according to Bell's model. The one-dimensional model predicts a logarithmic dependence of yielding forces on the loading rate, but velocity-independent elastic constants. Also more recent, refined one-dimensional treatments share this property. However, in recent molecular dynamics simulations of the nuclear transport receptor importin-beta and the outer shell of the southern bean mosaic virus, strongly velocity dependent elastic constants were seen, with yielding forces following Bell's model.
To resolve this discrepancy, we here present a two-dimensional relaxation model and show that it can explain all elastic properties seen in the atomistic simulations. In our model, relaxation modes perpendicular to the reaction coordinate are allowed to fluctuate and relax independently of the reaction coordinate. Contrary to Bell's model, a stiffening of macromolecules at high probe velocities can be explained, and the corresponding forces are correctly predicted. Combined with simple rate theory, our model also predicts the observed logarithmic dependence of yielding forces on the probe velocity.

877-Pos Board B677
Probing the Interactive Forces Between Bacterial Biofilm Cells and Surfaces with Atomic Force Microscopy
Anne E. Murdaugh, Kathy Aidala, Megan A. Ferguson, Eileen M. Spain, Megan E. Núñez.
Biofilms are complex aggregations of microbes formed at interfaces. An understanding of the initial formation, subsequent accumulation, and eventual eradication of bacterial biofilms is becoming increasingly important to a wide range of fields. In this study we use atomic force microscopy (AFM) to determine the cellular spring constants of intact, living Gram-negative and Gram-positive bacteria in simple biofilms. We also explore the mechanical characteristics of the bacterial predator Bdellovibrio bacteriovorus in comparison to that of its prey, E. coli, and the biophysical changes that take place during predation and consumption of the prey. In an effort to further understand the intra- and inter-bacterial interactions critical for biofilm formation and dynamics, we modify a force probe with live predator and prey cells. Using this ''biotip,'' we monitor in situ the attractive and adhesion forces between various cells, biofilms, and surfaces.

878-Pos Board B678
Molecular Mechanism for the Induction of Mesothelioma by Asbestos
Eric S. Taylor, Steven K. Lower, Brooke T. Mossman, Ann G. Wylie.
Mesothelioma is an incurable form of cancer located in the pleural lining around the lungs. It is associated almost exclusive with the inhalation of mineral fibers called asbestos. Despite the well-known association between asbestos and mesothelioma, little is know about the mechanism of induction. The binding of asbestos to epidermal growth factor receptor (EGFR), a transmembrane signal protein, has been proposed as a trigger switch for downstream signaling of kinases and expression of genes associated with cancer. Here we used atomic force microscopy (AFM) to investigate the binding of the extracellular domain of EGFR to two minerals, crocidolite versus riebeckite in buffer solution. Crocidolite [$Na_2(Fe,Mg)_5Si_8O_{22}(OH)_2$] is the asbestiform mineral most commonly associated with human mesothelioma. Riebeckite is chemically equivalent to crocidolite but has a non-asbestiform, fragmental habit, and it is not associated with cancer. AFM measurements revealed an attractive force between EGFR and each mineral. The rupture force of the mineral-EGFR bond increased with increasing loading rates. The correlation between rupture force and loading rate was stronger for crocidolite-EGFR than riebeckite-EGFR. The Bell model was used to estimate the bond lifetime for both mineral-protein pairs. The lifetime for the EGFR bond was roughly three times shorter for crocidolite than riebeckite. The fast but repeatable binding of EGFR with crocidolite fibers, which persist in the lungs for a period longer than a human's lifespan, may continually trigger the activation switch leading to chronic expression of genes involved in asbestos-related cancers.

879-Pos Board B679
Identification of oxLDL Components Affecting Endothelial Stiffness
Tzu Pin Shentu, Dev K. Singh, Papasani Subbaiah, Michael Cho, Irena Levitan.
Endothelial biomechanical properties have been suggested to play important roles in multiple endothelial functions. Our earlier studies have shown that endothelial stiffness is significantly increased by oxidized modifications of low-density lipoproteins (oxLDL). The goal of the current study was to identify the bioactive oxLDL components that are responsible for this effect. Our observations show that endothelial stiffness increases with progressive oxidation of LDL with strongly oxidized LDL having a larger effect than mildly oxidized LDL. Next, we systematically tested all the lipid fractions and the major bioactive lipid components of oxLDL for their effects on endothelial stiffness. We show here that the two fractions that contribute to the increase in endothelial stiffness are oxidized phospholipids (oxPC) and oxysterols. Furthermore, we show that oxidized 1-palmitoyl-2-arachidonoyl-sn-glycero-3-phosphocholine (oxPAPC), a major oxPC component, and 7 ketocholesterol and 7beta-hydroxycholesterol, the two major oxysterol components of oxLDL, play the key roles in endothelial stiffening. In addition, 27 hydroxycholesterol, a minor component of oxLDL, that is abundant in atherosclerotic lesions also induces an increase in endothelial stiffness. We also find that oxPAPC- and oxysterol-induced endothelial stiffening is fully reversible by cholesterol supplement suggesting that this effect should be attributed to changes in the lipid composition of the membrane. Indeed, we also show that significant amounts of oxidized PC products and oxysterols, specifically, 7-ketocholesterol accumulate in the membranes of endothelial cells exposed to oxLDL.

880-Pos Board B680
Ucp-1 from Pantoea Stewartii Affects Bacterial Ultrastructure and Flocculation
Tanya E.S. Dahms, John Stavrinides, Cheghaf Madarati.
Aphids are dependent on plants for nutrients and thus their gut microbiome often includes plant pathogens. Recently *rhs* (rearrangement hotspot) genes, implicated in insect-pathogen interactions, were discovered in the plant pathogen *P. stewartii* (Stavrinides *et al.* (2010) *Environmental Microbiology* 12, 147-155.). In *P. stewartii*, the *rhs* gene product, named Ucp-1 (youcannot pass 1), is responsible for conferring flocculating behaviour in the gut of the pea aphid, *Acyrthosiphon pisum*, ultimately resulting in its death. Absence of the gene in *P. stewartii*, gives rise to a mutant that is non-flocculating and non-lethal to the aphid, and transfer of the gene to *Escherichia coli* transforms it into a flocculating pathogen of aphids.
Here we use atomic force microscopy (AFM) and force spectroscopy (FS) to study the flocculating patterns, and cell surface ultrastructure, physical and chemical properties of wild type and *E. coli* cloned and over-expressing one of seven Ucp1-like proteins from the *Pantoea* genome. The Ucp-1 mutant, capable of transforming *E. coli* into a flocculating pathogen, has a distinct aggregation pattern and cell surface ultrastructure.

881-Pos Board B681
Effect of Cholesterol and Lipid Charge on Amyloid Fibril Formation on Model Lipid Membrane
Elizabeth Drolle, Francis Hane, Erin Faught, **Zoya Leonenko**.
Amyloid-beta 1-42 is known to form amyloid fibrils which are involved in Alzheimer's disease for which no cure is currently available. Normally an alpha helical structure, Amyloid-beta 1-42 can misfold into beta sheets forming neurotoxic oligomers and amyloid fibrils. Although fibril plaque formation is associated with biological membranes *in vivo*, the role of membrane heterogeneity and effect of cholesterol in the process of amyloid fibril formation and toxicity are not well understood, and therefore research in this area is of great interest and necessity. We used atomic force microscopy (AFM) to study Amyloid-beta fibril formation on the surface of lipid membrane. On neutral lipids, DPPC and DOPC the rate of adsorption and aggregation increases steadily as a function of time, whereas with charged lipids, DOPG and DOTAP adsorption happens much faster indicating stronger interaction with membrane. The surface of the lipid membrane becomes more disrupted in the case of positively charged DOTAP membrane. We also found that cholesterol induces domain formation in model membrane, which serve as a template for amyloid fibril formation.