

## BP 50: Posters - Cell Adhesion

Time: Wednesday 17:00–19:00

Location: Poster C

BP 50.1 Wed 17:00 Poster C

**Adhesion of oral bacteria to titanium surfaces of different roughness measured by single-cell force spectroscopy** — ●KORDULA SCHELLNHUBER, CHRISTIAN SPENGLER, NICOLAS THEWES, and KARIN JACOBS — Department of Experimental Physics, Saarland University, 66041 Saarbrücken

Bacteria adhere to virtually all surfaces and promote the formation of biofilms. In the oral cavity, these initial biofilms may lead to plaque and consequently to severe health problems. Therefore, understanding and controlling the process of bacterial adhesion to oral implants is of great importance for material science and medicine. A common material for dental prostheses is titanium due to its robustness and high biocompatibility with various tissues. We use single-cell force spectroscopy to study the adhesion of *Streptococcus mutans* to titanium surfaces of different roughness. In addition, we compare the bacterial adhesion to titanium with the adhesion to very smooth samples of hydroxyapatite, the mineral component of teeth. Furthermore, we investigate the adhesion of *Streptococcus mutans* to real oral biofilms of different ages.

BP 50.2 Wed 17:00 Poster C

**Single cell force spectroscopy and initial cell adhesion** — ●HENDRIK BREHME, PHILIPP WYSOTZKI, MARCO STUBBE und JAN GIMSA — Universität Rostock, Lehrstuhl für Biophysik, Gertrudenstraße 11a, D-18057 Rostock, Deutschland

For medical implants, there is a general risk for so-called "implant-associated infections". The starting point for a possible infection is the "race for the surface", in which bacteria and body cells compete in making initial contacts to the implant's surface. Bacteria which may successfully colonize the surface tend to form biofilms, which can hardly be fought by the immune system of the patient. In the experiments, the individual behavior of single cells during their initial adhesion was investigated using single cell force spectroscopy. For this, cells of the bone cell line MC3T3 or bacteria were attached to the cantilever and their initial adhesion behavior to different surfaces was measured using a NanoWizard II atomic force microscope (JPK, Berlin).

The setpoint and adhesion forces were correlated with the electric impedance of a microelectrode structure for single cell detection (single cell-interdigitating electrode structure; SC-IDES).

BP 50.3 Wed 17:00 Poster C

**Substrate elasticity and ligand affinity affect traction force evolution** — ●CHRISTINA MÜLLER and TILO POMPE — Institute of Biochemistry, Universität Leipzig, Germany

Mechanotransduction is known as one control mechanism for several basic cell functions, like proliferation, differentiation and cell death. We investigated early cell adhesion on hydrogels with an independent variation of substrate stiffness and affinity of the adhesion ligand fibronectin to the hydrogel surface. Thin film coatings of maleic acid copolymers on top of polyacrylamide hydrogel layers were fabricated to tune protein binding. The Young's modulus of the hydrogel was modulated between 2.5 kPa and 9 kPa. Human umbilical vein endothelial cells were monitored during the first two hours of cell adhesion by time-resolved cell traction force microscopy. Three different regimes of traction force generation were found. In the first regime (R0) cells spread fast, but traction forces were negligibly small. Regime R1 is characterized by a decelerated spreading and a succeeding force increase. After completion of spreading cells enter regime R2 with saturated forces. Substrate stiffness ligand and affinity were both found to affect the kinetics and absolute levels of traction force quantities. A faster increase and a higher saturation level of traction forces were observed for a higher substrate stiffness and a higher ligand affinity. The results show that cells perform varying proportions of work against conservative and dissipative forces. Finally, our findings complement recent modeling approaches and contribute to a better understanding of the dynamics of cell adhesion on viscoelastic substrates.

BP 50.4 Wed 17:00 Poster C

**Size, kinetics and free energy of clusters formed by ultra-weak bonds between glycolipids** — ●HANNES WITT<sup>1</sup>, MARIEELEN OELKERS<sup>1</sup>, FILIP SAVIC<sup>1</sup>, SHAHID I. AWAN<sup>2</sup>, DANIEL B. WERZ<sup>2</sup>, BURKHARD GEIL<sup>2</sup>, and ANDREAS JANSHOFF<sup>2</sup> — <sup>1</sup>Georg-August-

Universität Göttingen — <sup>2</sup>Technische Universität Braunschweig

Many biological processes, like cell motility or immunological recognition, rely on transient binding, which demands the use of weak, non-covalent bonds with fast binding and unbinding kinetics, characteristics ideally met by carbohydrate-carbohydrate-interactions (CCI). Here we employ atomic force microscopy (AFM) to study the trans-interaction of the trisaccharide Lewis X bound to a fluid lipid membrane. We show how even this ultra-weak interaction leads to the formation of binding clusters resulting in biologically relevant adhesion forces and describe this process with a simple diffusion-reaction-scheme, which allows us to access the bond number, reaction kinetics and free binding energies.

BP 50.5 Wed 17:00 Poster C

**Dynamics of actin stress fiber patterns in laterally constrained cells** — ●ANDREAS MÜLLER and TILO POMPE — Universität Leipzig, Institute of Biochemistry, Johannisallee 21-23, 04109 Leipzig, Germany

Living cells are subjected to many physical cues, such as viscoelasticity of the environment and spatial constraints. The latter holds especially true for cells in multicellular, compartmentalized organisms, like us. In previous work we found a bimodal behavior with regard to the formation of exterior and interior stress fibers and their spacing for human umbilical vein endothelial cells under lateral constraints [1].

We now observed a high robustness of this bimodal behavior against changes in biophysical and biochemical parameters, including substrate stiffness, also within cell types of higher contractility. We found that inhibition of myosin activity with blebbistatin or inhibition of ROCK with Y-27632 only lead to minor perturbations in the bimodal behavior. Furthermore, traction forces and strain energies of laterally confined cells were only slightly attenuated with increasing constraint and showed no apparent correlation to the two distinct actin cytoskeleton morphologies. Live cell imaging of stress fiber patterns revealed a fast switching between different stress fiber states within minutes, indicating that cells under lateral constraints permanently adapt to their surroundings.

[1] Müller, A., Meyer, J., Paumer, T., Pompe, T. Cytoskeletal Transition in Patterned Cells Correlates with Interfacial Energy Model. *Soft Matter*, 2014, 10, 2444-2452.

BP 50.6 Wed 17:00 Poster C

**Microbial adhesion on nanorough titanium: Insight into the nanostructure of the microbe-material-interface** — ●CLAUDIA LÜDECKE-BEYER<sup>1,3</sup>, MARTIN ROTH<sup>2,3</sup>, JÖRG BOSSERT<sup>1,3</sup>, and KLAUS D. JANDT<sup>1,3</sup> — <sup>1</sup>Otto Schott Institute of Materials Research, Chair of Materials Science, Friedrich Schiller University, Jena, Germany — <sup>2</sup>Leibniz Institute for Natural Product Research and Infection Biology, Bio Pilot Plant, Hans Knöll Institute, Jena, Germany — <sup>3</sup>Jena School for Microbial Communication (JSMC), Excellence Graduate School, Friedrich Schiller University, Jena, Germany

Implant-associated infections are primarily initiated by the adhesion of microorganisms on the implants' surfaces. Recently, materials with surface roughnesses in the nanometer range gained interest to reduce microbial adhesion, however, the mechanisms remained so far unclear. The aim of this study was to explore the unknown nanostructure of the microbe-titanium-interface to gain understanding of the physical mechanism of microbial adhesion as a function of nanoroughness. Microbial adhesion was investigated using physical vapor deposited titanium thin films as nanorough 2D model surfaces. We found evidence that with decreasing titanium surface peak density and decreasing specific titanium surface area the surface coverage with microbes was reduced. Investigating the structure of the microbe-material-interface indicated that the initial adhesion of the microbes is controlled by the number of nano contact points between the microbial cell and the material's surface. These findings support the development of new antibiotic-free strategies to prevent implant-associated infections.

BP 50.7 Wed 17:00 Poster C

**Nano contact points control initial microbial adhesion on biomaterials surfaces** — ●CAROLIN DEWALD<sup>1,2,3</sup>, CLAUDIA LÜDECKE-BEYER<sup>1,3</sup>, MARTIN ROTH<sup>2,3</sup>, JÖRG BOSSERT<sup>1,3</sup>, and KLAUS D. JANDT<sup>1,3</sup> — <sup>1</sup>Otto Schott Institute of Materials Research, FSU Jena,

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New antibiotic-free strategies are needed for prevention of biomaterials-associated infections. Our preliminary results indicated that microbial adhesion is controlled by the nano contact points between the microbes and the material's surface. The aim of this study was to use nanoparticles (NPs) for physically structuring materials' surfaces to modulate the number of possible contact points between the microbes and the nanostructured surfaces. 15 nm gold NPs were immobilized in different

concentrations to gold surfaces. Microbial adhesion on these surfaces as well as the nanostructure of the microbe-material-interface was investigated to gain understanding of the physical mechanisms of microbial adhesion as function of the material's nanostructure. With decreasing NP density, microbial adhesion was statistically significantly reduced. High resolution SEM revealed that the NPs controlled the initial contact between the microbial cells and the material's surface. We assume that the total adhesion strength is correlated with the NP density i. e. the contact point density. Our new findings suggest that adjusting the nanostructure of biomaterials' surfaces might be a promising antibiotic-free approach for controlling microbial adhesion.