MEASURING BIOPHYSICAL PROPERTIES OF ACTIVATED MONOCYTIC CELLS BY CAPILLARY MICROMECHANICS

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Introduction
During inflammation processes, monocytes undergo activation which results in significant rearrangement of the cytoskeletal actin network and, consequently, change of cell mechanical properties [Vicente-Manzanares, 2004]. Cell deformability might be used as a biomarker for chronic inflammatory diseases. The aim of the study was to quantify changes in cell mechanical properties of activated and non-activated cells and actin-disrupted cells, by measuring pressure-induced cell deformation through a tapered glass microcapillary.

Methods
HL-60 (human acute promyelotic leukaemia) cells were used as a model system for monocytes [Fleck, 2005]. Chronic inflammation was mimicked by stimulation with lipopolysaccharide (LPS, 15 min, 2 μg/ml). Actin structure was disrupted by exposure with Cytochalasin-D (Cyto-D, 20 min, 4 μM).

The deformation device [Wyss, 2011] consisted of a tapered glass capillary connected to a flexible tube (Fig.1). While the cell suspension was flown into the device, a cell became lodged in the tapered capillary. Both compressive (K) and shear (G) moduli were calculated based on cell deformation.

Results
Cell mechanical properties depend on the amount of deformation, i.e. the behaviour is non-linear (Fig 2). For small deformation, LPS-treated cells have higher compressive modulus compared to non-treated cells. However, LPS-treated cells show a decrease in compressive modulus as deformation increases. CytoD-treated cells show lower moduli.

Discussion
At small deformation, activated cells appear stiffer due to polymerization of cortical actin after activation. At high deformation, the compressive modulus of LPS-treated decreases. This might be due to disruption of cytoskeleton by increased stress or to remodeling of structures as in monocyte diapedesis. Cyto-D treated cells have lower moduli due to disruption of the actin structure. Mechanical properties of monocytes can be quantified using the microcapillary device, and are distinctly dependent on treatments that modify cells’ cytoskeleton.

References
R.A. Fleck et al., Clinical and Diagnostic Laboratory Immunology, 2005