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Cell mechanics and immune system link up to fight infections ANDREW EKPENYONG, Dept. of Physics, Creighon University, SI MING MAN, PANAGIOTIS TOURLOMOUSIS, SARRA ACHOURI, EUGENIA CAMMAROTA, KATHERINE HUGHES, ALESSANDRO RIZZO, GILBERT NG, Univ of Cambridge, UK, JOCHEN GUCK, Technical Univ. Dresden, Germany, CLARE BRYANT, Univ of Cambridge, UK — Infectious diseases, in which pathogens invade and colonize host cells, are responsible for one third of all mortality worldwide. Host cells use special proteins (immunoproteins) and other molecules to fight viral and bacterial invaders. The mechanisms by which immunoproteins enable cells to reduce bacterial loads and survive infections remain unclear. Moreover, during infections, some immunoproteins are known to alter the cytoskeleton, the structure that largely determines cellular mechanical properties. We therefore used an optical stretcher to measure the mechanical properties of primary immune cells (bone marrow derived macrophages) during bacterial infection. We found that macrophages become stiffer upon infection. Remarkably, macrophages lacking the immunoprotein, NLR-C4, lost the stiffening response to infection. This in vitro result correlates with our in vivo data whereby mice lacking NLR-C4 have more lesions and hence increased bacterial distribution and spread. Thus, the immune-protein-dependent increase in cell stiffness in response to bacterial infection (in vitro result) seems to have a functional role in the system level fight against pathogens (in vivo result). We will discuss how this functional link between cell mechanical properties and innate immunity, effected by actin polymerization, reduces the spread of infection.

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