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A THEORETICAL ANALYSIS FOR THE EFFECT OF SUBSTRATE ELASTICITY ON CELLULAR ADHESION

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ABSTRACT

Cell behavior is mediated by variety of physiochemical properties of extracellular matrix (ECM). Material composition, surface chemistry, roughness, and distribution pattern of cell adhesive proteins are among the ECM properties which are known to modulate various cellular physiological functions. Mechanical stiffness of ECM in particular is found to be a major regulator for multiple aspects of cellular function. Experiments show that cells in general, exhibit an apparent adhesion preference for stiffer substrates with a larger projected spread area with increasing the substrate stiffness. In addition, it seems that the effect of substrates elasticity is strongly coupled with adhesivity of the substrate; on relatively stiff substrates the spread area of the cells exhibits strong biphasic dependence to the changes in ligand density, whereas on soft substrates their limited spreading is much less sensitive to the density of surface ligands.

This study aims to propose a theoretical basis for the interplay between substrate elasticity and cellular adhesion, using an equilibrium thermodynamic model. Within this framework, the equilibrium contact area is assumed to ensure minimization of the free energy contributed by interfacial adhesive and repulsive interactions between the membrane and substrate as well as the deformation of cell and substrate. Hence, this thermodynamic model overlooks the contribution of intracellular signaling or actively regulated cytoskeleton and assumes that cell adhesion is solely a result of the balance between the membrane-substrate repulsive potentials, stored elastic energy, binding enthalpy, and mixing entropy of mobile receptors. The predictions of this purely *mechanistic* model for cell adhesion qualitatively follow the experimental results featuring the variation of cell spread area on compliant bio-adhesive substrates. This suggests that the mechanistic pathways inherent to membrane-substrate interactions may be equally important as intracellular signaling pathways to mediate the cellular adhesion.

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