XXIII CONVEGNO GRUPPO ITALIANO DI MECCANICA COMPUTAZIONALE

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II CONVEGNO GRUPPO BIOMECCANICA

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Paolo Fuschi
Aurora Angela Pisano
Editors

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Durotaxis of tensegrity cell units incorporating asymmetry

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The present contribution focuses on some recent results obtained by the authors concerning durotaxis of cell units anchored to the substrate through focal adhesion plaques, a phenomenon which is relevant to cell locomotion [1]. A mechanical pre-strained tensegrity model obeying a Neo-Hookean stress-strain law recently devised by the authors [2, 3, 4] is exploited to investigate how substrate stiffness gradients and asymmetric geometry affect the cell contractility and the growth of the focal adhesion plaque. The cytoskeleton is purposely reduced to its main components, that is actin filaments and microtubules forming a contractile mechanical system obeying the so-called tensegrity self-equilibrium principle [5]. The system contraction is triggered by means of inelastic pre-strains, that simulate pre-contraction and polymerization. In the adopted tensegrity, an element representative of the actomyosin complex is taken in parallel with another element corresponding to the microtubule [2, 3]. The former can only elastically elongate or inelastically contract without bending, while the latter is a compression-bearing buckling-prone element that can also polymerize.

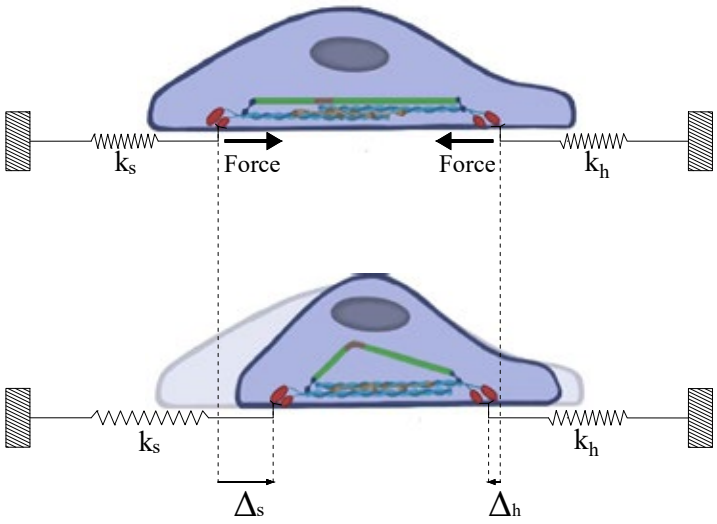

Figure 1: A contractile cell where the cytoskeleton is replaced by the adopted tensegrity and the cell contractility induces two equal forces at the leading and trailing edges of the cell where the plaques of the focal adhesions are located.

A scheme of the adopted mechanical framework is shown in Figure 1. The cell contractile activity produces two equal forces at the leading and trailing edges of the cell where the plaques of the focal adhesions are considered to be located. These forces, in turn, induce a thermodynamically consistent polymerization/depolymerization process of the focal adhesion plaques. As an effect of the mechanosensitivity of the devised structural system, the displacement Δ_s at the edge lying upon the softer substrate will be generally different from Δ_h detected at the edge placed on the hard part of the substrate. The cell net displacement Δ_N can be hence computed as [1] $\Delta_N = \Delta_s - \Delta_h$. The contractile system illustrated in Figure 1 exemplifies the positive durotaxis concept with the cell advancing towards the stiffer side. However, recently, it has been observed that some cells may migrate towards softer substrates [6], thus suggesting the existence of a negative durotaxis, or mollitaxis, effect.

The present contribution shows that both "classical" positive durotaxis and more recently unveiled mollitaxis can be retraced by means of the proposed essential model, the switching from one mechanism to the other depending on the combination of geometrical asymmetry, stiffness gradients, and inelastic pre-strains. Advantageously, the present model allows us to parametrically investigate the effect of a wide range of asymmetric configurations and stiffness gradients on the cell kinematics and how these affect the process of assembly and disassembly of the focal adhesion plaques subjected to the force exerted by the system.

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