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Françoise Argoul

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From Elasticity to Inelasticity in Cancer Cell Mechanics: A Loss of Scale Invariance

Francoise Argoul.

Laboratoire Ondes et Matière d'Aquitaine, Talence, France.

Soft materials such as polymer gels, synthetic biomaterials and living biolog-ical tissues are generally classified as visco-elastic or visco-plastic materials, because they behave neither as pure elastic solids, nor as pure viscous fluids. When stressed beyond linear viscoelastic regime, cross-linked biopolymer gels can behave nonlinearly (inelastically) up to failure. In living cells these networks exhibit high sensitivity to stress and propensity to local failure (plas-ticity). However, unlike synthetic passive gels, living cells can dynamically compensate these failures thanks to ATP driven reparation mechanisms and recover a sustainable networked cytoskeleton architecture, as long as they are kept in a suitable culture environment. When the failure events are too frequent or too strong or when the reparation mechanisms are not efficient, irre-versible losses of mechanical homeostasis and chronic diseases such as cancer may ensue. Here, we concentrate on chronic myelogenous leukemia (CML) as a model for stem cell cancer transformation. We compare the mechanical abil-ity of both primary (healthy donors and patients) and immature cells (TF1) transduced by the CML oncogene BCR-ABL to respond to mechanical stresses (nano-indentation experiments). Our experimental study shows that primary CML (CD34+) hematopoietic cells encounter more frequent brittle failure events than normal ones. This failure enhancement is also accompanied by a modification of the scale-invariant rheologic property of these cells. Interest-ingly, stiffening induced failure events are more visible on primary than on TF1 model cells, pointing out that their cross-talk with their native microenvi-ronment may also affect their transformation. Finally, combining these exper-imental studies with RNA-seq analysis on both control and BCR-ABL transduced cells, we highlight the actin cytoskeleton signaling pathway as significantly altered during CML transduction.

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