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

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Soft materials such as polymer gels, synthetic biomaterials and living biological 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 (plasticity). 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, irreversible 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 ability 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. Interestingly, stiffening induced failure events are more visible on primary than on TF1 model cells, pointing out that their cross-talk with their native microenvironment may also affect their transformation. Finally, combining these experimental 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.

References:

B. Laperrousaz, et al. *Physical Biology* 13 (2016) 03LT01.

C. Martinez-Torres et al. *Applied Physics Letters* 108 (2016) 034102.