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Extracellular matrix stiffness modulates the nuclear lamina organisations and set nuclear conditions for PRC2 repression.

Although the cells of a multicellular organism share the same genetic material, their functions, structures, and behaviours can vary significantly. Many of these differences arise from distinct gene expression programs. Environmental cues are integrated and mediate epigenetic changes that regulate cell activity, differentiation and development. Cells sense their microenvironment not only through soluble signals but also through mechanical cues. The responsiveness to tissue-level elasticity has important physiological and pathological implications. For instance, stiffening of the extracellular matrix promotes the invasive behaviour of cancer cells, supports the transformation of fibroblasts into cancer-associated fibroblasts (CAFs), prime mesenchymal stem cell (MSC) differentiation programs, and regulate epidermal stem cell fate. By combining hydrogel cell culturing, genomics and super-resolution microscopy, we found that extracellular matrix stiffness modulates the nuclear lamina composition and induces variation in chromatin motion. Furthermore, adaptative gene programs are in part controlled through Polycomb Repressive Complex 2 (PRC2) repression which is tempered by mechano-transduction and the nuclear lamina. Our work highlights mechano-dependent nuclear lamina compositions and changes in chromatin motions that set adaptative gene expression program control through PRC2.

Authors: Dr PIGEOT, Alexia (Institut de Génétique Moléculaire de Montpellier (IGMM)); Dr REY-MILLET, Martin; Dr ZINE EL AABIDINE, Amal (Institut de Génétique Moléculaire de Montpellier (IGMM)); Dr TRULLO, Antonio (Institut de Génétique Moléculaire de Montpellier (IGMM)); BYSTRICKY, Kerstin (CBI); Dr ANDRAU, Jean-Christophe (Institut de Génétique Moléculaire de Montpellier (IGMM)); Dr ESNAULT, Cyril (Institut de Génétique Moléculaire de Montpellier (IGMM))

Orateur: Dr ESNAULT, Cyril (Institut de Génétique Moléculaire de Montpellier (IGMM))