ID de Contribution: **29** Type: **Non spécifié**

Nuclear Beta-Actin and Nuclear Myosin I are required for rDNA/RNAP1 repositioning within the nucleolus after rDNA repair.

During DNA Repair, ribosomal DNA and RNA polymerase I (rDNA/RNAP1) are reorganised within the nucleolus and undergo relatively long-distance movements that are normally not observed except when cells are in mitosis.

UV lesions trigger the DNA repair reaction, blocking RNAP1 transcription and displacing the rDNA/RNAP1 complex at the periphery of the nucleolus. Because most repair proteins are present outside the nucleolus, this movement is believed to be important for the repair reaction to take place properly. Only when the repair reaction is fully completed, the rDNA/RNAP1 complex returns within the nucleolus.

The proteins and the molecular mechanism governing this movement remain unknown.

Here we show that Nuclear Myosin I (NMI) and Nuclear Beta Actin (ACTβ) are essential for the proper re-entry of the rDNA/RNAP1 within the nucleolus, after completion of the DNA Repair reaction.

We found that, in NMI and ACTβ depleted cells, the rDNA/RNAP1 complex can be displaced at the periphery of the nucleolus after DNA damage induction but cannot re-enter within the nucleolus after completion of the DNA Repair reaction. In these cells, repair is proficient and rDNA transcription normally restarts after the lesions-induced blockage. Both proteins act concertedly in this process. NMI binds the rDNA that is displaced at the periphery of the nucleolus during repair reactions while ACTβ brings the rDNA back within the nucleolus after DNA repair completion.

Our results reveal a previously unidentified function for NMI and ACTβ within the nucleolus and disclose how these two proteins work in coordination to re-establish the proper rDNA position after DNA repair.

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