Review of: "Vimentin Regulates Collagen Remodeling Through Interaction with Myosin 10"

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Potential competing interests: The author(s) declared that no potential competing interests exist.

Vimentin expression in epithelial cells is a hallmark for EMT transition. Vim has role in cell migration by generating contractile forces inside the cell. But the invasion mechanism of Vim is not defined. Cancer cells express collagenases which help in invasion. Vimentin is associated with one of the collagenase at the cytosolic tail- membrane type 1- matrix metalloproteinase (MT-1-MMP). MT-1-MMP is also found to be co localized with an unconventional myosin (Myo 10) whose expression affected MT-1-MMP levels. The aim was to study the interaction of Vim and Myo 10 and their regulation of collagen I.

Immunohistochemistry was done to show the co-localization of Vim and Myo 10 in the tumor tissue samples and the co-localization was significantly higher in the epithelial region and no difference in stroma of normal and tumor samples. They studied the interaction of Vim and Myo10 in the mouse embryonic fibroblast (wild type WT, Vim KO and Vim ULF- having point mutation in Vimentin that does not allow it to mature). There was significant difference in the protein expression levels of Myo 10 in WT and Vim ULF. Mean fluorescence intensity of the cells with Myo 10 at the tip was higher in WT compared to KO AND ULF. They also showed Vim and Myo 10 associated closely spatially and confirmed their interaction by performing Fret and SPR. Giving IPA3 treatment they proved that mature vimentin structure is required for proper aggregation of Myo 10 at the tips. They showed that the myosin 10 motility is regulated on vimentin expression and maturation by FRAP.

Next, they studied the co-effect of Vim and Myo 10 on the Collagen I (CoI). There was significant difference in the alignment and compaction of collagen I in Vim KO and ULF cells compared to WT. They also showed that myo10 KD also significantly affects the alignment and compaction of collagen I compared to WT. But Myo 1 KD in Vim KO showed no significant difference in collagen modelling. Collagen proteolysis was regulated my Vim expression and not its formation (since no significant difference between WT and ULF) was showed using neo antibodies against collagen I fragments. Finally, they showed significant difference in the effect of Vim on localization and motility of MT-1-MMP in Vim KO and ULF compared to WT. The findings of the paper shows expression of Vim regulating the proteolysis of MT-1-MMP to remodel the CoI to help cell migration in mouse embryonic fibroblast also extended to SW480 cancer cell lines.

Overall, paper is very well written and documented. Only my major concern is too many figure sub-section. Authors can add extra data in supplement section. Results are significant and concluding the finding.

Discussion is also very well documented with all relevant reference.