# Matrix Biology Highlights

*Edited by Maria Gubbiotti and Tom Neill*

## A Truly Radical Perspective for Vimentin-Induced Wound Repair

**Reference |** Lebert, D., Squirrell, J.M., Freisinger, C., Rindy, J., Golenberg, N., Frecentese, G., Gibson, A., Eliceiri, K.W., Huttenlocher, A. 2018. Damage-induced reactive oxygen species regulate vimentin and dynamic collagen-based projections to mediate wound repair. *ELife.* doi: 10.7554/eLife.30703

This manuscript by Lebert and Squirrell *et al*. investigated the role of ROS in the early stages of wound repair. Specifically, using an elegant zebrafish model coupled with live imaging and a GFP-labeled vimentin reporter, they discovered that redox signaling that occurs during wound formation provokes cells at the leading edge of the wound to induce vimentin expression. This amplified vimentin expression causes a consequent increase in collagen while simultaneously stimulating collagen re-organization to promote caudal fin repair. Ultimately, these new data demonstrate the utility and value of zebrafish as a model for wound repair while also providing a fresh perspective for future study of wound healing in mammalian systems.

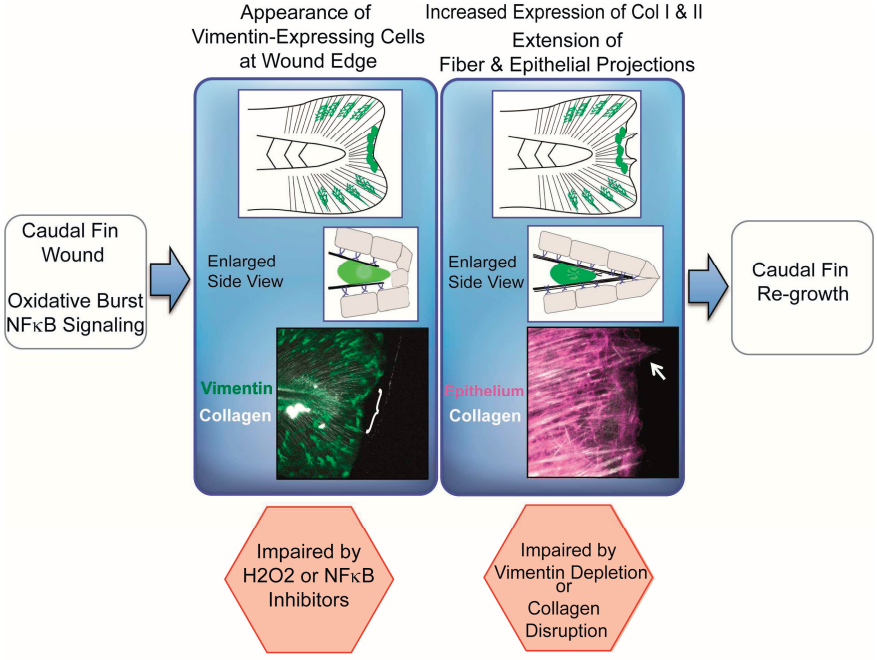


**Figure 1: Vimentin-dependent formation of collagen fiber-containing epithelial projections lead the progression of tissue regrowth**. At 18 to 24 hours after wounding, a de novo population of vimentin-expressing cells (green cells, noted with white bracket) emerges at the wound edge, an event dependent upon early wound signaling molecules. Approximately coincident with this appearance of cells, there is a vimentin-dependent increase in Type I and Type II Collagen RNA expression as well as the formation of collagen (white) and epithelial (magenta) projections at the wound edge (noted with white arrow). Additionally, disruption of collagen fibers, as occurs with thermal injury, inhibits projection formation and subsequent caudal fin regrowth. Figure kindly provided by Squirrell, J.

## Silence of the LAMB3 to Battle Papillary Thyroid Cancer

**Reference |** Jung, S.N., Lim, H.S., Liu, L., Chang, J.W., Lim, Y.C., Rha, K.S.[1], Koo, B.S. 2018. LAMB3 mediates metastatic tumor behavior in papillary thyroid cancer by regulating c-MET/Akt Signals. *Sci. Rep.* doi: 10.1038/s41598-018-21216-0.

LAMB3 is a gene that encodes one of the subunits of the extracellular matrix protein, LM-332. In this manuscript, Jung et al. demonstrate that LAMB3 is upregulated in papillary thyroid cancer (PTC) when compared to normal thyroid tissue. Mechanistically, reduction of LAMB3 diminishes migration and invasion of PTC cells in a manner determined by reduced signaling through the HGF/c-Met/PI3K/Akt pathways. Thus, LAMB3 could be a novel diagnostic marker as well as an enticing therapeutic target for the treatment of papillary thyroid cancer.

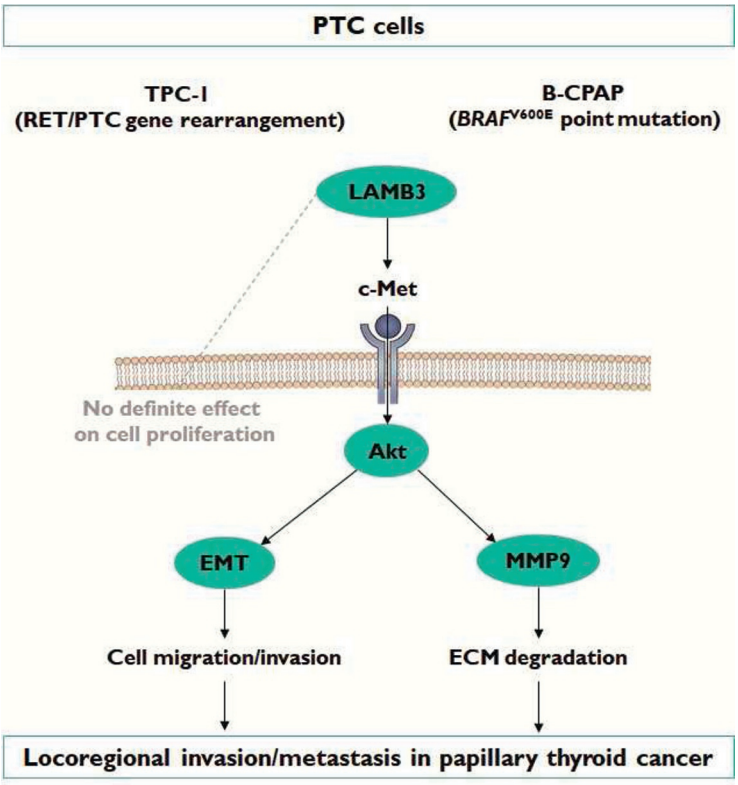


**Figure 2: LAMB3 stimulates signaling** via the c-Met receptor tyrosine kinase to induce epithelial-mesenchymal transition and matrix metalloproteinase activation resulting in increased invasion and migration in papillary thyroid cancer cells. Figure kindly provided by Koo, BS.