Stromal – epithelial cross-talk in tissue repair and cancer

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Abstract: There are remarkable parallels between wound healing and cancer at the histological, cellular and molecular levels. Therefore, identification of genes that control wound repair is of key relevance for cancer research. We identified the growth and differentiation factor activin A as an important regulator of both wound repair and skin carcinogenesis. Thus, upregulation of activin in keratinocytes at the wound site promoted different aspects of healing, in particular through activin’s paracrine effect on stromal cells. When excessive, these activities also promoted skin carcinogenesis through reprogramming of skin macrophages and fibroblasts into highly motile cells with strong pro-tumorigenic activities. As a second example, we found that upregulation and activation of the cytoprotective Nrf2 transcription factor in keratinocytes or fibroblasts accelerates wound repair, but also promotes tumorigenesis through protection of mutated (tumor) cells from reactive oxygen species and through activation of pro-tumorigenic matrix genes in fibroblasts. These results demonstrate that a thorough regulation of key drivers of wound healing is essential to promote normal tissue repair without inducing cancer formation. They also provide insight into the relevance of stromal-epithelial interactions in both wound healing and cancer development.

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