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Autore	YESHAQ Yosef
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Autore	Gabazza Esteban C
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Sommario/riassunto	Tissue fibrosis may occur for unknown causes or be the consequence of many pathological conditions including chronic inflammatory or infectious diseases, autoimmune disorders, graft rejection, or malignancy. On the other hand, malignant tumors have been identified in fibrotic tissues decades ago, and now accumulating evidence

suggests that fibrotic lesions enhance the risk of cancer in several organs such as liver, lungs, and breast. Disruption of an organ parenchymal cells and of its normal structural scaffold during tissue fibrogenesis appears to induce loss of cell polarity, promoting uncontrolled cell proliferation that may eventually lead to cancer development. Many cellular and molecular abnormalities including aberrant expression of microRNAs, genetic and epigenetic alterations, evasion or delayed apoptosis, unregulated intracellular signal pathways, and dysregulation or defective intercellular communications have been proposed to explain this link between fibrogenesis and carcinogenesis. However, the precise mechanisms of this fibrosis-to-cancer transition remain unclear. This book presents a collection of reviews and original articles summarizing recent advances in understanding the molecular mechanisms of cancer development in fibrotic organs.