ARF6 pathway enhances PD-L1 expression and fibrosis

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Video Byte

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Abstract

Immune checkpoints are an effective way that cancers evade the immune system, but they're not the only one. In the case of pancreatic ductal carcinoma, or PDAC, tumor fibrosis also plays an important role. To understand how fibrosis might translate to poor outcomes among patients with PDAC, researchers examined the ARF6-AMAP1 molecular pathway, which research suggests is activated during fibrosis. Findings revealed that AMAP1 correlated with elevated expression of PD-L1, a molecule that tumor cells present on their surface to elude attack by the immune system. AMAP1 was also linked to elevated fibrosis. Consistently, silencing AMAP1 in a mouse model of human PDAC reduced PD-L1 and fibrosis in their tumors. Suppressing the ARF6-AMAP1, therefore, could be one way to ensure that PDAC tumors can't hide from immune defenses, offering the prospect of more effective immunotherapies for patients with pancreatic ductal carcinoma.