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Strohecker and colleagues found that deletion of the essential autophagy gene Atg7 initially induced oxidative stress and accelerated the formation of Braf\(^{V600E}\)-driven lung tumors but eventually slowed tumor growth and prolonged survival. Atg7 deficiency led to an accumulation of morphologically and functionally defective mitochondria in Braf\(^{V600E}\)-driven lung tumors and rendered tumor cells dependent on exogenously supplied glutamine for survival. Braf\(^{V600E}\)-driven tumors may therefore become addicted to autophagy to sustain cell survival and proper mitochondrial function through the clearance of damaged organelles and recycling of metabolites for biosynthesis, and may thus be sensitive to autophagy inhibitors. For details, please see the article by Strohecker and colleagues on page 1272.

**Autophagy Sustains Mitochondrial Glutamine Metabolism and Growth of Braf\(^{V600E}\)-Driven Lung Tumors**


**Précis:** Autophagy ablation suppresses the growth of Braf\(^{V600E}\)-driven lung tumors by limiting glutamine availability and impairing mitochondrial function.

*See commentary, p. 1225*

**Targeting the Wnt Pathway in Synovial Sarcoma Models**


**Précis:** Constitutive activation of WNT/β-catenin signaling by the SYT-SSX oncogene is required for the initiation and progression of synovial sarcoma.

*See commentary, p. 1220*