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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 BrafV600E-driven lung tumors but eventually slowed tumor growth and prolonged survival. Atg7 deficiency led to an accumulation of morphologically and functionally defective mitochondria in BrafV600E-driven lung tumors and rendered tumor cells dependent on exogenously supplied glutamine for survival. BrafV600E-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.

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