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Defect in branched-chain amino acid (BCAA) catabolism has been recognized as a metabolic hallmark and therapeutic target for heart failure. As BCAA catabolic enzymes express ubiquitously, it is important to determine the cell-autonomous impact of BCAA catabolic defect in cardiomyocytes in vivo. In this issue, Chen Gao and the colleagues explored the cardiomyocytes specific BCAA catabolic defects in vivo. See the article in pages 1380–1390.

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