Objective: Brown adipose tissue (BAT) thermogenesis offers the potential to improve metabolic health in mice and humans. However, humans predominantly live under thermoneutral conditions, leading to BAT whitening, a reduction in BAT mitochondrial content and metabolic activity. Recent studies have established mitophagy as a major driver of mitochondrial degradation in the whitening of thermogenic brite/beige adipocytes, yet the pathways mediating mitochondrial breakdown in whitening of classical BAT remain largely elusive. The transcription factor EB (TFEB), a master regulator of lysosomal biogenesis and autophagy belonging to the MiT family of transcription factors, is the only member of this family that is upregulated during whitening, pointing toward a role of TFEB in whitening-associated mitochondrial breakdown. Methods: We generated brown adipocyte-specific TFEB knockout mice, and induced BAT whitening by thermoneutral housing. We characterized gene and protein expression patterns, BAT metabolic activity, systemic metabolism, and mitochondrial localization using in vivo and in vitro approaches. Results: Under low thermogenic activation conditions, deletion of TFEB preserves mitochondrial mass independently of mitochondriogenesis in BAT and primary brown adipocytes. However, this does not translate into elevated thermogenic capacity or protection from diet-induced obesity. Autophagosomal/lysosomal marker levels are altered in TFEB-deficient BAT and primary adipocytes, and lysosomal markers co-localize and copurify with mitochondria in TFEB-deficient BAT, indicating trapping of mitochondria in late stages of mitophagy. Conclusion: We identify TFEB as a driver of BAT whitening, mediating mitochondrial degradation via the autophagosomal and lysosomal machinery. This study provides proof of concept that interfering with the mitochondrial degradation machinery can increase mitochondrial mass in classical BAT under human-relevant conditions. However, it must be considered that interfering with autophagy may result in accumulation of non-functional mitochondria. Future studies targeting earlier steps of mitophagy or target recognition are therefore warranted.