Objective: The thermogenic activity of brown adipose tissue (BAT) holds the potential to enhance metabolic health in both mice and humans. However, the predominant exposure of humans to thermoneutral conditions often leads to BAT whitening, characterized by a reduction in mitochondrial content and metabolic activity. While mitophagy has been identified as a major contributor to mitochondrial degradation in the whitening process of thermogenic brite/beige adipocytes, the pathways involved in mitochondrial breakdown during the whitening of classical BAT remain largely unknown. The transcription factor EB (TFEB), a master regulator of lysosomal biogenesis and autophagy belonging to the MiT family of transcription factors, stands out as the only member upregulated during whitening, suggesting a potential role for TFEB in whitening-associated mitochondrial breakdown. Methods: Brown adipocyte-specific TFEB knockout mice were generated, and BAT whitening was induced by thermoneutral housing. The study encompassed the characterization of gene and protein expression patterns, BAT metabolic activity, systemic metabolism, and mitochondrial localization using in vivo and in vitro approaches. Results: Under conditions of low thermogenic activation, the deletion of TFEB was found to preserve mitochondrial mass independently of mitochondriogenesis in BAT and primary brown adipocytes. However, this preservation did not translate into enhanced thermogenic capacity or protection from diet-induced obesity. TFEB-deficient BAT and primary adipocytes exhibited altered levels of autophagosomal/lysosomal markers. Lysosomal markers were observed to co-localize and copurify with mitochondria in TFEB-deficient BAT, indicating the trapping of mitochondria in late stages of mitophagy. Conclusion: The study identifies TFEB as a driver of BAT whitening, mediating mitochondrial degradation through the autophagosomal and lysosomal machinery. This research provides proof of concept that interfering with the mitochondrial degradation machinery can increase mitochondrial mass in classical BAT under human-relevant conditions. However, potential implications must be considered, as interfering with autophagy may lead to the accumulation of non-functional mitochondria. Future studies targeting earlier steps of mitophagy or target recognition are therefore warranted.