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TFAM is required for maturation of the fetal and adult intestinal epithelium

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Abstract

During development, the embryo transitions from a metabolism favoring glycolysis to a metabolism favoring mitochondrial respiration. How metabolic shifts regulate developmental processes, or how developmental processes regulate metabolic shifts, remains unclear. To test the requirement of mitochondrial function in developing endoderm-derived tissues, we genetically inactivated the mitochondrial transcription factor, *Tfam*, using the *Shh-Cre* driver. *Tfam* mutants did not survive postnatally, exhibiting defects in lung development. In the developing intestine, TFAM-loss was tolerated until late fetal development, during which the process of villus elongation was compromised. While progenitor cell populations appeared unperturbed, markers of enterocyte maturation were diminished and villi were blunted. Loss of TFAM was also tested in the adult intestinal epithelium, where enterocyte maturation was

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