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

Manasa Srivillibhuthur[a,c12], Bailey N. Warder[a2], Natalie H. Toke[a], Pooja P. Shah[a], Qiang Feng[d], Nan Gao[d], Edward M. Bonder[d], Michael P. Verzi[a,b,*]

[a]Rutgers University, Department of Genetics, Human Genetics Institute of New Jersey (HGINJ), 145 Bevier Road, Piscataway Township, NJ 08854, USA.

[b]Rutgers Cancer Institute of New Jersey (CINJ), 195 Little Albany Street, New Brunswick, NJ 08903, USA.

[c]Lewis Katz School of Medicine at Temple University, 3500 N Broad Street, Philadelphia, PA 19140, USA.

[d]Department of Biological Sciences, Rutgers, The State University of New Jersey, Newark, NJ 07102, USA.

[*]Corresponding Author/Lead Contact: M. Verzi, Verzi@biology.rutgers.edu

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

[1]current affiliation
[2] These authors contributed equally