



Ciências
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Homeostatic control of energy metabolism by monocyte-derived macrophages

Multicellular organisms rely on inter-organ communication networks to maintain vital parameters within a dynamic physiological range. Macrophages are central to this homeostatic control system, sensing and responding to deviations of those parameters to sustain organismal homeostasis. Here, we demonstrate that dysregulation of iron (Fe) metabolism, imposed by the deletion of ferritin H chain (FTH) in mouse parenchymal cells, is sensed by monocyte-derived macrophages. In response, monocyte-derived macrophages support tissue function, energy metabolism, and thermoregulation via a mechanism that sustains the mitochondria of parenchymal cells. Mechanistically, FTH supports a transcriptional program promoting mitochondrial biogenesis in macrophages, involving mitochondrial transcription factor A (TFAM). Moreover, FTH sustains macrophage viability and supports intercellular mitochondrial transfer from donor parenchymal cells. In conclusion, monocyte-derived macrophages cross-regulate iron and energy metabolism to support tissue function and organismal homeostasis.

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