AK2 deficiency compromises the mitochondrial energy metabolism required for differentiation of human neutrophil and lymphoid lineages

Titre	AK2 deficiency compromises the mitochondrial energy metabolism required for differentiation of human neutrophil and lymphoid lineages
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Résumé en anglais	Reticular dysgenesis is a human severe combined immunodeficiency that is primarily characterized by profound neutropenia and lymphopenia. The condition is caused by mutations in the adenylate kinase 2 (AK2) gene, resulting in the loss of mitochondrial AK2 protein expression. AK2 regulates the homeostasis of mitochondrial adenine nucleotides (ADP, ATP and AMP) by catalyzing the transfer of high-energy phosphate. Our present results demonstrate that AK2-knocked-down progenitor cells have poor proliferative and survival capacities and are blocked in their differentiation toward lymphoid and granulocyte lineages. We also observed that AK2 deficiency impaired mitochondrial function in general and oxidative phosphorylation in particular - showing that AK2 is critical in the control of energy metabolism. Loss of AK2 disrupts this regulation and leads to a profound block in lymphoid and myeloid cell differentiation.
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