Non-cytotoxic copper overload boosts mitochondrial energy metabolism to modulate cell proliferation and differentiation in the human erythroleukemic cell line K562

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Copper is integral to the mitochondrial respiratory complex IV and contributes to proliferation and differentiation, metabolic reprogramming and mitochondrial function. The K562 cell line was exposed to a non-cytotoxic copper overload to evaluate mitochondrial dynamics, function and cell fate. This induced higher rates of mitochondrial turnover given by an increase in mitochondrial fusion and fission events and in the autophagic flux. The appearance of smaller and condensed mitochondria was also observed. Bioenergetics activity included more respiratory complexes, higher oxygen consumption rate, superoxide production and ATP synthesis, with no decrease in membrane potential. Increased cell proliferation and inhibited differentiation also occurred. Non-cytotoxic copper levels can modify mitochondrial metabolism and cell fate, which could be used in cancer biology and regenerative medicine.

Autophagy

Bioenergetics

Copper

Erythropoiesis

Mitochondria

Mitochondrial dynamics

adenosine triphosphate

copper

reactive oxygen metabolite

copper

aerobic metabolism

Article

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cell differentiation

cell fate

cell function

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metabolism

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Cell Differentiation

Cell Proliferation

Copper

Energy Metabolism

Humans

K562 Cells

Mitochondria