Despite a high rate of complete remission after treatment with genotoxic agents, the prognosis is poor in human acute myeloid leukemia (AML), due to frequent relapses caused by resistant leukemic clones (RLCs). Our goal is to understand the causes of the drug resistance for the development of new treatments eradicating RLCs and overcoming patient relapses. Here we will discuss about the role of mitochondrial oxidative metabolism in drug resistance of AML and about newly-identified metabolic features of patients harboring mutations in key metabolic enzymes isocitrate dehydrogenases.

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