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Front cover image: Schematic illustration of the main mitochondrial changes frequently occurring in cancer cells. The reprogramming of mitochondrial metabolism in many cancer cells comprises reduced pyruvate oxidation by PDH followed by the TCA cycle, increased anaplerotic feeding of the same cycle, mostly from Gln, whose entry in the mitochondrial matrix is facilitated by UCP2 up-regulation. This increases also the free fatty acids uptake by mitochondria, therefore β-oxidation is pushed to produce acetyl-coenzyme A, whose oxidation contributes to ATP production. In cancer cells many signals can converge on the mitochondrion to regulate the mitochondrial membrane permeability, which may respond by elevating the MPTP (PTP) threshold, with consequent enhancement of apoptosis resistance. ROS belong to this class of molecules since it can enhance Bcl2 and may induce DNA mutations. Dotted lines indicate regulation; solid lines indicate reaction(s).

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