Availability, not respiratory capacity governs oxygen consumption of solid tumors.

Abstract:
Contrary to conventional belief, the mitochondria of most cancer cells usually function normally, i.e., their respiratory capacity is not fundamentally impaired as compared to normal cells. Strong evidence against the misconception of mitochondrial dysfunction is provided by in vivo data clearly showing that O(2) availability is the major determinant of the O(2) consumption rate of cancer cells, independent of the means for increasing availability (e.g., by increasing blood flow or by elevating arterial O(2) content, the latter being accomplished either by an increase in the hemoglobin level and/or arterial hyperoxia). Additional support against the Warburg effect in its original concept comes from normal temperature coefficients (Q(10)) for O(2) consumption rates of malignant cells. Thus, the Warburg hypothesis postulating that mitochondrial dysfunction in cancer cells forces them to generate energy with a poor ATP yield through glycolysis appears to be elusive. Instead, due to a "reprogrammed" cancer cell metabolism, glycolysis is used to produce intermediates as building blocks for various biosynthetic pathways of cancer cells.