Modulation of mitochondrial large conductance potassium channel activity by infrared light

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Initially, mitochondria were treated only as a source of ATP synthesis in the cell. Subsequently, the role of mitochondria was shown to be more complex and connected also with Ca^{2+} buffering and reactive oxygen species (ROS) synthesis. Increased ROS synthesis and Ca^{2+} accumulation in mitochondria start an intracellular signaling pathway for necrosis and apoptosis in cells. It seems that mitochondrial potassium (mitoK) channels, present in inner mitochondrial membrane, are involved in all these functions. Mitochondrial potassium channels activity can be regulated by natural and synthetic compounds. Unfortunately, many channel modulators have the off-targets in the cell as was described. Previously reported regulation of mitoK channels activity by redox status of the respiratory chain may suggest a new target for functional intervention. It is well known that some proteins present in the respiratory chain are infrared light absorbers. Cytochrome c oxidase (COX) could be very important in this mechanism since it has four redox metal centers: the binuclear CuA, CuB, heme a and heme a3. All these metal centers are able to absorb the specific light wavelengths in red/infrared (IR/NIR) region. Hence, it is believed that COX and mitochondrial potassium channels are the main targets of the IR/NIR light irradiation leading to cell protection. Data obtained in our laboratory show that COX could be functionally interlinked with mitochondrial large conductance Ca^{2+}activated potassium (mitoBKCa) channel in the U87 cell line. Using patch-clamp technique for measurements of single channel activity, with an illumination system we have irradiated mitoBK$_{Ca}$ channel with two wavelengths: 760 nm and 820 nm. We observed that the in oxidised conditions (in presence of ferricyanide) the channel activity was inhibited and that mitoBK channel activity could be restored by illumination with 820 nm wavelength, suggesting that COX is involved in modulation of mitoBK$_{Ca}$ channel activity.

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