Research Day 2014

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11:00 AM - 2:30 PM
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Non-thermal Plasma Modulates Metastatic Prostate Cancer Homeostasis by Targeting Mitochondria Metabolism

BB-021

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Metastatic cancers currently remain incurable diseases. There is an urgent need for novel medical approaches for treatment of metastatic tumors, which develop resistance to chemo- and radiation therapy. Non-thermal plasma discharges have been studied for a variety of biomedical applications, including their ability to induce apoptosis as a method of cancer eradication. The cellular intrinsic apoptotic pathway is mediated by mitochondria, therefore the mitochondria energetics is a plausible target for cancer therapy. This work aimed to explore the intracellular effects of non-thermal plasma in metastatic prostate cancer cells.

The prostate cancer cells are characterized by very actively functioning mitochondria promoting these cells' ability to avoid apoptosis. We showed that non-thermal plasma targets DU145 cells' mitochondria directly causing alterations in the membrane potential, coupling the respiration and oxidative phosphorylation. Mitochondria vulnerability to reactive oxygen species generated by non-thermal plasma could be the mechanism of mitochondria injury. The moderate reduction of mitochondria membrane potential induced by plasma results in increased generation of superoxide radicals via modulation of both respiratory complexes I and III and by that enriching the pool of cell oxidative species. Additionally, although the cytosolic calcium level of prostate cancer cells does not change, the cell calcium signaling systems seem to be sensitized by plasma exposure. Stimulation of plasma treated cells with exogenous ATP resulted in sustained cytosolic calcium elevation compared to the oscillatory calcium waves observed in control cells. Prolonged cytosolic calcium elevation results in mitochondria and other intracellular injuries.Our data indicate that mitochondria-mediated alterations are one of the mechanisms by which non-thermal plasma induces apoptosis in aggressive prostate cancer cells.