

Effect of Weak Static Magnetic Fields on cell proliferation and reactive oxygen species of HT-1080 human fibrosarcoma cells

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Reactive Oxygen Species (ROS) are known to function as signaling molecules to regulate wide variety of cellular activities. ROS are derived from oxygen (O_2) through variety of mechanisms and primary source is the Cellular Respiration. Cellular Respiration is the process leads to generation of ATP. ATP, which is the main energy source used in cells, is formed from the oxidation of $NADH$ and $FADH_2$. The reduced electron carriers ($NADH$ and $FADH_2$) dump electrons onto the electron transport chain to power chemiosmosis ATP synthesis. Most intracellular ROS are derived from Superoxide (O_2^-) which is generated by the one electron reduction of (O_2). Superoxide then converted to Hydrogen Peroxide (H_2O_2) by Superoxide Dismutase (SOD). H_2O_2 plays an important role in many physiological processes such as hypoxic signal transduction, cell differentiation and proliferation.

The aim of this work is to study the effect of weak static magnetic fields (SMF) on these differentiation activities. The influx of Calcium (Ca^{2+}) through the plasma membrane can be associated with the activation of voltage-dependent calcium channels by the SMF. Many of the reported effects of moderate SMF can be explained on the basis of alterations in membrane calcium influx since Sodium (Na) channels are affected to a lesser degree than (Ca^{2+}) channels. To establish whether exposure to the field could influence the molecular biology of the cells, HT-1080 human fibrosarcoma cells were exposed to a magnetic flux density between $0.5 \mu T$ to $600 \mu T$ for 4 days in different orientations. Experiments are performed in a Mu-metal cage inside an incubator to reduce the background magnetic field. Temperature and CO_2 variations are continuously controlled. Exposure to SMF increased concentrations of mitochondrial (Ca^{2+}), membrane potential and cell growth rates. We observed nonlinear changes in ROS and oxidative stress. This nonlinear response to mitochondrial ROS is known as mitochondrial mitohormesis which considers ROS as essential signaling molecules. Our results showed that we can use weak SMF as a tool to signal biological systems and this may provide a new strategy for cancer therapy.