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Overcoming drug resistance in cancer through modulation of ROS

Amit K. Maiti
Oklahoma Medical Research Foundation, USA

Drug resistance in cancer is an overwhelming problem since drug-resistant cancer cells are harder to kill with the same drug. Cancer cells have higher ROS level than normal cells. Most cancer drugs are shown to increase Reactive Oxygen Species (ROS) in respective cancer cells that induces apoptosis, but continuous treatment with the same drug reduce cellular ROS levels and convert drug sensitive cancer cells into drug resistant cells. Subsequently, drug resistant cells have lower ROS content than drug sensitive cells. Concurrent with this observation, exogenous elevation of ROS in conjunction with drug resensitizes drug resistant cancer cells. Thus, constant maintenance of higher ROS level in cancer cells is a prerequisite for drug efficacy. Modulation of ROS-mediated genetic pathway genes is identified through microarray gene expression followed by gene-network analysis using Ingenuity Pathway Analysis (IPA). This reveals that anticancer drugs induce master regulatory genes to induce apoptosis in cancer cells but prolonged treatment with the same drug could act on NFE2L2-KEAP1 antioxidant system to reduce the ROS level in cancer cells, thus gaining resistance. Manipulating these master regulatory genes could be an efficient alternative to maintain higher ROS level in cancer for 'combinational chemotherapy' with the drug. Nevertheless, ROS level monitoring could be an efficient strategy to measure the drug efficacy in cancer patients.

Biography

AKM has completed his PhD in Jadavpur University, Kolkata, India and did his postdoctoral studies in Geneva University Medical School, Geneva, Switzerland. He is an Assistant professor in OMRF, published numerous papers and serves editorial board member in reputed journals.

Amit-Maiti@omrf.org