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Heightened state of oxidative stress in MCF-7 cells enhances growth potential and promotes EMT by increasing Akt and ERK activity

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An imbalance in cell proliferation, survival and apoptosis possibly stemming from genetic and/or environmental factors is often detected in cancer cells. Indeed, chronic exposure to estrogens and xenoestrogens (e.g., diethylstilbestrol and organochlorine pesticides) results in the generation of genotoxic metabolites and reactive oxygen species (ROS) and has been linked with breast cancer. Here, we report that chronic exposure of MCF-7 breast cancer cells to a heightened state of oxidative stress (HSOS) promotes cell proliferation via activation of G1/S and G2/M checkpoint transition. This was associated with a significant elevation in the positive cell cycle regulators, including cyclin D1 and cyclin A. Contrastingly, HSOS inhibited apoptosis, concomitantly with a decrease in BAX and caspase-3-mediated cleavage of PARP levels in MCF-7. An epithelial-to-mesenchymal transition (EMT) which plays a key role in cancer cells migration and invasion was enhanced in HSOS-treated MCF-7; as evidenced by the repression of E-cadherin and up-regulation of vimentin. Activation of PI3k/Akt and MAPK/ERK1/2 pathways appear to contribute to HSOS induced MCF-7 cell proliferation, migration and EMT. Collectively, the above data shed light on the importance of chronic oxidative stress in promoting cell growth and tumorogenesis and suggest that HSOS could be a useful target for breast cancer therapy.

Biography

Milad Bitar is working as a Professor in the Department of Pharmacology & Toxicology, Kuwait University. He has completed his Bachelor of Science from Oklahoma University (1977), Master of Science from University Maryland, Baltimore (1979). He has completed his PhD in 1983 from University Maryland and served as an Assistant Professor in Pennsylvania College Pediatric Medicine (1987-1990). He has published more than 35 articles in many international journals.

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