Moureq Alotaibi

PhD In Pharmacology and toxicology from Virginia Commonwealth University

alotaibimr@vcu.edu

Experience

Assistant Professor at King Saud University

Assistant Professor at King Saud University
January 2016 - Present (9 months)

Teaching Assistant at King Saud University
January 2009 - Present (7 years 9 months)

Currently, I am sponsored by king Saud university to pursue my PhD at Virginia commonwealth university.

Pharmacist at King Abdulaziz Medical City
January 2008 - January 2009 (1 year 1 month)

Outpatient Pharmacist.

Publications

Stilbene 5c, a microtubule poison with vascular disrupting properties that induces multiple modes of growth arrest and cell death.

Biochemical Pharmacology October 18, 2013

Authors: Moureq Alotaibi, Asnake B,, Di X, Beckman MJ,, Durrant D,, Simoni D,, Baruchello R,, Lee RM,, Schwartz EL,, Gewirtz DA.

The stilbene derivative, cis-3,4',5-trimethoxy-3'-aminostilbene (stilbene 5c), is a potentially potent antitumor agent that acts via binding to the colchicine-binding site in tubulin. The current studies were designed to investigate the effectiveness of stilbene 5c against the HCT-116 human colon cancer cell line and B16/F10 melanoma cells as well as human endothelial cell tube formation and tumor perfusion. This work indicates that stilbene 5c could potentially be effective against melanoma and colon cancer through the promotion of multiple modes of growth arrest and cell death coupled with anti-angiogenic and antivascular actions.

Autophagy, cell death and sustained senescence arrest in B16/F10 melanoma cells and HCT-116 colon carcinoma cells in response to the novel microtubule poison, JG-03-14.

Cancer chemotherapy and pharmacology November 21, 2012

Authors: Moureq Alotaibi

Combined antiproliferative effects of the aminoalkylindole WIN55,212-2 and radiation in breast cancer cells.

Journal of Pharmacology and experimental therapeutics November 20, 2013

Authors: Moureq Alotaibi

Cytotoxic autophagy in cancer therapy.

International Journal of Molecular Sciences June 5, 2014

Authors: Moureq Alotaibi

Autophagy is a process of cellular self-digestion, whereby the cell degrades subcellular materials in order to generate energy and metabolic precursors in order to prolong survival, classically under conditions of nutrient deprivation. Autophagy can also involve the degradation of damaged or aged organelles, and misfolded or damaged proteins to eliminate these components that might otherwise be deleterious to cellular survival. Consequently, autophagy has generally been considered a prosurvival response. Many, if not most chemotherapeutic drugs and radiation also promote autophagy, which is generally considered a cytoprotective response, in that its inhibition frequently promotes apoptotic cells death. Furthermore, it has been shown that conventional chemotherapeutic drugs and radiation alone rarely induce a form of autophagy that leads to cell death. However, there are multiple examples in the literature where newer chemotherapeutic agents, drug combinations or drugs in combination with radiation promote autophagic cell death. This review will describe autophagic cell death induced in breast tumor cells, lung cancer cells as well as glioblastoma, demonstrating that it cannot be concluded that stress induced autophagy is, of necessity, cytoprotective in function.

Radiosensitization by PARP Inhibition in DNA Repair Proficient and Deficient Tumor Cells: Proliferative Recovery in Senescent Cells.

Radiation research March 2, 2016

Authors: Moureq Alotaibi

Radiotherapy continues to be a primary modality in the treatment of cancer. In addition to promoting apoptosis, radiation-induced DNA damage can promote autophagy and senescence, both of which can theoretically function to prolong tumor survival. In this work, we tested the hypothesis that autophagy and/or senescence could be permissive for DNA repair, thereby facilitating tumor cell recovery from radiation-induced growth arrest and/or cell death. In addition, studies were designed to elucidate the involvement of autophagy and senescence in radiosensitization by PARP inhibitors and the re-emergence of a proliferating tumor cell population. In the context of this work, the relationship between radiation-induced autophagy and senescence was also determined. Studies were performed using DNA repair-proficient HCT116 colon carcinoma cells and a repair-deficient ligase IV(-/-) isogenic cell line. Exposure to radiation resulted in a transient arrest in the HCT116 cells while arrest was prolonged in the ligase IV(-/-) cells, however, both cell lines ultimately recovered proliferative function, which may reflect maintenance of DNA repair capacity. The PARP inhibitors, olaparib and niraparib, increased the extent of persistent DNA damage induced by radiation exposure as well as the extent of both autophagy and senescence. Neither cell line underwent significant apoptosis by radiation exposure alone or in the presence of the PARP inhibitors. Inhibition of autophagy failed to attenuate radiosensitization, indicating that autophagy was not involved in the action of the PARP inhibitors. As with radiation alone, despite sensitization by PARP inhibition, proliferative recovery was evident within a period of 10-20 days. While inhibition of DNA repair via PARP inhibition may initially sensitize tumor cells to radiation via the promotion of senescence, this strategy does not appear to interfere with proliferative recovery, which could ultimately contribute to disease recurrence.

Tumor Cell Recovery from Senescence Induced by Radiation with PARP Inhibition
Radiation Research 2016
Authors: Moureq Alotaibi

Inhibitors of poly(ADP-ribose) polymerase (PARP) are clinically used as single-agent therapy for tumors with BRCA1 or BRCA2 mutations. One approach to expanding the use of PARP inhibitors to a wider range of tumors is to combine them with cytotoxic chemotherapy or radiotherapy. Preclinical studies in experimental animals and tumor cells in culture indicate that PARP inhibition modestly sensitizes most tumor cells to ionizing radiation. Studies of cell behavior after these combined treatments show that radiosensitization is manifested predominantly in an increase in prolonged growth arrest and senescence, with little or no contribution from apoptosis. The secretory phenotype associated with senescence can target these tumor cells for immune surveil-lance, and therefore increased senescence can effectively contribute to tumor control. However, the possible recovery of senescent cells and re-entry into cell cycle after prolonged arrest also needs to be considered. Such recovery could lead to tumor recurrence, yet may not be reflected in short-term assays commonly used to assess radiosensitization.

Languages

Arabic (Native or bilingual proficiency)
English (Professional working proficiency)

Skills & Expertise

Cell Culture
Lecturing
Molecular Biology
Pharmacology
Science
University Teaching
Western Blotting
PCR
Biochemistry
Clinical Research
Genetics
Microscopy
Cell Biology
Animal Models
Data Analysis
Research
Healthcare

Education

Virginia Commonwealth University
Doctor of Philosophy (PhD), Pharmacology and Toxicology, 2012 - 2015
Virginia Commonwealth University

Master's degree, Pharmacology and Toxicology, 2010 - 2012
Activities and Societies: Vice president of Saudi Students Organization at VCU.

King saud university

Bachelor's degree, Pharmacy, 2002 - 2008

Organizations

American Association for Cancer Research

March 2012 to Present

Courses

Independent Coursework

2015 Clinical and Translational Research Course for
Ph.D. Students at the National Institute of Health (NIH)
Clinical Center

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Contact Moureq on LinkedIn