Tentative Outline

Special Issue for Drug Metabolism Letters

Guest Editor: Dr. William L. Stone

Pro-oxidant Drugs and Cancer Therapy

Aims and Scope:

Cancer cells often exhibit an increased level of intrinsic oxidative stress due to increased production of reactive oxygen species (ROS) and a decreased level of some key antioxidant mechanisms. Moreover, the increased level of intracellular ROS in cancer cells has been found to be an underlying cause for the expression of many key cancer phenotypes. This, however, is a dual edged-sword since chemotherapeutic drugs or agents that cause oxidative stress can selectively kill cancer cells that already have a compromised ability to withstand additional oxidative stress compared to normal cells. Many of the successful chemotherapeutic therapies used in clinical practice inadvertently act by a generating additional oxidative stress in cancer cell. Nevertheless, the systematic and deliberate design of pro-oxidant drugs (or polydrugs) that selectively induce apoptosis in cancer cells is an effort that has only recently begun in earnest. A key part of this ongoing effort is the search for oxidative stress biomarkers that could help predict which cancer patients would benefit by pro-oxidant drug therapy.

Subtopics:

- The evidence for increased oxidative stress in cancer cells.
- The role of reactive oxygen species in signal transduction and cancer phenotype expression
- Oxidative stress and the Warburg effect
- Drugs, polydrugs, prodrugs and nutraceuticals that selectively kill cancer cells by inducing oxidative stress
- Biomarkers for oxidative stress and the design of targeted pro-oxidant drugs

Tentative submission timeline:

- Solicit contributions from potential authors along with proposed abstracts -Dec ’14 to Feb ’15.
- Select final contributors- Feb ‘15
- Deadline for final articles – May’15