Tentative Outline

Special Thematic Issue for Current Drug Targets (CDT)
Targeting genomic evolution and cancer progression
Guest Editor: Dr. Masood Shammas

Aims & Scope:

Genomic instability and telomere maintenance (by telomerase and/or homologous recombination) are critical lifelines of cancer cells. Genomic instability which seems to arise early, at premalignant stage (such as Barrett’s esophagus), and gradually intensifies — leading to a series of genomic changes, some of which underlie progression through successive stages of disease, development of drug resistance, and poor clinical outcome. The ability to constantly evolve, not only enables the cancer cell to acquire new characteristics for development and progression of disease, but also presents a great challenge for cancer treatment and diagnosis. Moreover, the changes acquired as a consequence of genomic instability may also predict patient outcome. Genomic instability can be a consequence of a number of factors which can be extrinsic (such as exposure to harmful agents in food and environment and/or chemotherapeutic agents used in cancer treatment) or intrinsic (such as food metabolites and/or aberrations in pathway/s involved in genome maintenance). The mechanisms underlying genomic instability and their activation during carcinogenesis are not fully understood and identification of these mechanisms (both extrinsic and intrinsic) could help in development of novel strategies for cancer prevention and treatment. Recently, the role of inflammation in cancer has also emerged as of great significance in translational cancer research. It has been demonstrated that inflammatory reactions can lead to aberrant expression of genes involved in DNA repair or maintenance, leading to genomic instability. Gastrointestinal tissues derived from diseases which have inflammation related to oncogenic process, including Barrett's esophagus, chronic viral hepatitis, and inflammatory bowel disease, frequently display aberrant expression/function of activation-induced cytidine deaminase, a protein involved in DNA repair/maintenance.

We invite investigators to contribute review and/or original research papers describing recent findings in the fields of genomics/genomic instability, inflammation, and/or the environmental/dietary factors affecting cancers. The purpose of the research published under this topic will be to:

- Understand molecular mechanisms and consequences of genomic instability and inflammation in cancer. Manuscripts may provide novel information in these fields separately or linking them together.
- Identification of new prognostic tools and novel therapeutic strategies, targeting genomic instability and clonal evolution, telomere maintenance, and inflammation.
- Identification of new potential carcinogens, evaluate impact of chemotherapeutic agents on genomic instability and evolution, and present/test new ideas to reduce exposure, minimize harmful genomic impact of genotoxins (including chemotherapeutic agents) and other ideas to prevent cancer or its progression.

Schedule:

- Manuscript submission deadline: May 2020
Contacts:

Guest Editor: Dr. Masood Shammas,
Affiliation: Lead Scientist, Department of Medical Oncology, Harvard (Dana Farber) Cancer Institute, Boston, MA 02115, Harvard Medical School at VAMC West Roxbury, MA 02132, USA.
Email: Masood_shammas@dfci.harvard.edu

Any queries should be addressed to cdt@benthamscience.net