Tentative Outline
Special Issue for CURRENT CANCER DRUG TARGETS
Guest Editor: Prof. Dr. Masatoshi Kitagawa

Ubiquitin E3 ligases as molecular targets or tools for advanced cancer therapy

Aims & Scope: E3 ubiquitin ligases (E3s) are critical players in the quantitative control of important cellular proteins including cancer-associated proteins via the ubiquitin–proteasome pathway. In cancer cells, the depletion of E3s targeting oncogene products or proteins promoting cancer development, including the E3 Skp1–Cullin–F-box (SCF)–Fbw7, results in irregular stabilization and accumulation of oncogenic targets. Moreover, the overexpression of E3 tumor suppressor gene products or negative regulators of cancer development, including SCF–Skp2 and MDM2, causes the depletion of anti-oncogenic substrates, which can lead to carcinogenesis. Some E3s such as TRAF2 and BRCA1 ubiquitylate their substrates to promote their instability as well as their activation or functional change. This ubiquitin chain linkage-dependent nonproteolytic function of E3s is also associated with human cancers. This thematic issue first introduces functions and substrates for the SCF-, RING-, and HECT-type E3s involved in human cancer. E3s that regulate cancer-associated cellular processes including epigenetic regulation, the DNA damage response, the cell cycle, apoptosis, signal transduction, stem cell function, and epithelial mesenchymal transition are also discussed about their functions and substrates with the aim of identifying novel molecular targets for cancer diagnosis and therapy. Moreover, protein knockdown technology using E3 activity is introduced as an application of E3 for advanced cancer therapy.

Key words: E3 ubiquitin ligases, proteasome, F-box protein, RING-finger, HECT, TRB family, cell cycle, cancer stem cell, EMT, DNA damage response, chromatin, protein knockdown

Subtopics:

1. F-box proteins contributing in cancer development.
2. HECT- and RING-type E3 ligases involved in human cancers.
3. Ubiquitin-mediated control of EMT
4. TRB family as a regulator or substrate of ubiquitin system in cancer development
5. Ubiquitin-mediated quantitative and qualitative control of proteins contributing in DNA damage response and cancer
6. Ubiquitin ligase involved in chromatin regulation
7. Protein knockdown technology: an application of ubiquitin ligase for cancer therapy

Schedule:

Manuscript submission deadline: April 30, 2015

Peer Review Due: May 31, 2015

Revision Due: June 30, 2015

Notification of acceptance by the Guest Editor: July 15, 2015

Final manuscripts due: July 31, 2015