J Cancer Sci Ther 2013, 5:10
http://dx.doi.org/10.4172/1948-5956.S1.030

OMICS Group Conferences
Accelerating Scientific Discovery

3rd World Congress on
Cancer Science & Therapy
October 21-23, 2013 DoubleTree by Hilton Hotel San Francisco Airport, CA, USA

Inhibition of topoisomerase catalytic activity by ruthenium complexes: Search for new anticancer agent

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In a search for both therapeutic and diagnostic agents, transition metal complexes are found of growing significance. The metal complexes constitute a class of chemotherapeutics, which are widely used in the clinic as antitumor and antiviral agents. One of the targets for the cancer chemotherapy are topoisomerases, a class of enzymes in the nucleus of all living cells, which alter the topological states of DNA via breakage and reunion of phosphodiester bonds of one or both DNA strands. DNA topology is critically important in transcription, replication and chromosome structure. It plays several key roles in DNA metabolism and chromosome structure, as primary cytotoxic target for a number of clinically important DNA intercalating agents such as doxorubicin.

The inhibition of topoisomerase largely depends on the nature of the complexes and ligands, and the presence of uncoordinated sites in the skeleton of coordinated ligands. Keeping these reports in view, it was thought worthwhile to explore some new ruthenium-DMSO complexes containing chalcones. These complexes are characterized by spectroscopy, single crystal X-ray crystallography and their DNA cleavage and topoisomerase inhibitory activities are monitored using gel electrophoretic mobility assay technique. A detailed account of such studies will be presented.

Carcinogenesis by stem cell misplacement- A novel cancer theory and its implications

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For rather a long period of time, it has been widely accepted that cancer was caused by gene mutations which is termed somatic mutation theory (SMT). But now the SMT theory has met with increasing doubts, disbelieves, and been facing challenges from other alternative theories. From a pathological perspective, and with the evidences from molecular pathology, hisopathology, and epidemiology, the study shows that the 80 year old paradigm of carcinogenesis process from atypia to *in situ* carcinoma to invasive cancer is wrong. Since 90% of the human cancers are epithelially derived "carcinomas", the turnover of this paradigm naturally negates the SMT theory for SMT sits on this model. The invasive cancer grows out in the stroma *de novo* from misplaced stem cells rather than deriving from the transformed *in situ* carcinoma cells. Also, the talk will show evidences that cancer cells are not so-believed "apoptosis-resistant", but rather "short-lived sick cells". In fact, it is the increased cell death which makes the cancer cells proliferate ceaselessly to compensate for the cell loss, and move to new locations for better chance of survival. This novel cancer theory is designed as SCMT, stem cell misplacement theory. This theory is backed by huge amount of solid evidences and will bring revolutionary conceptual changes to our understanding and treatment of cancer, e.g., *in situ* carcinomas are not genuine cancer biologically, thus should not be treated the same as cancer.

Biography

Rui-An Wang is a Professor of Pathology. After graduation from medical school, he first worked as a histology and embryology teacher and both his MS and Ph.D. degree thesis were on the study of gut endocrinology. In his postdoctoral training, he did reproductive biology, and then shifted to the mammary gland development and carcinogenesis by using molecular biology and gene modified animal techniques. His large gamut of research gave him a pair of eyes by which he sees a lot of scientific issues differently. The SCMT theory he formulated views cancer in a rather different way, and possibly will bring some change to the field.