

DATE:
Thursday,
7 August 2008**VENUE:**
Centre for Life Sciences
Auditorium, Level 1**TIME:**
4.00 - 5.00 pm***Chemical and Biological Mechanisms Linking Inflammation to Cancer and Other Diseases*****Professor Peter Dedon**

Professor of Biological Engineering & Toxicology,
Dept of Biological Engineering, Massachusetts Institute of Technology (MIT);
Associate Department Head & Deputy Director,
MIT Center for Environmental Health Sciences;
Chair of the MIT Committee on Radiation Protection

Host: Professor Ong Choon Nam
Director, Life Sciences Institute
Professor, Dept of Community, Occupational & Family Medicine
Yong Loo Lin School of Medicine, NUS

Biography

Peter Dedon is Professor of Biological Engineering and Toxicology at the Department of Biological Engineering at MIT. Following graduation with a BA degree in Chemistry from St. Olaf College in 1979, he obtained an MD and a PhD in Pharmacology from the University of Rochester in 1987, performing his thesis research on the chemistry of cancer chemotherapeutics with Prof Richard Borch. He pursued postdoctoral studies in chromatin biology with Prof Martin Gorovsky at the University of Rochester, and in the chemical biology of DNA-cleaving antibiotics with Prof Irving Goldberg at Harvard Medical School before joining the Toxicology faculty at MIT in 1991. He currently serves as Associate Department Head, Deputy Director of the MIT Center for Environmental Health Sciences and Chair of the MIT Committee on Radiation Protection. Prof Dedon has served in senior leadership positions in three major scientific societies, on the editorial boards of several international scientific journals, and on dozens of review panels for a variety of private and government agencies, including the NIH, NSF, DOE, and DOD.

Prof Dedon's research program addresses the fundamental chemistry of cancer and other human diseases, with a focus on the chemical mechanisms that link inflammation to disease. He has published extensively on the development and application of ultra-sensitive chemical tools to characterize and quantify damaged biomolecules in human cells, with the goal of developing biomarkers of exposure, pathophysiology and disease risk. The Dedon group's hypothesis-driven approach has revealed striking differences in the quantity and chemistry of damage in DNA and RNA in inflamed tissues, with implications for the development of blood- and urine-based biomarkers of inflammation. His research group is currently extending these analytical approaches to a systems level aimed at spectral quantification of damaged molecules and normal metabolites in cells exposed to drugs, toxins and cytokines. Recent studies are aimed at the many components of the nucleotide pool and the dozens of secondary modifications in RNA.

Abstract

Epidemiological evidence points to a cause-and-effect relationship between chronic inflammation and cancer, most notably with chronic bacterial and viral infections. The basis for this relationship lies in the chemistry and biology of the immune response to the infection, a critical facet of which involves infiltration and activation of macrophages and neutrophils that secrete a battery of reactive oxygen, nitrogen and halogen species intended to eliminate the infectious agent. These reactive species, however, also cause damage to metabolites, polyunsaturated fatty acids, proteins, carbohydrates and nucleic acids in surrounding host cells, with the full spectrum of oxidation, nitration, nitrosation and halogenation chemistry. We have sought to characterize and quantify the biomolecules damaged during inflammation, with the goals of identifying the critical chemistry occurring during inflammation, defining mechanisms that link inflammation to disease and developing small molecule biomarkers of inflammation. This presentation will address recent comparative studies of DNA and RNA as targets for radical-induced damage caused by chemical mediators of inflammation. There are striking differences in both the chemistry and quantity of damage in the two forms of nucleic acid in tissues from infected and exposed mice, with RNA emerging as a central player in the cellular response to inflammation and as a potentially better biomarker of inflammation and oxidative stress. The presentation will also address a new role for the native secondary modifications of nucleobases in tRNA as biomarker signatures of cellular exposure to inflammation.

ALL ARE WELCOME

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