Marianne Staretz
Chairperson of Chemical and Physical Sciences at Cedar Crest College
Allentown, PA, US

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Description
Marianne Staretz, Ph.D., did her doctoral research on the mechanism of the colchicine-tubulin interaction in relation to cancer. A multi-disciplinary approach combining the techniques of organic synthesis, medicinal chemistry and biochemistry was used. She went on to do postdoctoral research at the American Health Foundation, a non-profit research institution dedicated to disease prevention. Her research focused on the effects of isothiocyanates, dietary inhibitors of carcinogenesis, on the metabolism of and formation of DNA adducts by carcinogenic tobacco-specific nitrosamines and benzo(a)pyrene. Staretz has continued some of the cancer prevention research at Cedar Crest College by examining the cancer prevention mechanism of organoselenium agents. She has also expanded some of the toxicology experience gained at the American Health Foundation into the area of forensic toxicology and has several ongoing research projects in this area.

Industry Expertise
Education/Learning, Research

Topics
Bioorganic Chemistry, Organic Synthesis, Medicinal Chemistry, Biochemistry, Toxicology

Affiliations
American Chemical Society (ACS) : Member, Northeastern Association of Forensic Scientists (NEAFS) : Member, American Academy of Forensic Science (AAFS) : Member

Education
State University of New York at Binghamton
Ph.D. Bioorganic Chemistry

University of Scranton
B.S. Biochemistry

Articles
Effects of benzyl isothiocyanate and phenethyl isothiocyanate on benzo[a]pyrene metabolism and DNA adduct formation in the A/J mouse
Carcinogenesis
2000-01-01
Benzyl isothiocyanate (BITC) inhibits lung tumorigenesis induced in A/J mice by benzo[a]pyrene (B[a]P). In contrast, phenethyl isothiocyanate (PEITC) does not. We tested the hypothesis that BITC inhibits B[a]P tumorigenicity in mouse lung by inhibiting DNA adduct formation, and compared the effects of BITC and PEITC...

Inhibition of DNA cytosine methyltransferase by chemopreventive selenium compounds, determined by an improved assay for DNA cytosine methyltransferase and DNA cytosine methylation.
Carcinogenesis
1998-01-01
The organoselenium compounds benzyl selenocyanate (BSC) and 1,4-phenylenebis(methylene)selenocyanate (p-XSC), as well as sodium selenite, are effective chemopreventive agents for various chemically induced tumors in animal models at both the initiation and postinitiation stages...

Evidence for an Important Role of DNA Pyridyloxobutylation in Rat Lung Carcinogenesis by 4-(Methylnitrosamino)-1-(3-pyridyl)-1-butanone: Effects of Dose and Phenethyl Isothiocyanate
Cancer Research
1997-01-01
The tobacco-specific nitrosamine, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), selectively induces lung tumors in F344 rats. NNK is metabolically activated to intermediates that methylate and pyridyloxobutylate DNA. To explore the importance of pyridyloxobutyl DNA adducts in NNK-induced rat lung tumorigenesis, the first study in this report examined levels of these adducts in whole lung and pulmonary cells of F344 rats treated with different doses of NNK (0.3, 1.0, 10.0, and 50 mg/kg; 3 x weekly for 2 weeks)...

Comparative Metabolism of the Tobacco-Related Carcinogens Benzo[a]pyrene, 4-(Methylnitrosamino)-1-(3-pyridyl)-1-butanone, 4-(Methylnitrosamino)-1-(3-pyridyl)-1-butanol, and N\'\'-Nitrosonornicotine in Human Hepatic Microsomes
Drug Metabolism and Disposition
1997-01-01
We compared the metabolism in human hepatic microsomes of three tobacco smoke carcinogens believed to be involved in the induction of cancer in humans: benzo[a]pyrene (BaP), 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNK), and N\'\'-nitrosonornicotine (NNN). The metabolism of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL), a major metabolite of NNK, was also investigated. Although the metabolism of some of these compounds by human enzymes or tissue preparations has been previously examined in some studies, they have never been compared in the same human hepatic samples. Moreover, there have been no previous reports of NNAL metabolism by human tissues or enzymes...

In Vitro and In Vivo Reduction of Erythrocyte Sorbitol by Ascorbic Acid
Diabetes
1989-01-01
The in vitro accumulation of sorbitol by human erythrocytes incubated in a physiological glucose medium was found to be strongly reduced by the addition of ascorbic acid (AA). A maximal inhibition of sorbitol in the erythrocytes of 98.3% occurred when the concentration of AA was at its peak in the cells...
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