COLLEGE OF PHARMACY
University of Cincinnati Medical Center

SUMMER RESEARCH OPPORTUNITIES
FOR UNDERGRADUATE WOMEN

APPLICATION DEADLINE: MARCH 3, 2003

The College of Pharmacy is pleased to offer the following research project(s) for the summer of 2003. Interested students are urged to contact the faculty member(s) directing the project(s) that most interest them. By contacting the faculty member, you can discover more about the project, learn what your responsibilities will be, and if possible, develop a timetable for the twelve-week research period.

DNA Replication and Cell Proliferation

Professor Carol A. Caperelli
117 Wherry (513) 558-0730 FAX: (513) 558-0978
E-Mail:Carol.Caperelli@UC.Edu

De novo purine biosynthesis provides the purine nucleotide starting materials for RNA and DNA synthesis and is essential for DNA replication and cell proliferation. It had long been speculated, and more recently demonstrated, that inhibition of this metabolic pathway could provide an effective approach for cancer chemotherapy. The first of two folate-dependent enzymes in the pathway, glycinamide ribonucleotide (GAR) transformylase, has been validated as a cancer chemotherapeutic target. We are pursuing studies to elucidate the mechanism and structure-function of human GAR transformylase. Our structural studies, in collaboration with Dr. Janet Smith at Purdue, will provide a platform for the structure-based design of specific inhibitors of human GAR transformylase as potential chemotherapeutic agents. Current projects include cloning, purification, and characterization of site-directed mutants of the human enzyme and kinetic characterization of potential inhibitors of the enzyme.

MDMA – Drug of Abuse

Professor Gary Gudelsky
110 Wherry (513)558-5735 FAX: (513)558-0978
E-Mail: Gary.Gudelsky@UC.Edu

The methamphetamine analogue, 3,4-methylenedioxymethamphetamine (MDMA, 'Ecstasy') continues to be a popular drug of abuse despite evidence in animals and humans that the drug produces long-term alterations in serotonin neurons. Researchers have viewed MDMA as selectively "neurotoxic" to serotonin axon terminals in the brain on the basis of findings that the repeated administration of MDMA results in reductions in serotonin concentrations in the brain. However, there is little histopathological evidence to support the contention that MDMA truly is neurotoxic. Recently, neuronal

degeneration resulting from traumatic brain injury in humans has been shown to be accompanied by the proteolytic cleavage of the cytoskeletal protein tau. The intent of the current project is to utilize this biomarker, viz., cleaved tau, of neurotoxicity to establish that MDMA induces axonal degeneration in the rat brain.