Instructions for Abstract Submission

1. Abstracts are due before February 25, 2003
2. Please format abstract exactly according to the "Example Abstract" provided on the 3rd page of this document.
3. Please submit your abstract to Gagan Singh in BSB Room 508 (Department of Pharmacology) or Marge Riley in the Graduate School of Basic Medical Sciences Office by February 25, 2003.
4. Submission of final abstract MUST include:
   a) A printed copy of your abstract
   b) A saved copy of your abstract on a 3.5" diskette

The 15th Annual Graduate Student Research Forum

Abstract Submission Application

Please type or print legibly and submit to Gagan Singh in BSB Room 508 (Department of Pharmacology) or Marge Riley in the Graduate School of Basic Medical Sciences Office by February 25, 2003:

1. Student Information

_____

Last Name First Name Middle Initial

_____

Department

_____

Telephone Number Email Address

☐ M.S. Student ☐ Ph.D. Student Years enrolled in graduate School: _____

2. Faculty Advisor Information

_____

Name

_____

Department

3. Abstract and Presentation Preference

_____

Title of Abstract

_____

Presentation Preference: ☐ Oral ☐ Poster

Low Dietary Sodium Intake Affects Renal Microvascular 20-Hydroxyeicosatetraenoic Acid (20-HETE) Levels; Relationship to Cyclooxygenase (COX) Activity

Monica Cheng, Pharmacology

Faculty Advisor: Dr. M. A. Carroll

Rat preglomerular microvessels (PGMVs) release cytochrome P450 (P450) derived HETEs. Further, angiotensin II (AII) stimulates 20-HETE release, an effect mediated via AT_2 receptors coupled to phospholipase C activity. We now report on P450-HETE release by microdissected interlobar and arcuate arteries obtained from male Sprague-Dawley rats. Metabolism of ^{14}C arachidonic acid (7 μM) in the presence of NADPH (1mM) and indomethacin (Indo; 2.8 μM) to P450-HETEs was higher in arcuate (3.64±0.60%) compared to interlobar arteries (0.77±0.18%) based on HPLC retention times. However, when COX was active (- Indo), levels of HETEs were reduced to 0.47±0.08% by arcuate and 0.43±0.10% by interlobar arteries, respectively. Low dietary salt treatment (LS; 0.05% NaCl) for 7 days increased 20-HETE levels by 2-3 fold over controls (0.4% NaCl), e.g., 20-HETE release in the presence of NADPH and Indo from interlobar and arcuate arteries of LS treated rats was 6.36±1.78 and 14.31±4.64 ng/mg protein, respectively, based on GC-MS quantitation. However, 20-HETE levels in PGMVs were diminished when Indo was omitted from the incubate. Thus, 20-HETE levels vary within the renal microvasculature. Low dietary salt intake induces 20-HETE formation by both arcuate and interlobar arteries. The diminished 20-HETE levels in PGMVs when COX was not inhibited are, presumably, related to 20-HETE metabolism by COX-2 to prostaglandin analogs, the increased COX-2 activity, elicited by low salt intake, then serving as a metabolic pathway for 20-HETE.