

## Research Night 2009

### Instructions for Abstract Submission

#### ***General information:***

- All research that has not previously been presented at Research Night is eligible for submission.
- The deadline for submission is close of business February 13, 2009. No abstracts will be accepted after that date.
- To submit an abstract, please send an e-mail to [resabs@ochsner.org](mailto:resabs@ochsner.org) with the word-processed document as an attachment.
- For questions, please contact Rita Premo at (504) 842-7396 or [resabs@ochsner.org](mailto:resabs@ochsner.org).

#### ***Formatting and content requirements:***

##### **On the title page, please include:**

- Complete contact information for each author, including affiliation, highest degree(s) received (e.g., MD, MS, RN, MPH), mailing address, e-mail address, and phone and fax numbers
- Designated primary contact author
- Designated presenter
- List of 3 to 5 keywords that describe the content of the subject
- Relevant therapeutic area, such as Anesthesiology, Cellular Immunology, General Surgery, Hospital Medicine, Infection Control, Nephrology, Nursing, Orthopedics, or Vascular Surgery
- IRB number, if applicable
- Notice if you do not want your abstract published (in which case, just the title and authors will be printed).

##### **The abstract itself should include:**

- **No** figures, tables, or references.
- One-inch margins on all sides and left justification
- Headings of:
  - For original research: Background, Objectives, Methods, Results, and Conclusions
  - For case reports: Introduction, Case Report, and Discussion
- 250 words or less.
- All abbreviations written out on first reference
- Units of measure for any laboratory results, etc.
- Generic drug names
- Genus and species names in italics

Failure to fulfill these requirements will result in the draft being returned to the primary author.

An example of a correct abstract follows.

## **THE ROLE OF THE RENIN-ANGIOTENSIN SYSTEM IN MEDIATING SALT-OVERLOAD RENAL INJURY IN SPONTANEOUSLY HYPERTENSIVE RATS**

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**Background:** We have previously demonstrated that dietary salt excess induces cardiovascular and renal injury and that the local tissue renin-angiotensin system (RAS) may mediate heart damage.

**Objectives:** We sought to examine whether RAS is involved in the pathogenesis of renal damage induced by salt overload.

**Methods:** Male, 8-week-old spontaneously hypertensive rats (SHRs) were divided into 4 groups. The control group was given regular chow; the remaining 3 groups were given chow with 8% salt. In addition, the third group was given the angiotensin II receptor blocker (ARB) candesartan (10 mg/kg/d), and the fourth another ARB, losartan (30 mg/kg/d). Systemic hemodynamics and indexes of renal function were determined after 8 weeks of treatment.

**Results:** Compared with the controls, mean arterial pressure increased in salt-loaded rats ( $182 \pm 5$  vs.  $164 \pm 4$  mmHg) and was not decreased by candesartan ( $178 \pm 5$  mmHg) or losartan ( $183 \pm 4$  mmHg). Indexes of renal function, including renal blood flow ( $1.7 \pm 0.3$  vs.  $3.2 \pm 0.4$  mL/min/g), glomerular filtration rate ( $0.5 \pm 0.1$  vs.  $1.1 \pm 0.1$  mL/min/g), and urinary protein excretion ( $111 \pm 7$  vs.  $21 \pm 4$  mg/day) were significantly ( $p < 0.05$ ) and adversely affected by salt overload; they were completely restored with both drugs.

**Conclusions:** These results demonstrate that dietary salt excess adversely affects renal function, hemodynamics, and structure. Angiotensin receptor blockade did not affect arterial pressure but prevented other adverse effects of salt overload, indicating that renal damage was not dependent on arterial pressure but, more likely, on another mechanism involving RAS. This is further supported by the findings that two different ARBs exerted similar effects.