



RESEARCH THE POSSIBILITIES....
THE TWELFTH ANNUAL
NCSSSMST STUDENT RESEARCH SYMPOSIUM
June 2-5, 2005

ABSTRACT GUIDELINES FOR PUBLICATION

Dear Participant:

Please submit this information to your teacher by May 7, 2005. Your teacher will forward it to Villanova University:

E-mail your abstract as an attachment in WordPerfect or Microsoft Word for Windows (do not send Macintosh files) to: gina.sementelli@villanova.edu. A confirmation of receipt will be sent. If no confirmation is received by May 14, 2005, please email again or call Ms. Gina Sementelli directly at 610-519-8029.

Please follow these guidelines:

Maximum of 300 words; one page; double-spaced; including the following information:

- Student name, High School, Mentor (s) name (s)
- Title
- Text Including: Purpose of experiment, Method used, Results, and Conclusions
- *Optional:* include any possible research applications (minimal reference to previous work may be included)

Thank you for your interest in participating in this exciting opportunity!

Sincerely,

Gina M. Sementelli
Assistant Director of Admission
Coordinator of Engineering & Science Recruitment

Sample abstract from University of Miami undergraduate research publication:

Sharon Lewis

University of Miami

Dr. Bethea and Dr. Mosser, The Miami Project to Cure Paralysis

Expression of iNOS and NF- κ B in the Spinal Cord Two Months Post-Injury

Traumatic spinal cord injury often results in paralysis, which is presently incurable. The immediate effect of injury to the cord is necrosis, the death of cells. Necrosis sets off an inflammatory response that triggers secondary cell death mechanisms including excitotoxicity and apoptosis (programmed cell death). Several factors are released and activated as part of the inflammatory response including nuclear factor- κ B (NF- κ B), tumor necrosis factor- α (TNF- α) and inducible nitric oxide (iNOS). NF- κ B is a transcription factor that mediates inflammation and TNF- α is a pro-inflammatory cytokine. Both are thought to be important regulators of injury-induced apoptosis. iNOS is the enzyme responsible for generating nitric oxide, which produces toxicity in cells.

Previously, suppression of TNF- α was shown to decrease neuronal loss, facilitate locomotor recovery, and attenuate disruption to the blood-brain barrier in closed brain injury (Shomhi et al., 1996). The present study examines the effects of anti-TNF- α in the spinal cord two months post-injury. Following a moderate spinal cord contusion injury, neutralizing antibody to TNF- α or saline control was administered i.v. to rats 30 minutes and behavioral and anatomical data were obtained up to two months post-injury. Presence of NF- κ B and iNOS in injured spinal cord tissue sections was detected using immunospecific antibodies and expression of iNOS was additionally quantified by western blot analysis. Total lesion area was traced using a computer-aided image analysis program, and the total volume of the lesion was determined.

Well-defined lesion areas were identified by neuronal loss and presence of inflammatory cells. Up to two months post-injury, both NF- κ B and iNOS were localized in neurons as indicated by double-staining with a neuronal-specific marker. Evidence for iNOS was also indicated by western blot analysis. These results indicated a chronic inflammatory response due to traumatic spinal cord injury.