



## Call for Abstracts and Abstract Submission Form

2012 Student Research Symposium  
Wednesday, April 25, 2012

**SUBMISSION DEADLINE: Tuesday, April 17, 2012, 4:00 pm CDT**

The ULM Student Research Symposium Task Force is pleased to formally request abstracts of completed research from ULM undergraduate and graduate students for presentation at the *12th Annual Student Research Symposium*. In order to submit, the student must have been enrolled during the 2011 - 2012 academic year. Submissions should be made to a Symposium Representative within your college. A list of those members can be found [here](#).

Questions may be directed to committee chair Dr. Tammy Parker or your college representative. **The heading for the abstract should include the title, authors, academic college and department, and the name of the University (see sample abstract). Abstracts should be typed or printed entirely within the box below, using Times New Roman 10 pt. Font.**

**CASPASE-8 PATHWAY MEDIATES THE APOPTOTIC EFFECTS OF TOCOTRIENOLS IN NEOPLASTIC (+SA) MOUSE MAMMARY EPITHELIAL CELLS.** Sumit Shah and Paul W. Sylvester, College of Pharmacy, The University of Louisiana at Monroe, Monroe, LA 71209-0470

Tocotrienols are members of the vitamin E family of compounds that exhibit significant anticancer activity. Although, tocotrienols have been shown to induce programmed cell death in neoplastic (+SA) mouse mammary epithelial cells, the exact mechanism mediating apoptosis is presently unknown. An initial step in apoptosis is the activation of “initiator” caspases (caspase-8 or -9) that subsequently activate “effector” caspases (caspase-3, -6 and -7) and ultimately lead to DNA fragmentation and cell death. Studies were conducted to determine whether tocotrienol-induced apoptosis is mediated by the activation of caspase-8 pathway or caspase-9 pathway. +SA cells were grown in culture and maintained on serum-free media containing either, 0-50 $\mu$ M tocotrienol-rich fraction of palm oil (TRF), 0-20 $\mu$ M  $\gamma$ -tocotrienol ( $T^3$ ), or 0-400 $\mu$ M  $\alpha$ -tocopherol (T). Treatment-induced apoptosis was assayed by DNA fragmentation using horizontal agarose gel electrophoresis, and viable cell number was assayed using MTT colorimetric assay. Caspase-3, -8, and -9 activity was measured using colorimetric assay kits and active caspase-3, -8, and -9 levels were determined by western blot analyses. Effect of selective caspase-8, -3, and -9 inhibitors on tocotrienol-induced cell death was measured using colorimetric caspase activity assays and Western blot analyses. Results showed that treatment with cytotoxic doses of TRF or  $\gamma$ - $T^3$  induced apoptosis in +SA cells in a time- and dose-dependent manner, whereas treatment with 0-400 $\mu$ M  $\alpha$ -T was not found to be cytotoxic. 20 $\mu$ M  $\gamma$ - $T^3$  or 50 $\mu$ M TRF induced > 50% +SA cell death within 24hr and resulted in a significant increase in caspase-8 and caspase-3, but had no effect on caspase-9 activity. Co-treatment of either 20 $\mu$ M  $\gamma$ - $T^3$  or 50 $\mu$ M TRF with 1 $\mu$ M selective caspase-8 or caspase-3 inhibitors completely block tocotrienol-induced apoptosis and activation of caspase-8 and -3, respectively. In summary, these findings demonstrate that tocotrienol-induced apoptosis, in neoplastic +SA mouse mammary epithelial cells, is mediated by the activation of caspase-8 and caspase-3, and is independent of caspase-9 activation. Supported by NIH grant CA86833.

Please type or print only within the boundaries of the box.

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**Hometown (City/State or Country):**

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**Presentation Preference: Poster**

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