IN VIVO FLORESCENT IMAGING OF HOLLOW POLYMERIC NANOCAPSULES LOADED WITH ALEXAFLOUR 750. Venkata Suresh Patthipati[1], Christopher Osgood[1], James Swanson[1], Ramjee Balasubramanian[2], Kalpana Mahadevan[2], & Sangbum Han[2], [1]Department of Biological Sciences, ODU, Norfolk, VA, [2]Department of Chemistry and Biochemistry, ODU, Norfolk, VA. Nanoparticle based imaging strategies hold significant promise in addressing current diagnostic and therapeutic challenges. However, previous nanomaterials failed to clear effectively from the circulation via the renal route. This work demonstrates the use of highly fluorescent, water-soluble, resorcinarene nanocapsules (122 nm) and their effectiveness in biomedical imaging. These hollow polymeric nanocapsules loaded with Alexa Fluor 750 fluoresce brightly in the urinary bladder of mice under the *in vivo* fluorescent imager indicating that their major route of clearance is through the kidney. TEM analysis of the urine recovered from the mice showed the presence of intact nanocapsules. Such florescent nanocapsules can be very effective in visualizing the parts of the urinary tract. They are particularly useful in abdomino-pelvic surgeries ranging from caesarian delivery to laparoscopy. The use of these nanocapsules are not restricted to the imaging as they could be loaded with a host of molecules making them efficient drug delivery vehicles in treating cancer and other pathologies.

THE DIFFRENTIAL ROLE OF CBP UBIQUITIN LIGASE ACTIVITIES IN p53 REGULATION. Oluwatoyin E. Akande & Steven R. Grossman, Dept. of Microbiology & Immunology, Virginia Commonwealth Univ., Richmond, VA 23298. The acetyltransferase CREB-Binding Protein (CBP) is a known transcriptional co-activator involved in p53 regulation and has been shown to encode cytoplasmic, but not nuclear, E3 autoubiquitination and p53-directed E4 ubiquitin ligase activities. In this work, we sought to determine the regulation of differential ubiquitin ligase activities between nuclear and cytoplasmic CBP. Understanding how CBP regulates p53 ubiquitination and stability may lead to potential therapeutics that can modulate p53 stability in cancer cells. We show that a nuclear interacting factor represses cytoplasmic CBP E3 (autoubiquitination) ligase activity in unstressed U2OS cells. We incubated immunoprecipitated cytoplasmic CBP from U2OS cells with either nuclear lysates or CBP- immunodepleted nuclear fraction. Our results showed reduced CBP E3 autoubiquitination in the mixtures when compared to purified cytoplasmic CBP alone. We further showed that DNA damage by doxorubicin (dox) in U2OS cells activated E3 ligase activity in the nuclear fraction. These observations may provide a possible explanation for the inactive nuclear E3 ubiquitin ligase activity but active cytoplasmic E3 and p53-directed E4 ubiquitin ligase activities of CBP in unstressed cells. The dox dependent activation of nuclear CBP E3 autoubiquitination may result from dox dependent inactivation or re-localization of the nuclear inhibitor to other cellular compartments. Further work will identify the nuclear interacting factor and in parallel, determine if the differential ubiquitin ligase activity observed between cytoplasmic and nuclear CBP is otherwise dependent on post-translational modifications of CBP itself.

ENRICHMENT OF CAPTIVE SQUIRREL MONKEYS. LaCheryl A. Ball & Eric L. Walters, Department of Biology, Old Dominion University, Norfolk, VA 23529. Food enrichment is a technique used by the zoo industry to promote overall wellness of animals in captivity. I measured responses of captive, *Saimiri sciureus* squirrel

monkeys to food enrichment at the Virginia Zoo (Norfolk, VA). The research involved determining pre-treatment activity levels in order to test the effect of food enrichment on post-treatment activity levels. I hypothesized that foraging and active behaviors would increase as follows: baseline<post-enrichment<treatment. The experiment was divided into three phases: the first of which provided baseline data on the population's behaviors and activity levels prior to enrichment. The second phase involved the introduction of enrichment feeders on alternating treatment and control days. The third phase involved gathering post- treatment behavioral data, which determined if there were any protracted effects of food enrichment on behavior after the feeders were removed. Ultimately, introduction of food enrichment resulted in a 21% increase in foraging behaviors of both the adult male and juvenile males and a 16% increase in the adults females. In conclusion, food enrichment was a successful method of promoting foraging behaviors and increasing activity levels in captive squirrel monkeys and has important implications for increased health and well-being of captive primates.

EPIGENETICS AND ALZHEIMER'S DISEASE: DISTINCT PATTERN SHIFTS IN DNA METHYLATION. Noor M. Taher, Courtney A. McKenzie, Rebecca C. Garrett, Matthew S. Baker & Gary D. Isaacs, Department of Biology and Chemistry, Liberty University, Lynchburg VA 24502. Amyloid beta (Aβ) plaques are one hallmark of Alzheimer's disease. Despite ongoing research, there remains some ambiguity surrounding the role of Aβ in the pathogenesis of this neurodegenerative disease. Even more obscure, however, are the epigenetic changes these plaques cause to neurons. To that end, we wanted to shed more light on the changes in DNA methylation neurons incur when treated with Aβ in vitro. In order to accomplish this, we isolated DNA from Aβ-treated and control neurons and differentially digested the two samples with either a methylation-sensitive or a methylation-insensitive restriction endonuclease. Amplified fragments were then co-hybridized to a commercial promoter microarray. Data analysis revealed a subset of genomic loci that shows a significant change in DNA methylation following Aβ treatment. After mapping these loci to nearby genes, we discovered high enrichment for cell-fate genes that control apoptosis and neuronal differentiation. Finally, we incorporated these genes in a possible model suggesting the means by which Aβ contributes to the brain shrinkage and memory loss seen in Alzheimer's disease.

CHARACTERIZATION OF *CITROBACTER RODENTIUM* TRANSMISSIBLE COLONIC HYPERPLASIA AND IMMUNE RESPONSES IN STREPTOMYCIN-TREATED AND CONVENTIONAL MOUSE MODELS. M. W. Canfarotta[1], M. H. VanTil[1], D. A. DeWitt[1], T. A. Snider[2] & A. J. Fabich[1], [1]Department of Biology and Chemistry, Liberty University, Lynchburg VA 24502 & [2]Center for Veterinary Health Sciences, Oklahoma State University. *Citrobacter rodentium* is an enteric murine pathogen similar to the human pathogen enterohemorrhagic *Escherichia coli* (EHEC) as it forms an attaching and effacing (A/E) lesion during gastrointestinal infection. Previous studies have utilized *C. rodentium* as a mouse model for EHEC disease in humans, however there is currently no comprehensive study characterizing the course of infection in specific mouse models with or without streptomycin treatment. The use of streptomycin effectively depletes the facultative anaerobic niche allowing for the direct study of *C. rodentium* colonization, whereas a model lacking antibiotic treatment