August 9-10, 2007

2007 Ohio Student Research Forum

Wright State University
Dayton, OH

RESEARCH ABSTRACT FORM

TITLE: The Effect of DNA Damaging Agents on DNA Repair Pathway

AUTHOR: Javier Baez

MENTOR(S): Dr. Alo Ray, Dr. Song Qin, and Dr. Altaf A. Wani

INSTITUTION: The Ohio State University

The preservation of genomic stability is paramount to the survival and propagation of all cellular organisms. Genomic DNA is continuously exposed to a wide variety of exogenous and endogenous genotoxic agents, including radiation, chemical reagents, and internal metabolic compounds, that introduce DNA lesions. Repair of the DNA requires several factors that sense the damage, transmit the signal and eventually remove the lesion. Additionally, there are numerous DNA repair pathways that deal with different types of DNA damage caused by DNA damaging agents. Exposing yeast mutants (deficient in protein factors) to varying DNA damaging agents can help better understand the DNA damage repair process, as well as to identify the DNA repair pathway that specific protein factors are involved in. For these experiments, we used several yeast mutants to examine their growth sensitivity to two different DNA damaging agents; methyl methanesulfonate (MMS) and hydroxyurea (HU). Ten-fold serial dilution spot tests on solid DNA damaging media were performed to examine colony formation and determine sensitivities to DNA damaging agents. For each experiment the survival and growth of the wild-types (WT) and mutants were compared. Our results demonstrate that the depletion of Rif1 protein causes sensitivity to MMS when compared to isogenic wild type strains whereas exposure to HU does not show any sensitivity. The phenotype suggests that yeast Rif1 plays a role in DNA damage repair and genomic stability. Based on these results, further experimentation is underway to more accurately quantify the survival rates of the rif1Δ and WT strains in the presence of specific MMS concentrations. Moreover, investigation is in progress to determine if Rif1p is required for DNA replication fork stability and processing after alkylation damage.