Project ID: 171

Senior Division

Cellular and Molecular Biology

Alyna Figueroa
Central Union High School
Gr. 12

The Effect of Ozone on Epithelial Cell Growth

The purpose of this experiment was to determine the effect of ozone exposure on cell growth. The null hypothesis was: ozone exposure has no effect on epithelial cell growth.

To perform this experiment, 20 cell culture tubes in four groups were prepared the following way: 5 control = 0 min, 5 low exposure = 30 min, 5 medium exposure = 1 hour, 5 high exposure = 2 hours. Then tubes were massed using an analytical balance, incubated at 37 degrees celsius for three days, massed again after incubation, and the data was recorded and compared. This process was repeated for another 20 cell culture tubes but for two weeks. After two weeks, the control tube samples cells were observed under a microscope.

The average percent change in mass found for the control group was 0.78% (three days) 1.814% (two weeks), the low was 0.536% (three days) and 1.622% (two weeks), the medium was 0.152% (three days) and 0.112% (two weeks), and the high was 0.118% (three days) and 0.286% (two weeks). After performing the Fisher exact test, the calculated one-tailed p-value was 0.02955665025. This is lower than the significance value 0.05.

In conclusion, the null hypothesis was rejected because the calculated one-tailed P-value from the Fisher exact test was lower than the significance value of 0.05 and the average percent change in mass between exposure levels showed a trend of decrease in mass, and the microscopic observation showed animal cells.

Project ID: 172

Senior Division

Cellular and Molecular Biology

Yunshu Hao

Sage Creek High School

Gr. 11

Enable AAV to Target NK Cells for CAR Cell Therapy in Vivo

CAR cell therapy for cancer has demonstrated success, but the ex-vivo transformation of immune cells into CAR cells is time-consuming, hindering therapy effectiveness. Shifting this process in-vivo could substantially reduce the therapy time. However, current in-vivo delivery methods face safety and specificity challenges.

In my study, I used peptides to modify Adeno-Associated Viruses (AAV), a gene delivery tool with safety advantages, enabling the AAV to specifically target immune cells. Natein, a CD56-binding protein, was selected as the peptide source, as CD56 is widely distributed on NK cells, a major type of immune cells. Through computer analysis, I predicted the structure of Natein and identified eight potential peptides binding directly to CD56.

Furthermore, I designed 8 AAV variants, each incorporating a CD56-binding peptide. To assess transduction ability, I infected NK92 cells with these AAV variants and the wild-type AAV (efficiency control). Additionally, I evaluated specificity by transduction of NK92 and Jurkat cells (specificity control). The results revealed that an AAV variant led to 42% of NK92 cells expressing EGFP, compared to 0.04% for control. The infectivity of this variant on Jurkat cells remained at 5.2%, almost unchanged compared to that of the wild-type AAV, indicating that the enhanced transduction is specific to CD56-expressing NK cells.

The results demonstrated that a novel AAV variant exhibited high efficiency and specificity in targeting and transducing NK cells. This presents a promising vehicle for delivering CAR genes to NK cells, enabling in vivo cell transformation for CAR cell therapy.

Project ID: 173

Senior Division

Cellular and Molecular Biology

Ethan Leem

Canyon Crest Academy

Gr. 9

Paraquat Exposure Triggers Microglial Activation through Neuroinflammatory Pathways

Paraquat dichloride is used in herbicides and is associated with numerous neurodegenerative diseases. Cheap and effective, paraquat use is unregulated in developing countries. Paraquat in herbicides is ~0.2%. However, paraquat's neurological effects and breadth of subsequent neurodegeneration is unclear. Alzheimer's disease (AD) has been assumed to stem from neuron issues. However, microglia, specialized central nervous system macrophages, induce AD through excessive phagocytosis of synapses. Microglia phagocytosis is characterized by their "activated form." Abundance in herbicides warrants an analysis of how paraquat activates microglia, which would signal neuroinflammation. I hypothesize paraquat exposure will activate the BV2 microglial cell line through neuroinflammatory pathways.

To examine if paraquat activates microglia, different concentrations of paraquat were administered to BV2 cells. The paraquat concentration that was tested in this experiment is 1, 5, 10 and 100 micro-Molar. Herbicide concentrations are 8mM, which is ~1000x greater, indicating chronic paraquat exposure leads to neurodegeneration. Morphological and population change in BV2 cells were quantitated. To observe a neuroinflammatory pathway, western blotting was performed using a primary antibody for phosphorylated AKT, which is activated in microglia under inflammation. Also, western blotting was performed for P38 MAPK using corresponding antibodies. P38 MAPK protein is highly activated in aging brains.

BV2 microglial cells exhibited activation when exposed to 10 micro-Molar paraquat in 1-2 days. 100 micro-Molar paraquat killed BV2 cells within 48 hours. P38 MAPK increased in 24-48-hours when treated at 1 or 5 micro-Molar paraquat. Phosphorylated-AKT increased in 48 and 72-hour treatments.

Project ID: 174

Senior Division

Cellular and Molecular Biology

Medha Nandhimandalam
Westview High School
Gr. 12

Mechanisms of Somatic Gene Variation

The most prevalent neurodegenerative disease among patients aged greater than sixty-five is Alzheimer's disease. One of the factors that is thought to contribute to Alzheimer's is genetic mosaicism, a phenomenon whereby cells from the same organism have non-identical genomes. One process that contributes to genomic mosaicism is the creation of gencDNAs', a process by which RNA is retranscribed into cDNA and reinserted elsewhere in the genome. The hypothesis was that DNA damage leads to gencDNA formation. To investigate this phenomenon, DNA breaks were introduced in differentiated and undifferentiated neural progenitor cells using H2O2. The chromosomal DNA was extracted and used as a template for qRT-PCR with the Taqman probe on the samples. The Taqman probe specifically amplifies processed intronless genes. The PCR products are sized around 300 bp. The Taqman probe is specifically activated when this product is the processed gencDNA. The results showed that increased Taqman probe activation occurs following H2O2 treatment, indicating that gencDNAs increase after inducing DNA damage. The results support the hypothesis that DNA damage promotes the insertion of gencDNAs within the genome.

Project ID: 175

Senior Division

Cellular and Molecular Biology

Gavin Ni
La Jolla Country Day School
Gr. 11

Effects of a Stress-protective Protein from Tardigrade on Stress-resistance and Aging of Yeast

AWARDS:

CSEF Qualified

The study of stress-resistance is important for understanding the aging process and addressing the unmet needs of people affected by aging related disorders. In this study, I used Yeast, where oxidative stress plays a critical role in aging, as a model system to test the stress-resistant properties of Dsup, a protein from Tardigrade. Tardigrade is well-known for its capability to survive under extreme conditions such as in outer space or in vacuum. This incredible capability can be partially attributed to Dsup. This project is designed to test if the functional effects of Dsup are applicable to other organisms.

To test my hypothesis that expression of Dsup increases the cells' resistance to oxidative stress, I utilized an engineered yeast strain, identified a condition to induce Dsup expression without generally affecting the health of yeast cells, exposed these yeast cells to hydrogen peroxide to cause oxidative stress and compared their survival with wild type yeast cells by using a fluorescent dye to quantify cell death.

My results showed that at four hours of exposure to hydrogen peroxide, the Dsup-expressing cells exhibited statistically significant lower cell death than the wild type cells, showing 37% of mean fluorescence intensity of wild type cells. These results showed that Dsup expression allowed yeast cells to better cope with oxidative stress conditions. This study provides a better understanding of the functional role of Dsup in other organisms and could lead to new ideas to enhance stress resistance and extend longevity.

Project ID: 176

Senior Division

Cellular and Molecular Biology

Shreeya Patel
Del Norte High School
Gr. 11

Activation of Tumor-Suppressor p53 in Oncogene-Induced Senescence

AWARDS:

Society for In Vitro Biology
CSEF Qualified

Cellular senescence occurs when a cell permanently stops dividing in response to severe DNA damage or oncogenic stress but does not undergo cell death. This phenotype is characterized by damaged DNA, altered gene expression and inflammation. Senescence is a hallmark of aging, and senescent cell inflammation has been linked to a range of age-related diseases, including cancer. Therefore, targeting senescence is a valuable approach to slowing progression of these diseases. Senescence can be induced in many ways: through irradiation, drugs, or expression of oncogenes. The tumor suppressor p53 is a prominent regulator for these senescence processes, and our lab has recently shown that a drug that activates p53 is able to suppress irradiation-induced inflammation from senescent cells. However, activation of p53 has not been extensively studied in oncogene-induced senescence, leaving a gap in knowledge about how p53 works with this model. Here, I have explored the intricacies of oncogenic RAS-induced senescence, specifically relevant to p53. Interestingly, our results showed that p53 activation is different in oncogene-induced senescence compared to irradiation-induced senescence. This may be due to the role of p53 regulator MDM2, which may interact with different downstream targets in oncogene-induced senescence. As escape from oncogene-induced senescence often leads to tumor growth and proliferation, these results provide insight into the characteristics of this type of senescence. Our data contributes to our understanding of the role of senescence in cancer and aging, and these results are relevant to therapies that prevent cancer and the harmful effects of senescence.

Project ID: 177

Senior Division

Cellular and Molecular Biology

Sophia Tran

Scripps Ranch High School

Gr. 11

Exploring Anatomical Variability in the Cerebral Cortex of the Human Brain

Folding on the surface of the brain is not random; rather, it is based on the pattern of connections between areas of the cerebral cortex and subcortical areas. Thus, it has functional significance. The current study uses a comparative approach to study the variability in surface anatomy that human brains exhibit across individuals. We examined the brain of three species of primates: Macaque (Rhesus macaque), Baboon (Papio hamadryas), and human (Homo sapiens). The goal of the study is to determine whether anatomical differences between two hemispheres of the same brain (intra-subject) are more, less, or equivalent to the differences between hemispheres in different brains (intra-subject).

Our results show that inter-hemispheric difference in lower primates are less significant than in humans and equivalent to difference between individuals. In humans, both inter-hemispheric and inter-subject differences are more prominent and are equivalent, fact that corroborates the prominent hemispheric functional specialization that characterizes the human brain.