



CALIFORNIA STATE SCIENCE FAIR  
2012 PROJECT SUMMARY

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| <b>Name(s)</b><br>Easun Arunachalam   | <b>Project Number</b><br><b>S0502</b> |
| <b>Project Title</b><br><b>A Novel Method of Increasing Phytoremediation of Heavy Metals in Wastewater through the Fenton Reaction</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>The research goals of my experiment were to determine if soybean (Glycine max) plants would absorb a greater amount of iron when the iron was at a higher oxidation state and to determine the pH at which the phytoremediation of iron is most effective. Iron was used as a model in this study due to its relatively low toxicity as compared to that of other heavy metals.</p> <p>It was hypothesized that phytoremediation of Fe<sup>(3+)</sup> would be more effective than that of Fe<sup>(2+)</sup>, and a pH of 5 would be most conducive to iron absorption.</p> <p><b>Methods/Materials</b><br/>G. max seeds were grown in water for two weeks, and were placed in jars containing Iron(II) sulfate heptahydrate [FeSO(4). 7H(2)O] solution. Hydrogen peroxide solution was added to change the oxidation state of the free iron (II) ions, as described by the following reaction:<br/>Fe<sup>(2+)</sup> + H(2)O(2) --&gt; Fe<sup>(3+)</sup> + .OH + OH<sup>-</sup></p> <p>The pH of the solutions containing H(2)O(2) was adjusted to 5, 6, 7, or 8. The control samples contained G. max seedlings in iron (II) sulfate solution without hydrogen peroxide. Test strips were used to determine the concentration of iron present in solution two days and four days after the beginning of the phytoremediation. Thirty-two trials were conducted with a sample size of 1600 seeds.</p> <p><b>Results</b><br/>G. max absorbed a greater amount of iron at a higher oxidation state, and the least amount of dissolved iron was detected in solutions of pH 8.</p> <p><b>Conclusions/Discussion</b><br/>It appeared that phytoremediation in samples containing iron in a higher oxidation state was more effective, and that a pH of 8 is most conducive to iron absorption. The reactions occurring within the solutions of pH 8 were further investigated with a view to determining if the iron (III) hydroxide precipitate formation resulted in the unexpectedly low dissolved iron levels. The results of this follow-up experiment indicate that the greatest amount of iron was actually removed in solutions of pH 5, substantiating my hypotheses.</p> |                                       |
| <b>Summary Statement</b><br>This study investigates whether a higher oxidation state and altered pH level will improve the efficacy of heavy metal removal by hyperaccumulating plant species.  |                                       |
| <b>Help Received</b><br>Mother helped obtain materials and assemble poster board.   |                                       |



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| <b>Name(s)</b><br><b>Vikas C. Bhetanabhotla</b>   | <b>Project Number</b><br><b>S0503</b> |
| <b>Project Title</b><br><b>A Novel Approach to Combating Brain Tumors Using Hyperpolarized Carbon 13 NMR</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>The purpose of this research is to study how tumor cell growth can be stopped through inhibition of key energy producing pathways, such as Glutaminolysis. A new method to inhibit Glutaminolysis by hyperpolarizing Glutamine using Dynamic Nuclear Polarization (DNP) in C13 NMR is proposed. This method also has applications to Glycolosis, as well as to all cancer cells.</p> <p><b>Methods/Materials</b><br/>DNP is a form of hyperpolarization used in NMR which spin polarizes both the nucleus and electrons of an atom. Spin polarizing the electrons leads to a violation of the Pauli Exclusion Principle, which causes Glutamine to lose its ability to react chemically with other molecules. The first step of Glutaminolysis, which triggers the entire process, is the conversion of Glutamine to Glutamate. However, since the hyperpolarized Glutamine can no longer react chemically, Glutamate cannot be produced, and Glutaminolysis is inhibited. In order to maximize the rate and effectiveness of the inhibition of Glutaminolysis, the optimal conditions for DNP to occur were determined through investigation of the effect of pH on the rate of magnetization transfer in the sample and the spin lattice relaxation time (T1).</p> <p><b>Results</b><br/>It was found experimentally that the pH is directly proportional to the rate of transfer of magnetization and inversely proportional to T1. Glutamine was also magnetized, and it was found that the magnetized Glutamine had a much higher pH, which allows for a faster transfer of magnetization in the sample, enhancing the effects of DNP and allowing for highly effective inhibition of Glutaminolysis.</p> <p><b>Conclusions/Discussion</b><br/>My new method ultimately provides effective inhibition of Glutaminolysis in tumor cells, thus cutting off the cells' energy supply and killing them. I also observed that the pH of a sample is directly proportional to the rate of transfer of magnetization and inversely proportional to T1 of that sample, and that when magnetized, the pH of Glutamine rose, allowing for effective inhibition of Glutaminolysis. My new method also applies to Glycolosis, the other main energy-producing pathway in tumor cells, as well as to all cancer cells in general.</p> |                                       |
| <b>Summary Statement</b><br>In this study, I propose a new method to inhibit Glutaminolysis using DNP in C13 NMR to ultimately kill tumor cells.  |                                       |
| <b>Help Received</b><br>My faculty mentor at school advised me on some of the technical aspects of my study.  |                                       |



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| <b>Name(s)</b><br><b>Eric R. Bryan</b>   | <b>Project Number</b><br><b>S0504</b> |
| <b>Project Title</b><br><b>Effect of Temperature + Electrode Size on Electric Production + Growth of Electric Production of Microbial Fuel Cells</b>   |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>The objective is to increase efficiency of a microbial fuel cell. It is predicted that increased temperature and surface area will increase the voltage the microbial fuel cell produces.<br><b>Methods/Materials</b><br>Nine microbial fuel cells (MFC) were built using plastic containers, carbon cloth and copper wire, using mud as the source of bacteria in the anode. Each MFC was placed in one of three different temperature environments and built with one of three electrode surface areas. Voltage was taken from these MFCs twice daily for five days, then the mud was refreshed and the experiment repeated for a total of 4 trials.<br><b>Results</b><br>The results showed that increased temperature increased the voltage, up to 27°C, and had no effect on the growth of the voltage over the five days. Increased surface area of the electrode increased the voltage but had no effect on the growth of the voltage. The combination of the variables had no effect on both the voltage and its growth rate. Based on the data from this experiment, for an optimized MFC, the fuel cell should be in an environment of 27°C, and have a large 40 cm <sup>2</sup> electrode.<br><b>Conclusions/Discussion</b><br>MFCs are devices that utilize the chemical reactions of anaerobic bacterial respiration to create electricity. The results supported my hypotheses and gave a good general range for temperature, and showed that maximizing electrode surface area improves efficiency. The data also showed that temperature had a larger effect on the voltage than the electrode size, which leads to the conclusion that improving the bacteria's environment is more effective than improving aspects of MFC design. Microbial fuel cell technology can be a very useful technology in the emerging renewable energy market. With its capability to process waste and power itself at the same time, an integrated MFC would be the perfect counterpart to a waste treatment facility. |                                       |
| <b>Summary Statement</b><br>This project is designed to improve the electrical production efficiency of a microbial fuel cell through the variation of temperature and electrode surface area.   |                                       |
| <b>Help Received</b><br>Parents provided financial aid   |                                       |



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| <b>Name(s)</b><br>Nikhil Buduma  | <b>Project Number</b><br><b>S0505</b> |
| <b>Project Title</b><br><b>PTX-Mediated Inhibition of Lymphocyte Trafficking into the Lungs:<br/>Considerations for an Improved Whooping Cough Vaccine</b>   |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>Whooping cough, a disease characterized by a paroxysmal cough, is caused by the bacterium <i>Bordetella pertussis</i>. The current study aims to demonstrate that the pertussis toxin (PTX), a major component of the current vaccine against whooping cough, impairs the activation of the adaptive immune system. Specifically, this study intends to show that PTX inhibits lymphocyte recruitment to the lungs by downregulating the expression of lymphocyte trafficking receptors (TRs).</p> <p><b>Methods/Materials</b><br/>Four Balb/c murine models of human respiratory infections were implemented: uninfected mice, mice infected by <i>B. pertussis</i>, mice infected by <i>B. pertussis</i> TOX6 (A mutant for the PTX gene), and mice infected by <i>B. parapertussis</i> (a close relative that overlaps 90% of the virulence factor genes but does not express PTX). Lung cross-sections were taken at 5 days, 10 days, and 25 days post infection (p.i.) and analyzed using standard HE staining and immunofluorescence staining for neutrophils, macrophages, B cells, and T cells. In addition, peripheral blood was analyzed for the expression of alpha4beta7, alpha4beta1, CD11a, and Psel-L TRs using flow cytometry. Similar flow cytometric analyses were performed in vitro on T cells co-cultured with lung dendritic cells from infected mice 5 days p.i.</p> <p><b>Results</b><br/>HE staining and immunofluorescence labeling of lung cross-sections revealed that the recruitment of adaptive immune cells, in particular B cells and T cells, was severely delayed during infection by <i>B. pertussis</i> compared to both control groups. In addition flow cytometry analysis revealed two trafficking receptor populations, alpha4beta7 and alpha4beta1, that were downregulated on memory T cells at 5 days p.i. during <i>B. pertussis</i> infection. The in vitro co-culture demonstrated similar results, though the differences in alpha4beta7 expression were not revealed as significant by statistical analyses.</p> <p><b>Conclusions/Discussion</b><br/>This study demonstrates that PTX delays the recruitment of adaptive immune cells to the lungs. This observed delay is likely to be mediated by a PTX-dependent downregulation of alpha4beta7 and alpha4beta1 TR expression, due at least partly to defective communication between resident dendritic cells and T cells. These results suggest that vaccine formulations should avoid the use of PTX because of its potential for preventing the infant's immune system from developing a sufficient defense.</p> |                                       |
| <b>Summary Statement</b><br>My project aims to demonstrate that the pertussis toxin, a major component of the current whooping cough vaccine, interferes with the proper functioning of the adaptive immune system.  |                                       |
| <b>Help Received</b><br>I conducted this research under the guidance of Dr. Tzvia Abramson at San Jose State University.   |                                       |



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| <b>Name(s)</b><br><b>Eric S. Chen</b>   | <b>Project Number</b><br><b>S0506</b> |
| <b>Project Title</b><br><b>MicroRNA: A New Way to Fight Pancreatic Cancer</b>   |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>My project is to evaluate microRNA (miRNA) as a new treatment for pancreatic cancer. I believe restoring tumor suppressor miRNAs in pancreatic cancer cells will make them less aggressive and more sensitive to chemotherapy.</p> <p><b>Methods/Materials</b><br/>I first examined the expression of tumor suppressor miRNA in pancreatic cancer cell line BxPC-3 by a method called Taqman Real-Time PCR assay. By using liposomes with the miRNAs in transfection, I increased the expression of the miRNAs in the pancreatic cancer cells. Additionally, I examined proliferation and sensitivity of BxPC-3 cells transfected with miR-148a to the chemotherapy drug Gemcitabine. I also performed Western blot analysis to study the downstream targets of miR-148a.</p> <p><b>Results</b><br/>My results showed that miR-148a was greatly reduced in the pancreatic cancer cell line BxPC-3. I also found that upon expression of miR-148a, cell proliferation is reduced, and that the cells were more sensitive to chemotherapy drug Gemcitabine because miR-148a controls apoptosis of cancer cells.</p> <p><b>Conclusions/Discussion</b><br/>My conclusion is that restoring the expression of tumor suppressor miRNAs in pancreatic cancer suppresses the tumorigenic traits and has the potential to help treatment of pancreatic cancer by making it more susceptible to chemotherapy.</p> |                                       |
| <b>Summary Statement</b><br>I try to find a new treatment for pancreatic cancer.  |                                       |
| <b>Help Received</b><br>Used lab equipment at UCSD under the supervision of Dr. Gen-Sheng Feng  |                                       |



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| <b>Name(s)</b><br>Sara N. D'Souza  | <b>Project Number</b><br><b>S0507</b> |
| <b>Project Title</b><br><b>New Mechanism and Neuronal Activation of Brain by Peripheral Leptin: Potential Applications in Obesity</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>Failures to maintain energy balance contributes largely to obesity. The body produces Leptin, a protein in proportion to food intake and stored adipose mass, and acts on brain to maintain energy homeostasis. General understanding about Leptin's action on brain is that it penetrates BBB. In this study, I hypothesize that CSF is an alternative route for Leptin to act on the brain. My research is to help understand the mechanism of leptin action which should be helpful in developing new drugs to treat obesity.</p> <p><b>Methods/Materials</b><br/>Method: Whole rat brains previously dosed with Leptin used from ongoing institutional research were acquired and 30 microm thick coronal tissue sections were collected in antifreeze solution. Pre-staining was done, and tissues were incubated with goat serum and Triton X. Between each step multiple PBS rinses were performed. Sections were incubated with a pSTAT3 antibody. The sections were incubated in a secondary antibody followed by incubation in ABC solution. Biotinylated tyramide and H2O2 were added followed by streptavidin FITC conjugate. Sections were rinsed, mounted, and images were acquired with confocal microscopy.<br/>Materials: Rat brains, Tissue slicer, Anti-freeze solution, NaOH, H<sub>2</sub>O(2), Glycine, SDS, Triton X-100, Goat serum, Rabbit anti pSTAT3 antibody, Biotinylated Anti-Rabbit IgG antibody, ABC complex, Biotinylated Tyramine, DAB, Nickel Sulfate, Streptavidin FITC conjugate, Glass Slides, Well Plate, Staining Mesh, Incubator, ProLong antifade, DAPI, and Confocal microscope</p> <p><b>Results</b><br/>Using pSTAT3 as a marker for Leptin activation and by constant repetition, extensive experimental conditions, I was able to see Leptin-neuronal expression throughout all ventricular regions of the brain, thus proving my hypothesis that the CSF is the pathway of Leptin entry and neuronal activation in the brain.</p> <p><b>Conclusions/Discussion</b><br/>Since neurons of the hypothalamus have Leptin receptors and the CSF has significant amounts of Leptin, I hypothesized that I would observe Leptin-neurons along the ventricular regions of the brain by following the pSTAT3 expression via immunohistochemistry.<br/>My research conclusively proved that leptin activates key neurons in the brain via the CSF pathway. This research has significant implications and will alter the focus for future Obesity studies. To the best of my knowledge this is the first report describing CSF as an alternate route of entry for mechanism of action of Leptin.</p> |                                       |
| <b>Summary Statement</b><br>This is a novel mechanism of leptin action in the brain using the Cerebrospinal fluid as an alternate route, which activates key neurons in the ventricular regions to maintain energy balance.  |                                       |
| <b>Help Received</b><br>Used laboratory facilities at Amylin Pharmaceuticals under the supervision of Dr. Guibao Gu (MD);  |                                       |



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| <b>Name(s)</b><br><b>Bianca N. De</b>  | <b>Project Number</b><br><b>S0508</b> |
| <b>Project Title</b><br><b>Modeling the Effects of Angiostatins and Mitotic Inhibitors on Vascularized Tumor Growth</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>This model attempts to examine the effects of both angiostatic inhibitors, also known as angiostatins, and mitotic inhibitors on the growth of various attributes of a tumor as it proceeds through vascularization, namely volume, number of live cells, and radius.</p> <p><b>Methods/Materials</b><br/>Scilab, a mathematical programming language similar to Matlab, was used to create a representation of the growth of a tumor. Generalized equations were created for the growth, and public-domain data was used to calibrate the equations to the growth of observed tumors. The avascular and necrotic stages of growth were modeled to create a more comprehensive picture of the progress of the tumor. However, these two stages had fixed growth patterns as no significant parameters were in place. The vascular stage was modeled with parameters created to represent the presence and strength of angiostatins and mitotic inhibitors in the tumor. The model was run with various values in place for the strength of the two medications and the results observed.</p> <p><b>Results</b><br/>Angiostatic inhibitors were shown to have a larger impact on the tumor growth than mitotic inhibitors. Angiostatic inhibitors had a particularly pronounced effect on vascularly-supplied cells, preventing their formation. Mitotic inhibitors limited the division of all cells without regard to the nutrient supply of the cell.</p> <p><b>Conclusions/Discussion</b><br/>Angiostatins were more effective than mitotic inhibitors because they prevented the creation of the faster-growing vascularly-supplied cells. The mitotic inhibitor reduces the rate of growth for all the cells, but still allowed the creation of the relatively aggressive cells. There are many practical applications of these results. The model can demonstrate the effects of various cancer medications on the growth of a tumor. During cancer treatment, the impact of a drug on a patient can be reliably predicted before the drug is ever administered. The fact that angiostatins are more effective than mitotic inhibitors is also of considerable interest. It means that during cancer treatment, angiostatic drugs will be of more use to prevent metastasis.</p> |                                       |
| <b>Summary Statement</b><br>Scilab was used to model a tumor and determine the comparative efficacies of mitotic inhibitors and angiostatins on the growth of a tumor; the angiostatin was found to be more effective.   |                                       |
| <b>Help Received</b>   |                                       |



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| <b>Name(s)</b><br><b>William Du</b>   | <b>Project Number</b><br><b>S0509</b> |
| <b>Project Title</b><br><b>Novel Insights into Ultraconservation in Human Genes</b>   |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>In my research, my goal was to determine if there were nucleotide sequences that are 100% identical between the human cDNA and other organisms. If so, I would determine what these sequences are responsible for and attempt to find ultraconserved sequences not properly annotated in the human genome.</p> <p><b>Methods/Materials</b><br/>Highly conserved regions between animals were located through a series of BLAST searches. Regions were then selected if they had a Poisson value below <math>1 \cdot 10^{-100}</math>. In one aspect of my research, DAVID, a gene annotation tool, was used to group the genes into clusters determined by function. In the second aspect, I searched for ultraconserved exonic regions that are in the predicted track of our genome but are not present in the known track. By using a second BLAST search, I discovered genes that are highly conserved between another organism's known track and the human's predicted track and should be taken into consideration in the next revision of the human genome.</p> <p><b>Results</b><br/>Overall, over 10,000 sequences were identified to be ultraconserved. Subsequently, genes containing these sequences are predominantly involved in essential central dogmatic function. In addition, several genes upon closer inspection appear to play a key role in development and tumor suppression. Finally, two genome incongruences were discovered in the human genome; due to the correlation between the function of human genes and other organism genes, there is a high chance that this section of DNA should be annotated into the human known track.</p> <p><b>Conclusions/Discussion</b><br/>The human, chicken, mouse, frog, platypus, and sea squirt last shared a common ancestor hundreds of millions of years ago. Genetic drift causes the nucleotide sequences of genes to change even if their protein sequences remain essentially identical. Regions displaying 100% nucleotide identity over long (greater than 100 base pairs) stretches are statistically unlikely and may indicate the presence of previously unknown genomic functions. This research provides insight into highly conserved nucleotide sequences across various genomes in order to identify the function of these regions, mainly central dogmatic functions, and misannotations in the human genome. The results of the experiment are the foundation of research in potential gene therapy of diseases and provide understanding of genes that are ultraconserved.</p> |                                       |
| <b>Summary Statement</b><br>In my research, I discovered ultraconserved sequences between human cDNA and other organisms in order to link them to their function and better annotate the human genome.  |                                       |
| <b>Help Received</b><br>Used lab equipment at UC Davis Genome Center under supervision of Dr. Ian Korf, Participant in UCD Young Scholars Program   |                                       |



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| <b>Name(s)</b><br><b>Brian L. Hie</b>   | <b>Project Number</b><br><b>S0510</b> |
| <b>Project Title</b><br><b>PI3-Kinase and STAT3 in Cancer</b>   |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>Two important causes of oncogenic transformation, the phosphoinositide 3-kinase (PI3K) pathway and the STAT3 transcription factor, have until recently been thought to be separate. New studies have suggested a link between PI3K and STAT3, mediated by the protein kinase BMX, part of the TEC kinase family of non-receptor tyrosine kinases. This link between two important signaling proteins may be exploited for cancer therapy.</p> <p><b>Methods/Materials</b><br/>This study first examined ten human cancer cell lines for differential phosphorylation of STAT3 after TEC inhibition. Phosphorylation of STAT3 and other important downstream targets of PI3K was analyzed by Western blot. This study also tested the effects of drug combination treatment on H1047R-transformed 10T1/2 cells through a cell viability assay.</p> <p><b>Results</b><br/>In the cell lines experiment, TEC kinase inhibition by LFM-A13 reduces STAT3 phosphorylation in some cell lines, a result consistent with the hypothesis that BMX bridges the PI3K-STAT3 link. However, cell lines with mutations in KRAS and EGFR show unchanged or even enhanced STAT3 phosphorylation with TEC kinase inhibition, suggesting that these cells bypass PI3K and directly phosphorylate STAT3 through mutated receptor tyrosine kinases. In the drug combinations experiment, the combination of Rapamycin and LFM-A13 showed a synergistic interaction, most likely through inhibition of parallel pathways that converge on a similar oncogenic phenotype.</p> <p><b>Conclusions/Discussion</b><br/>There is evidence for TEC kinase mediation of the PI3K-STAT3 link in human cell lines. This, however, is not universal and is more complicated than suggested by previous experimentation. TEC inhibitors in cancer therapy may be best suited in drug combinations. Further study will determine the full practical application of TEC kinase inhibition in treating PI3K-induced oncogenesis and other cancers.</p> |                                       |
| <b>Summary Statement</b><br>The project examined the cell signaling link between PI3-kinase and STAT3 in a variety of cell lines and as a potential target for cancer therapy.  |                                       |
| <b>Help Received</b><br>Jonathan Hart, PhD. provided instruction in the scientific background and laboratory techniques required for this project. He assisted with experimental design and was available for questions. The project was conducted at the lab of Peter K. Vogt, PhD. at the Scripps Research Institute.   |                                       |



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| <b>Name(s)</b><br><b>Teri Lambros</b>  | <b>Project Number</b><br><b>S0511</b> |
| <b>Project Title</b><br><b>The Anticancer Effects of Certain Dietary Supplements through the Inhibition of NF-kB</b>   |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>This work evaluated the anticancer properties of dietary substances such as curcumin and Si-Wu-Tang (SWT) by measuring the activity of NF-kB in human cell lines. Our hypothesis is that certain dietary substances such as curcumin and SWT lower the activity of NF-kB, thus have anticancer properties.</p> <p><b>Methods/Materials</b><br/>The human cell line HEK 298 was used. The cells were cultured and transfected with a plasmid that carried both the NF-kB and luciferase genes. The transfected cells were induced with TNF-alpha (except of the negative control group) and treated with curcumin or SWT. The activity of luciferase was measured using a commercial luciferase assay system. The groups were as follows: Group 1, non-induced non-treated cells (negative control). Group 2, cells induced with TNF-alpha and non- treated (positive control). Group 3, cells induced with TNF-alpha and treated with 40ug/ml curcumin. Group 4, cells induced with TNF-alpha and treated with a high concentration (256ug/ml) of SWT. Group 5, cells induced with TNF-alpha and treated with a low concentration (26ug/ml) of SWT. For each group N=6.</p> <p><b>Results</b><br/>The average luciferase activities were, 39.3, 73.8, 42.2, 43.5, 61.3 for the negative control, positive control, curcumin, high SWT concentration and low SWT concentration groups, respectively. The luciferase activity of the positive control group was significantly higher compared to the negative control, curcumin and high SWT concentration groups. Thus the activity of NF-kB was significantly lower in cells treated with curcumin or high SWT concentration.</p> <p><b>Conclusions/Discussion</b><br/>Cells induced with TNF-alpha only and non-treated showed high luciferase activity which translates to high NF-kB and carcinogenic activity, whereas cells treated with curcumin or high concentration of SWT even though were induced with TNF-alpha showed a significantly lower NF-kB activity compared to the positive control. These results indicate that curcumin and high concentration of SWT reduce the activity of NF-kB and have anticancer properties. We continue testing more dietary substances.</p> |                                       |
| <b>Summary Statement</b><br>We evaluated the anticancer activity of dietary substances such as curcumin and Si-Wu-Tang by measuring the activity of NF-kB in a human cell line.  |                                       |
| <b>Help Received</b><br>This work was done under the supervision and in the lab of Dr Ying Huang, College of Pharmacy Western University of Health Science.  |                                       |



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| <b>Name(s)</b><br>Gwendolyn G. Lee  | <b>Project Number</b><br><b>S0512</b> |
| <b>Project Title</b><br><b>Impact of vRNA and M1 Protein on the Structure and Budding Mechanism on the Influenza A Virus</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>The purpose of this research is to elucidate the structure and budding mechanisms of the influenza A virus using cryo-electron tomography. This project also aims to compare budding in mutant strains with that in wild-type viruses in order to highlight budding defects that may serve as potential targets for future antiviral therapy. With this information, vaccines and medications that target specific components of the budding process, thereby inhibiting viral proliferation, can be developed.</p> <p><b>Methods/Materials</b><br/>The influenza virus samples were prepared using the influenza A/Udorn/72 (H3N2) strain, as well as the influenza A/WSN/33 (H0N1) strain. The budding mutant was created using the M1[R101A] mutation in the WSN strain. The samples were maintained in Madin-Darby canine kidney (MDCK) cells. Samples were flash-frozen in preparation for cryo-electron microscopy. Cryo-electron tomography (cryoET) was used to analyze these virus samples.</p> <p><b>Results</b><br/>The tomographic reconstructions of the influenza A/Udorn/72 (H3N2) strain display three major morphologies: filamentous particles, spherical particles, and chains of particles resembling beads on a string. Some particles were completely devoid of vRNA. The aberrant chains of particles were linked by viral membranes, forming junction points, or by interlocked surface proteins. The reconstructions of the influenza A/WSN/33 mutant strain reveal a major aberration, in which the middle section of certain particles collapsed, forming only a thin layer.</p> <p><b>Conclusions/Discussion</b><br/>The lack of vRNA in certain virions suggest that RNPs are not required in the budding process. The chains of particles indicate budding "hot spots" that are particularly conducive to budding. These locations can serve as targets for antiviral drugs. The M1 protein appears to play a role in the structure of the virus. Furthermore, there appears to be a correlation between the presence of M1 and that of surface proteins.</p> |                                       |
| <b>Summary Statement</b><br>In order to elucidate the structure and budding mechanism of the influenza A virus, samples of wild type and mutant influenza virus were cultured, imaged using cryo-electron microscopy, and analyzed using tomography reconstruction technique  |                                       |
| <b>Help Received</b><br>Dr. Hong Zhou allowed me to use laboratory equipment and taught me how to use computer reconstruction software. Dr. Nayak allowed me to use his laboratory space and equipment.   |                                       |



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| <b>Name(s)</b><br><b>Kenneth Y. Lee</b>   | <b>Project Number</b><br><b>S0513</b> |
| <b>Project Title</b><br><b>The Role of Testosterone in Hepatocyte Apoptosis in High Fat Diet-Induced Non-alcoholic Fatty Liver Disease: Year 2</b>  |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>The objective of this study was to discover whether or not hepatocyte apoptosis due to non-alcoholic fatty liver disease (NAFLD) is mitochondria-dependent, and thus follows the intrinsic apoptotic pathway. Furthermore, this study was intended to explore testosterone's potential ability to prevent apoptosis by inhibiting mitochondrial release of DIABLO in the intrinsic apoptotic pathway.<br><b>Methods/Materials</b><br>From a previous separate study, male rats were randomly placed into four groups: intact rats on regular chow diet (RCD), intact rats on HFD, castrated rats on HFD, and castrated rats on HFD with testosterone replacement. The rats were fed ad libitum for 15 weeks, sacrificed, and liver tissue was collected and fixed with formalin. These samples were used for the apoptosis detection in this study. Western blot was used to evaluate levels of Smac/DIABLO (23 kDa) in hepatocyte cytosol.<br><b>Results</b><br>Western blot results between the intact groups showed that HFD-induced NAFLD resulted in significantly greater mitochondrial release of DIABLO into hepatocyte cytosol. The comparison between castrated groups showed a significantly reduced concentration of DIABLO in hepatocyte cytosol after treatment with testosterone.<br><b>Conclusions/Discussion</b><br>It was concluded that one main mechanism behind testosterone's protective effect in the liver is inhibiting release of DIABLO into hepatocyte cytosol. This study thus further develops the HFD-induced NAFLD model, and may forge a path toward eventually attenuating NAFLD. |                                       |
| <b>Summary Statement</b><br>My study identified one main way that testosterone prevents hepatocyte apoptosis in the liver due to non-alcoholic fatty liver disease.   |                                       |
| <b>Help Received</b><br>Used lab equipment at the Los Angeles Biomedical Research Institute under the supervision of Dr. Yue Jia and Dr. Ronald Swerdloff   |                                       |



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| <b>Name(s)</b><br><b>Meredith Paloma Lehmann; Virgil Anderson Woods</b>   | <b>Project Number</b><br><b>S0514</b> |
| <b>Project Title</b><br><b>In vivo Drug Assembly</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>Fragment-based drug design is used to construct single high affinity drugs from many small, low affinity components. A pervasive problem with such drugs is binding to receptors at disease sites and in other healthy parts of the body with deleterious side effects. We design drugs comprised of similar small components that bind to receptors at the disease site and proteins present only at the target. The approach is illustrated for Rheumatoid Arthritis (RA). Conventional drugs like Enbrel inhibit TNF receptors in the diseased joints and in healthy parts of the body. Type II collagen is present only in joints, save for trace amounts in the eyes and ears. Our redesign of Enbrel binds to both TNF receptors and to Type II collagen, resulting in unprecedented precision in targeting desired drug action to joints.</p> <p><b>Methods/Materials</b><br/>We performed a number of binding affinity calculations. For a two fragment drug like Enbrel, we calculated the percentage of each fragment that will bind to a receptor and the percentage of the assembled drug that will bind to the disease-area-localized assembly protein and to the receptor at chemical equilibrium. We used this information to compute the percentage of bound receptors given specified affinities and drug and organizer molecule concentrations, permitting the calculation of the ratio of bound receptors in the target area to bound receptors outside it. We used the public domain program Autodoc to build molecular models for Enbrel, Type II collagen alpha helices, and TNF receptors and set flexible residues for Type II collagen at the ends where it tends to be more flexible. We then used the public domain program Vina to calculate the affinity of Enbrel alone to a TNF receptor and of the alpha helix/Enbrel molecule to both local Type II collagen and a nearby TNF receptor. We took the resulting affinities and plugged them into our earlier calculations to calculate the ratio of bound receptors inside to bound receptors outside the target region.</p> <p><b>Results</b><br/>We achieved ratios in above 10:1 with many in excess of 100:1, far above the ratios on the order of 1:1 attained by current drugs.</p> <p><b>Conclusions/Discussion</b><br/>Our results demonstrate the feasibility of designing drugs that confine their actions only to diseased areas in the body. We will use drug simulation programs next to predict and evaluate drug interactions within the body in concert with information from drug fragment libraries.</p> |                                       |
| <b>Summary Statement</b><br>We design drugs that target only diseased parts of the body by forcing them to bind to proteins present only at the target.   |                                       |
| <b>Help Received</b><br>Our parents helped by proofreading the paper and with the printing of our poster.   |                                       |



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| <b>Name(s)</b><br><b>Enrique Lorenzo; Vanessa Mendoza; Francine Rubio</b>  | <b>Project Number</b><br><b>S0516</b> |
| <b>Project Title</b><br><b>Leaf Chromatography</b>   |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>With this experiment, we wanted to find the effects of temperature on pigmentation. To obtain adequate results and ultimately conduct the most conclusive chromatography test, we first tested each element of the experiment itself to find the best materials to utilize when conducting our final experiment. Once we conducted five experiments to give us the best type of paper, chemical, method, time, and color leaf to use, we finally took two leaves (one at 6.67 degrees Celsius and the other heated for 20 s) and conducted our experiment.</p> <p><b>Methods/Materials</b><br/>For each experiment, we took the leaves and crushed them into a chemical in a beaker and left them alone for two hours so that the solvent could extract the pigment. We then hung a strip of paper from a pencil sitting atop the beaker so that only the tip of the paper was in the chemical at the bottom of the beaker to allow the mobile phase (the pigments running up the paper) to begin. Afterward, we measured the distance between each pigment and observed their vivid vibrancy to record our results.</p> <p><b>Results</b><br/>We found that computer paper was the best to use because of the cellulose fibers being more tight-knit, the pure acetone was most effective because it yielded more distinct lines of different pigments, the green colored leaves yielded more differentiation as well, the coin method was not as effective as using the solvent during the stationary phase, recently-picked leaves held better results during the mobile phase, and finally colder temperatures yielded a wider variance and increased pigmentation in leaves than warmer temperatures did.</p> <p><b>Conclusions/Discussion</b><br/>This, we discovered, was very applicable to real life concepts because in the East Coast, farther from the equator, color change in leaves is more vibrant because of colder temperatures. Chromatography can also be used in crime scene investigations to match pigmentation in lipstick or other substances. We were limited in our materials and our time in the lab, but we still conducted six successful experiments. We would like to expand our experiment to find if pollution has an effect on pigmentation or even find why artificial pigments fade in the sun and if that is related to temperature.</p> |                                       |
| <b>Summary Statement</b><br>It is about the effects of temperature on the vibrancy and differentiation of pigmentation of leaves when a chromatography test is conducted.  |                                       |
| <b>Help Received</b><br>Enrique's dad helped to paint the board and put magnets on the back to ensure that they can stand atop each other and Ms. Pearce helped us get ahold of the chemicals we needed.   |                                       |



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| <b>Name(s)</b><br><b>Haley A. Lu</b>  | <b>Project Number</b><br><b>S0517</b> |
| <b>Project Title</b><br><b>AsparagYES or AsparagNO?</b>   |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>The objective is to determine the pattern of inheritance of the particular gene that allows one to smell an unpleasant odor in his or her urine after the consumption of asparagus and to use the Hardy Weinberg equation in order to find the values $p$ , $q$ , $p^2$ , $q^2$ , and $2pq$ . $P$ and $q$ are the allele frequencies for each allele. $P^2$ and $q^2$ are the frequency of homozygotes, where $p^2$ is the frequency of the dominant homozygotes and $q^2$ is the frequency of recessive homozygotes. The frequency of heterozygotes is represented by $2pq$ . This information will provide the knowledge required to ascertain the alleles of each tested individual to determine the genotypes.<br><b>Methods/Materials</b><br>I interviewed different families and constructed pedigrees of 217 individuals to find the pattern of inheritance. I wrote a letter and gave it to those who participated in my experiment to request their participation, inform them briefly on my project topic. They were told not to participate if they were allergic to asparagus. 217 subjects, male and female, from ages 10-75 answered whether or not they were able to smell an odor in their urine after eating asparagus. After collecting data, research and completing many family pedigrees, I determined the pattern of inheritance of the gene. I inserted my data into the Hardy Weinberg equation and found the $q$ , $p$ , $q^2$ , $p^2$ , and $2pq$ values and the number of individuals for each genotype.<br><b>Results</b><br>I learned that the pattern of inheritance for this trait is autosomal dominant. By finding what $q^2$ (.32) is first, I completed the Hardy Weinberg equation. I determined that out of the 217 individuals that I tested, 70 of them were homozygous recessive, 42 individuals were homozygous dominant, and 105 were heterozygous.<br><b>Conclusions/Discussion</b><br>I concluded that the pattern of inheritance for the gene that allows a person to detect an odor in his or her urine after eating asparagus is autosomal dominant. Using the pedigrees and coupling that with the Hardy Weinberg equation, the resulting genotypes were discovered. The data suggests the ability of a person to detect an odor in his or her urine is a result of a genetic pattern passed down from his or her biological parents. |                                       |
| <b>Summary Statement</b><br>Using pedigrees and the Hardy Weinberg equation, I determined the pattern of inheritance of the gene that allows one to be able to detect an odor in one's urine after eating asparagus and the genotypes of the tested individuals.  |                                       |
| <b>Help Received</b><br>217 individuals volunteered to respond to the Yes or No question.   |                                       |



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| <b>Name(s)</b><br><b>Tracy Ly</b>   | <b>Project Number</b><br><b>S0518</b> |
| <b>Project Title</b><br><b>Nanohydrogel for Treating Cancer by Efficient Delivery of Resveratrol</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>The main goal is to improve the water solubility of resveratrol to prevent it from being metabolized too quickly. By polymerizing resveratrol with polyethylene glycol to make a hydrogel, it is hypothesized that the aqueous solubility of resveratrol can be increased. Also, by tuning the polymerization time, it is hypothesized that the particle size can be modulated, which affects the route of how cells can internalize the nanoparticles. The hydrogel consisting of resveratrol is hypothesized to still exhibit cytotoxic activities on different cancer cell lines.</p> <p><b>Methods/Materials</b><br/>Biosafety cabinet, Incubator, Pipettors and pipette tips, HeLa cell line, T98G cell line, Resveratrol, Acrylated resveratrol, Polyethylene glycol diacrylate (PEG), Irgacure, DMEM, Ethanol, MTT Assay Reagent, UV-vis spectrophotometer, Microscope, Eppendorf tubes, Dynamic Light scattering machine, centrifuge</p> <p><b>Results</b><br/>Aqueous solubility was improved at 1:10 molar ratio of acrylated resveratrol to PEG. This makes it easier to dissolve resveratrol in water to increase the cytotoxic effects towards the cells. The results of the MTT assay which determines cell viability show at least a 70% decrease in cell viability at all concentrations lower than 100 uM after 24 hours of dosing. The most significant decrease was seen in 4 uM in cell line T98G. At this concentration, resveratrol had 90% cell viability versus the hydrogel which had viability of less than 10%. The optimal concentration of PEG to acrylated resveratrol was found to be 1:10. By using DLS, it was observed that 1:1 and 1:5 samples were not uniform in size due to its partial solubility which thus forms aggregates.</p> <p><b>Conclusions/Discussion</b><br/>The goal of making a hydrogel was to help solubilize resveratrol in water to improve cellular uptake and expand the bioavailability. At a 1:10 concentration of resveratrol to PEG, we can observe that it becomes completely soluble compared with 1:1 or 1:5. At lower molar ratios of resveratrol to PEG, it becomes only partly soluble which decreases cellular uptake efficiency. At increasing concentrations of resveratrol and the hydrogel, all cells seem to be non-viable. However at lower concentrations, the hydrogel has increased the cytotoxic effects at least seven-fold. All in all, the nanohydrogel increased the EPR Effect for resveratrol in the cancer cells with an effect that tested cell lines exhibit higher cytotoxicity as compared to treating with free resveratrol.</p> |                                       |
| <b>Summary Statement</b><br>Creating a nanohydrogel to optimize the delivery of resveratrol, an anti-tumor agent, for treating cancer.  |                                       |
| <b>Help Received</b><br>I used professional lab equipment under the supervision of Dr. Young Jik Kwon at UCI.   |                                       |



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| <b>Name(s)</b><br>Natalie Ng   | <b>Project Number</b><br><b>S0519</b> |
| <b>Project Title</b><br><b>Development of a Novel Biomarker Discovery Tool to Identify Clinical Signatures from Deconvoluted Expressions</b>   |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>Tissue heterogeneity is a major confounding factor in microarray-based gene expression analysis. Gene expression deconvolution is an innovative method to overcome this problem by decomposing the global gene expression into pure cell expression subprofiles. Differential analysis can then be performed on the deconvoluted expressions to identify disease-related genes that may otherwise be undetectable. The primary objectives of this project are the development of a novel biomarker discovery tool to be used in conjunction with statistical gene expression deconvolution to identify prognostic signatures for breast cancer patients. This project has two major components: (1) development of a novel biomarker discovery tool and, (2) development and application of a statistical deconvolution method to identify gene signatures which can be used as prognostic predictors of disease outcomes.</p> <p><b>Methods/Materials</b><br/>Microarrays of Affymetrix Human Genome U133A Array platform were used in the cell type biomarker discovery workflow. Cell type specific biomarkers were identified using statistical significance tests (ANOVA and TukeyHSD) and up-regulation ratio computation. Microarray dataset GSE2034, from a published breast cancer study, was used to demonstrate the application of gene expression deconvolution to identify clinical signatures. An automated statistical gene expression deconvolution procedure was developed based on a two-step iterative algorithm implemented in Matlab.</p> <p><b>Results</b><br/>Biomarkers of immune, stromal, and tumor cell types have been identified and verified. Correlations of cell type biomarkers were excellent in the training and validation sets. The biomarkers provided biological identification of the deconvoluted expressions to their corresponding cell types. Using statistical gene expression deconvolution, gene signatures that can discriminate breast cancer patients according to clinical outcomes (relapse versus relapse-free) have been identified.</p> <p><b>Conclusions/Discussion</b><br/>A novel biomarker discovery tool and an automated statistical gene expression deconvolution procedure were developed to analyze global gene expressions of breast tumor samples to identify prognostic gene signatures predictive of clinical outcomes. Functional analysis of prognostic signatures provided insights into molecular pathways associated with tumor progression and metastasis.</p> |                                       |
| <b>Summary Statement</b><br>I designed and developed a novel biomarker discovery tool and an automated statistical gene expression deconvolution protocol to analyze breast tumor samples and identify prognostic gene signatures predictive of clinical outcomes.   |                                       |
| <b>Help Received</b><br>My mentor, Dr. Neta Zuckerman (Stanford University, City of Hope Hospital) provided guidance and feedback. Professor Peter Lee (Stanford University, City of Hope Hospital) provided a summer internship position.   |                                       |



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| <b>Name(s)</b><br><b>Preethi Padmanaban</b>  | <b>Project Number</b><br><b>S0520</b> |
| <b>Project Title</b><br><b>GM Plants: Manipulating Extracellular pH to Reduce Antibiotic Transformation Frequency in E. coli</b>   |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>Genetically modified plants usually contain a gene of interest as well as an antibiotic marker gene, which can be transferred to soil bacteria, causing a myriad of problems. The objective of this project is to determine a soil pH that would reduce genetic transformation while being conducive to plant growth.<br><b>Methods/Materials</b><br>E. coli was grown in an LB broth culture, then made competent using solutions of MgCl <sub>2</sub> and CaCl <sub>2</sub> . Tris-HCl buffer solutions of pH 5-9 were added to agar plates and LB broth. The plasmid used was p-ARAr, a vector with RFP and ampicillin resistance. The E. coli was transformed and grown for 40 hours in each respective pH. Additional trials with ampicillin, arabinose, and the buffer were plated. Smaller intervals of pH and dilutions were later tested to narrow down results.<br><b>Results</b><br>The E. coli grown naturally in each pH showed the most colony-forming units at pH 7. The bacteria transformed and grown at pH 8 had the highest number of transformants per plate. Those at pH 5, 6, and 9 had considerably fewer successful colonies. The bacteria grown at a 1:100 dilution had a higher frequency of transformed bacteria across pH levels, as compared to the undiluted bacteria.<br><b>Conclusions/Discussion</b><br>The optimal pH for transformation in E. coli is between pH 8.25 and pH 8.5. This range would thus be the range to avoid in soil. As transformation was observed most frequently at a lower population density, it is likely that soils with a higher concentration of bacteria and microbes would have a lower risk of gene transfer. Disregarding the extreme pH levels of 5 and 9, the targeted soil pH would be pH 6, as this would both reduce transformation frequency and allow GM plants to grow normally. |                                       |
| <b>Summary Statement</b><br>E. coli was transformed and grown at a range of pH levels to determine the pH value that is least conducive to genetic transformation, simulating the scenario of GM plants transferring resistance genes to environmental bacteria and plants.  |                                       |
| <b>Help Received</b><br>Used high school lab equipment under guidance of Ms. Huong Tran; Received reagents, plasmid, and bacteria from Dr. Katy Korsmeyer of SCCBEP.   |                                       |



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| <b>Name(s)</b><br><b>Jiho Park</b>   | <b>Project Number</b><br><b>S0521</b> |
| <b>Project Title</b><br><b>Modeling and Molecular Dynamics Simulations of Membrane-Bound Aromatase Reveal Novel Druggable Sites</b>  |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>The objectives of the project are to: 1) Develop a new method of modeling full-length membrane-bound proteins systems for the application of molecular dynamics; 2) Use molecular dynamics and computational solvent mapping for the identification of novel druggable sites; 3) Identify the pharmacophores of inhibitors targeting these sites, and 4) Compare the quality of analysis derived from molecular dynamics simulation over analysis derived from the crystal structure.<br><b>Methods/Materials</b><br>The system (consisting of the protein, heterogeneous membrane, and waterbox) was constructed using CHARMM-GUI and Maestro. NAMD 2.7b on the SDSC Trestles and TACC Ranger supercomputers ran minimization and equilibration of the system, as well as 250 nanoseconds of free molecular dynamics. The resulting data was analyzed and visualized using VMD, UCSF Chimera, and Tcl scripts. FTMap performed computational solvent mapping, and DelphiElec computed electrostatics.<br><b>Results</b><br>Data from the molecular dynamics simulation was highly consistent with experimental data, validating the novel procedure used to build the model and process it for molecular dynamics. In addition, computational solvent mapping discovered two novel druggable sites for next-generation aromatase inhibitors to target - the heme proximal cavity and the active site channel. Furthermore, the overlapping of organic solvent molecules revealed a general pharmacophore for new inhibitors targeting these sites. The comparison of the ensemble-averaged electrostatics with the crystal structure electrostatics also pointed out flaws of the current theory of a higher-order aromatase structure, which was based on analysis of the crystal structure alone.<br><b>Conclusions/Discussion</b><br>The method used in this project in to construct a system of full-length membrane-bound protein was validated with experimental data, and is much more efficient than previously used methods. This method can be used as a precedent for the molecular dynamics of other membrane-bound proteins of interest. Furthermore, the use of molecular dynamics for the discovery of novel druggable sites was validated with the identification of the heme proximal cavity and the active site channel. The project also indicated the flaws of using the crystal structure for making conclusions, as evidenced in the example of aromatase oligomerization and electrostatics. |                                       |
| <b>Summary Statement</b><br>I developed a novel method of preparing membrane-bound proteins for molecular dynamics and applied to to aromatase to discover new binding sites that future inhibitors can target to treat estrogen-dependent breast cancer.  |                                       |
| <b>Help Received</b><br>Dr. Rommie Amaro mentored my project and allowed me to access the computational resources available to the Amaro Lab. Additionally, she and Dr. Luke Czapla answered any questions I had, gave me advice, suggested various things, helped me with programs, and edited my report.   |                                       |



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| <b>Name(s)</b><br><b>Christiane H. Pham</b>   | <b>Project Number</b><br><b>S0522</b> |
| <b>Project Title</b><br><b>It Runs in My Blood! The Relationship between Malaria Resistance and the Duffy Antigen</b>   |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>The main purpose of the project is to determine the nature of malaria resistance and analyze how the differences in the gene affect it. The gene is called the Duffy antigen/chemokine receptor (DARC), and its mutation has been attributed to resistance of the virulent disease, malaria. By figuring where the mutation occurs, better methods can be employed in order to combat this deadly disease.</p> <p><b>Methods/Materials</b><br/>Phylogenetic trees are generated using a computer program called ClustalX and FigTree using 40 DNA sequence samples in order to display using a graphic if any obvious differences are apparent. The DNA samples used were gathered from NCBI and included: 27 Homo sapiens, three Pongo pygmaeus, three Gorilla gorilla, one Pan paniscus, and six Pan troglodytes. The trees generated also show the relationship between Homo sapiens (humans) and other non-human primate DNA sequences in order to find the specific gene and see if these two share the same type of mutation.</p> <p><b>Results</b><br/>Obvious differences were observed in the phylogenetic trees that were generated. Evolutionary wise, all samples should line up in the relative same area on the tree, however some samples were outliers, suggesting there is a mutation, specifically, the target Duffy mutation. In a tree that compared all samples (human and non-human), the outliers in the human tree and non-human tree both shared differences, indicating the mutation could be a shared mutation.</p> <p><b>Conclusions/Discussion</b><br/>Malaria is an extremely dangerous disease caused by the parasite, Plasmodium falciparum or Plasmodium vivax. The Duffy antigen/chemokine receptor (DARC) is a binding element on the surface of the blood cell, and it is what the Plasmodium parasite uses to attach itself onto the red blood cell. Studies have shown that people that are lacking or have a mutation of the Duffy antigen do not have the components in order for Plasmodium to attach and propagate, allowing them to be resistant to malaria.</p> |                                       |
| <b>Summary Statement</b><br>Using phylogenetic trees to view the relationship of Duffy antigen mutations and malaria resistance in various species.   |                                       |
| <b>Help Received</b><br>Angela Vu for project board design assistance; Dr. Jay Vavra for project guidance; Jennifer Black for hypothesis assistance; Robert Seid and Amanda Lee for general research assistance; Megan Morikawa for suggestion on what alignment program should be used.  |                                       |



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| <b>Name(s)</b><br><b>Roxana Rodriguez</b>   | <b>Project Number</b><br><b>S0523</b> |
| <b>Project Title</b><br><b>Milk in Your Tea? The Effect of Milk on Green Tea Flavonoids</b>   |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>The objective is to determine if milk affects green tea flavonoid concentration, and if so, which milk causes the most change in amount of flavonoids. Flavonoids, which are abundant in green tea, are capable of antioxidant activities.<br><b>Methods/Materials</b><br>Three samples of tea were prepared; green tea, green tea with skim milk, and green tea with whole milk. Ten milliliters of each sample were then centrifuged. The supernatant was then taken from each sample and an aluminum chloride colorimetric assay was performed on each. Percent transmittance of color was then measured quantitatively using a spectrophotometer for every group. Results were measured at 510 nanometers and recorded. The entire procedure was repeated for every trial conducted.<br><b>Results</b><br>Averages of percent transmittance were taken from the total trials conducted. Green tea had an average transmittance of 18.46, skim milk and green tea had 17.06 percent transmittance, and whole milk with green tea had 42.975 percent transmittance. The solution with a higher concentration of flavonoids will have a lower transmittance, as the green tea and green tea with skim milk had. Whole milk with green tea had the highest percent transmittance, meaning it contained the least amount of flavonoids.<br><b>Conclusions/Discussion</b><br>The milk which had the most effect on flavonoid concentration was whole milk, while skim milk had concentrations of flavonoids similar to green tea. This could be correlated with the amount of fat found in each milk. Recent research has shown that because milk caseins can affect flavonoid concentration in tea, the antioxidant capabilities of tea within humans can be affected. The results of the trials suggest that whole milk should be evaded in tea if these claims are true. |                                       |
| <b>Summary Statement</b><br>My project seeks to determine which kind of milk, when added to green tea, will affect the flavonoid concentration of the tea.  |                                       |
| <b>Help Received</b><br>Parents bought supplies, Ms. Ramirez de la Cruz supervised and gave advice, Samantha Leon helped design board and provided research materials.  |                                       |



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| <b>Name(s)</b><br><b>Brooke J. Rothschild-Mancinelli</b>   | <b>Project Number</b><br><b>S0524</b> |
| <b>Project Title</b><br><b>The Effect of Natural Solar UV Radiation on the G1 Checkpoint in S. pombe</b>   |                                       |
| <b>Objectives/Goals</b><br>The purpose of my project is to test whether natural solar UV radiation is energetic enough to damage DNA to the point where cell cycle checkpoints are required.   |                                       |
| <b>Abstract</b><br><b>Methods/Materials</b><br>I took advantage of the fact that caffeine is known to override cell cycle checkpoints, allowing cells to divide with damaged DNA. If the damage is great enough, death will occur. I grew the fission yeast <i>Schizosaccharomyces pombe</i> in liquid YES medium. I then exposed diluted cultures cells to natural solar UV radiation in Whirlpack bags (control dark, control light, 10 mM caffeine light, 10 mM caffeine dark) to allow damage the DNA in the yeast cells. I took 10 $\mu$ L subsamples throughout the day and plated them on agar. I incubated the plates for two days at 36°C and saw that there was a gradation in the presence of colonies throughout the day. No growth represented the cells that died because the caffeine overrode their checkpoints when the DNA was damaged. I did this on Aug 27, 2011. To model annual variation in solar UV damage, I took light readings at different times of the year and compared them to my readings taken during the survival experiments. |                                       |
| <b>Results</b><br>My results showed that different amounts of UV do have an effect on the cell growth, as shown clearly with the caffeine-treated cells.   |                                       |
| <b>Conclusions/Discussion</b><br>From this I conclude that natural solar UV radiation is strong enough to damage the DNA and the checkpoints are needed to make sure that DNA is not replicated when it is damaged.  |                                       |
| <b>Summary Statement</b><br>To see if natural solar UV radiation is sufficient to damage DNA to the point where cell cycle checkpoints are crucial in cellular division.   |                                       |
| <b>Help Received</b><br>Mother helped come come up with experimental design and proofread  |                                       |



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| <b>Name(s)</b><br><b>Brittany R. Salyers</b>   | <b>Project Number</b><br><b>S0526</b> |
| <b>Project Title</b><br><b>Got Protein?</b>  |                                       |
| <b>Objectives/Goals</b><br>To measure the amount of protein in commercial milk products:cow, goat, soy, almond, rice, and coconut.   |                                       |
| <b>Abstract</b><br><b>Methods/Materials</b><br>Preparation and analysis:<br>1. If necessary, dilute each milk product with a volumetric flask (i.e. 1:100) 2. Transfer 100 ul each milk product or its dilution into labeled tubes 3. Prepare a #0# test tube or blank (only buffer) used as a reference to zero the Spec 20 at 595nm. 4. Add 6.0 mL of Coomassie Brilliant Blue reagent into each test tube (n=3). 5. After Bradford reagent added, wait 5 min and read absorbance. 6. Calculate mean $\pm$ SD (n=3) of absorbance values for each milk product and record them in data table. 7. Use equation from standard curve to determine protein in grams.<br><b>Materials:</b><br>1. 8 milk products (four plant and four animal); 2. Coomassie Brilliant Blue G 250 dye; 3. BSA (Bovine Serum Albumin); 4. Spectrophotometer 20; 5. cuvettes or test tubes (for Spec 20); 6. micropipettors and tips; 7. vortex mixer; 8. test tubes (for milk dilutions); 9. test tube rack; 10. 1 standard timer; 11. Permanent Lab Markers; 12. Lab book (i.e. Composition book); 13. 0.1 M KH <sub>2</sub> PO <sub>4</sub> (Potassium Dihydrogen Phosphate); 14. 0.1 M NaOH (sodium hydroxide); 15. volumetric flasks (i.e. 10mL, 100 mL, and 1L); 16.deionized water. |                                       |
| <b>Results</b><br>Chocolate and skim milk,from cows, had similar measured protein content 5.4 vs 6.3g protein/8oz. Of 3 plant-derived milk products, only soy had a measured protein content similar to animals.<br>Other plant derived products had negligible protein content  |                                       |
| <b>Conclusions/Discussion</b><br>The general trend of results was: experimentally determined values were lower than values found on assorted product labels; 0.2 g/8 oz serving,and 1.0 g/8 oz respectively.The 83% between these values represents the percent that my values differed from the label.<br><br>Conclusion: The results support my hypothesis; bovine milk has similar protein content as other animal derived milk products. Of the 3 plant-derived milk products tested, only soy had a measured protein content similar to animals. Other plant derived products (rice and coconut) had negligible protein content. Plant derived milks don't contain a complete protein and #can't be used as infant formula# because they won't support health and normal growth.  |                                       |
| <b>Summary Statement</b><br>Measure the amount of protein in the top four plant and top four animal milk products in the United States.  |                                       |
| <b>Help Received</b><br>Neighbor provided posterboard, Dr. Malhotra provided many of my materials and a lab, my Dad assisted (when needed) in the lab and obtained Coomassie Brilliant Blue reagent for experiment, BSA, etc; my Mom proof read most of my papers; my sister gave me support; and my Uncle at AAA chemicals gave me  |                                       |



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| <b>Name(s)</b><br><b>Manita Singh</b>   | <b>Project Number</b><br><b>S0528</b> |
| <b>Project Title</b><br><b>Effects of Polycaprolactone and UV Treated Poly (methyl methacrylate) Electrospun Fibers on Stem Cell Differentiation</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>The use of polymers to construct 3D biomimetic scaffolds resembling the extracellular matrix (ECM) possesses great potential in tissue engineering. The goal of this study is to engineer 3D electrospun scaffolds of both UV treated Poly (methyl methacrylate) (PMMA) and unmodified Polycaprolactone (PCL) that can successfully induce Dental Pulp Stem Cells to differentiate into the osteogenic lineage to create regenerative bone tissue in vitro.</p> <p><b>Methods/Materials</b><br/>Electrospinning is a process that involves the use of an electrical voltage to generate scaffold fibers on the nanoscale from polymer solutions. Two FDA approved polymers, PMMA and PCL, were used in this study. The effect of fiber diameter was tested by producing 6 um, 1 um, and nanofibers of PMMA. The effect of fiber orientation was tested by producing random oriented fibers and parallel oriented fibers. PMMA fibers were further modified by UV Plasma treatment, in order to increase the surface hydrophilicity of the scaffold.</p> <p><b>Results</b><br/>Confocal microscopy images reveal the success of UV plasma treatment in enhancing the adhesive properties of the PMMA scaffolds, allowing the DPSCs to proliferate along the electrospun fibers. Images of the vinculin stained cells suggest that the interaction between the cell cytoskeleton and the scaffold surface played a pivotal role in morphologically inducing DPSC differentiation in vitro. Atomic Force Microscopy analysis indicates a relationship between cell-cell and cell-surface scaffold hardness. Energy dispersive X-ray analysis, scanning electron microscopy, and mercury lamp images reveal that UV-treated PMMA nanofibers and PCL fibers of random orientation were the most successful scaffolds to induce DPSC differentiation and biomineralization.</p> <p><b>Conclusions/Discussion</b><br/>This study has identified the optimal PMMA and PCL electrospun scaffold characteristics for the morphological induction of DPSC differentiation into osteoblasts without the aid of a chemical inducer. This study also suggests a strong relationship between focal adhesion sites and stem cell differentiation, in addition to establishing UV Plasma treatment as a successful method to induce scaffold hydrophilicity and enhance scaffold adhesiveness. These polymeric scaffolds are the future of tissue engineering.</p> |                                       |
| <b>Summary Statement</b><br>The goal of this study is to engineer 3D electrospun scaffolds of both UV treated Poly (methyl methacrylate) (PMMA) and unmodified Polycaprolactone (PCL) that can successfully induce Dental Pulp Stem Cells to differentiate into osteoblasts.  |                                       |
| <b>Help Received</b><br>This study was conducted at Stony Brook University's Garcia Materials Science and Engineering Center, under the supervision of Dr. Vladimir Jurukovski.   |                                       |



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| <b>Name(s)</b><br>Aryo Sorayya  | <b>Project Number</b><br><b>S0529</b> |
| <b>Project Title</b><br><b>Overcoming the Cold Chain: Designing a Novel Freeze-Stable Vaccine</b>   |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>To design a novel vaccine that does not lose its potency upon freezing as an alternative to freeze-sensitive aluminum-based vaccines.</p> <p>A lipid blend-complex made of natural, biodegradable lipids might be a good alternative to Aluminum-based adjuvants. If the antigen-lipid blend complex withstands freeze-drying (lyophilization) without loss of activity, it will also be stable after freezing because during freeze-drying the vaccine will be frozen at -45 oC.</p> <p><b>Methods/Materials</b><br/>The immunogenicity of two liposomal vaccine formulations (liquid and lyophilized) was compared to that of an Aluminum phosphate (Adju-Phos) based vaccine using chicken egg Lysozyme as a model protein. The Lysozyme was entrapped in liposomes and adsorbed to Adju-Phos. As control, Lysozyme solution in 10% sucrose without adjuvant was used. Concentrations of entrapped and unbound Lysozyme were measured using UV Spectrophotometry with each measurement repeated three times.</p> <p>Each formulation was injected into four mice (i.e. 16 mice total) intramuscularly on days 0 and 14. Blood was collected on Day 28. The mouse antibody response to each vaccine was measured in diluted sera of immunized and non-immunized mice by an Indirect ELISA method. The concentration of antibody in each mouse was measured twice at eight different dilutions.</p> <p><b>Results</b><br/>Both liquid and lyophilized liposomal vaccines gave a significant 3-6-fold immunogenic response greater than that of the Lysozyme solution without adjuvant. The lyophilized liposomes appeared to be slightly better (around 2 fold) than the liquid non-lyophilized liposomes. Adju-Phos Lysozyme vaccine had the highest immune response that was 9-fold more than the Lysozyme solution. Statistically, the lyophilized liposomes and Adju-Phos had similar immune responses.</p> <p><b>Conclusions/Discussion</b><br/>Using a natural lipid composite as an adjuvant, it was possible to manufacture a vaccine with entrapped protein antigen that had a significant immunogenic response in mice. This natural lipid composite did not lose its immunogenic activity upon lyophilization and might thus be used as a freeze-stable vaccine as an alternative to Aluminum salt adjuvants.</p> |                                       |
| <b>Summary Statement</b><br>A novel freeze-stable vaccine with potent immunogenic IgG induction in mice similar to that of Aluminum-based vaccines was successfully designed and tested in vivo.  |                                       |
| <b>Help Received</b><br>Used lab equipment at HTD Biosystems under the supervision of Dr. Rajiv Nayar; Mice immunization was conducted at Pacific Biolabs   |                                       |



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| <b>Name(s)</b><br><b>Ramya Swami; Sanjna Verma</b>   | <b>Project Number</b><br><b>S0530</b> |
| <b>Project Title</b><br><b>Effects of Dietary Vitamin D on the Progression of Breast Cancer Tumors in Mice</b>   |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>Breast cancer is one of the leading causes of death among women in the USA, and numerous studies have shown a correlation between low levels of vitamin D in the blood and increased risk of cancer.<br>Objective: To determine whether the anti cancerous properties calcitriol exhibits will affect the progression breast cancer (calcitriol is the active hormonal form of dietary vitamin D in the body)<br>Hypothesis: If a subject diagnosed with breast cancer is treated with high concentrations of dietary vitamin D (vitD), then the progression of tumor growth will be slowed.<br><b>Methods/Materials</b><br>Female mice were divided into 3 groups that differed on dietary concentrations of vitamin D. There was a control group with normal diet;a high vitD diet group to induce high vitD levels in the system;a low vitD diet group to induce low vitD levels in the system. After one week, the mice were injected with breast cancer cells and monitored weekly for changes in body weight, tumor size and appearance. At the end of 7 weeks, tumors from 15 mice were excised and processed for determination of specific gene expressions. Treatment was continued in the remaining animals for an additional 2 weeks to assess differences in tumor growth rates. Methods for gene expression include RNA isolation and quantitative real-time PCR.<br><b>Results</b><br>There was no apparent difference between low vitD group and the control. Mice that were fed a high vitD diet demonstrated smaller tumor size and a two week delay in the appearance in the tumors. Using six genes for gene expression analysis, it was determined that the vitD was significantly inhibiting the growth of the cancer cell from local synthesis of calcitriol. Genes were CYP27B1, CYP24, Estrogen, COX2, CYP19, and vitD receptors, all of which drive cell proliferation.<br><b>Conclusions/Discussion</b><br>The experiment successfully demonstrated the beneficial effects of high vitD on the progression of breast cancer. With the results from the gene expression analysis, it was shown that a local synthesis of calcitriol within the tumor decreased cancer cell proliferation. Due to time constraints, the low vitD diet group was not sufficiently deficient in vitD levels and therefore exhibited similar characteristics to the control diet group. However, comparing the high vitD diet group to the control still helped to formulate the conclusion that the progression of breast cancer was slowed for mice treated with high vitD diets. |                                       |
| <b>Summary Statement</b><br>Derived from numerous studies, calcitriol exhibits anti-cancerous properties which provides the basis for the experimental setup that dietary vitamin D will slow the progression of breast cancer   |                                       |
| <b>Help Received</b><br>Used lab equipment at Stanford University under the supervision of Dr. Srilatha Swami and Dr. David Feldman  |                                       |



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| <b>Name(s)</b><br><b>Tanay Tandon</b>  | <b>Project Number</b><br><b>S0531</b> |
| <b>Project Title</b><br><b>Novel Detection of Hematological Cancer: A Proposed Conductivity Based Analysis for Early Leukemia Cell Identification</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>The objective of this research was to define a novel method of detecting hematological malignancies (blood cancers) based on conductivity fluctuations within the blood. This study aimed to lower blood cancer detection costs, with focus on portable cancer detection devices. As opposed to current methods of detection (flow cytometry, cell cytogenetics), which are confined to laboratories; the proposed form of detection was aimed to function in home-based environments, and provide instantaneous blood readings. Such a method could revolutionize blood cancer detection, by simplifying the detection process, and reducing costs.</p> <p><b>Methods/Materials</b><br/>The research was conducted by isolating cell samples of healthy Canine White Blood Cells (WBCs), and diseased ML3 Leukemia Canine cells. Following this, the cells were separated and re-concentrated through centrifugal process into 4 experimental concentrations. The YSI-80 Conductivity Meter was then used to take milliSiemens/cm Conductivity readings for each of the experimental groups over various trials. The mS/cm Conductivity data was then consolidated to chart the conductivity values of ML3 Leukemia cells in comparison to healthy WBCs. Based on differences in conductivity readings; Leukemia would be distinguishable.</p> <p><b>Results</b><br/>The data showed a 2% drop in conductivity in Leukemia Cell groups in comparison to healthy WBC samples. The healthy blood cell groups maintained average conductivity readings of 13.7385 mS/cm, while the Leukemia test groups had a 13.57725 mS/cm average reading. This conductivity drop between the samples was proven as a statistically significant trend using T-Test analysis with a P-Value of .002. Furthermore, the data proved the CFU-GM Myeloblastic Absorption Theory, which explains the conjecture behind Leukemia cell conductivity drops, and validated the produced data trends.</p> <p><b>Conclusions/Discussion</b><br/>The research has shown that Leukemia is distinguishable from healthy blood samples based on Conductivity readings. This method of Leukemia detection reduces detection costs by 99.75%; and could maximize portability by operating in the format of a handheld conductivity meter. The proposed conductivity based method is comparable to a #thermometer# for blood cancer, by providing a low-cost preliminary detection method. Furthermore, the detection method could help Leukemia relapse victims monitor blood health from within their own homes.</p> |                                       |
| <b>Summary Statement</b><br>I created a novel method of detecting blood cancer based on Conductivity; that could eventually lower costs, and detect Leukemia earlier.  |                                       |
| <b>Help Received</b><br>Dr. Valerie Morris at the Fred Hutchinson Cancer Research Center gave access to lab facility and answered questions. Dr. Majeti from Stanford answered questions through email, and in person.   |                                       |



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| <b>Name(s)</b><br><b>Anna T. Thomas</b>  | <b>Project Number</b><br><b>S0532</b> |
| <b>Project Title</b><br><b>Exploring Neural-Immune Synergy: TNF Inhibition Protects Against Maternal Illness Induced Neuronal Dysfunction in Autism</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>Intrigued by structural parallels between the neuronal synapse and immunological cell-cell junction, I first decided to explore whether the inhibitory neurotransmitter GABA could function at the "immunological synapse" to regulate the response to infection. Second, as maternally inherited GABAR mutations and maternal infection are the most common factors contributing to autism spectrum disorders (ASD), and autistics often experience immunological dysfunction, I theorized that a neural-immune interaction, analogous to the interaction in vitro, could play a major role in ASD. I thus aimed to characterize the long term impact of maternal infection on GABAergic interneurons in the brain. The third aim was to identify a preventative strategy against maternal infection induced ASD.</p> <p><b>Methods/Materials</b><br/>I treated RAW 264.7 macrophages with LPS, an immune stimulant, and muscimol (GABA agonist) or bicuculline (antagonist). ELISA and Griess tests assessed production of hypoxia inducible factor, the cytokines TNF-a and IL-6, and nitric oxide. To characterize long term impacts of maternal infection on the offspring and determine a preventative measure, I performed immunohistochemistry, microscopy, and image analysis using ImageJ on brain tissue of WT and TNFR1 knockout mice exposed to maternal infection.</p> <p><b>Results</b><br/>Remarkably, inhibition of GABAergic signaling by bicuculline increased cytokine and nitric oxide production; heightened GABAergic signaling inhibited inflammation and hypoxia. Immunohistochemistry identified three interneuron populations impacted in the adult hippocampus and cortex. Multiple linear regression analysis revealed a novel relationship between infection and autistic like behavioral impairment, identifying a key role of parvalbumin interneurons in modulating behavior. In a novel result, TNFR1 knockout restored cortical interneuron density to within 4.2% of normal levels.</p> <p><b>Conclusions/Discussion</b><br/>While previous studies have identified GABA receptors in immune cells, this study is the first to demonstrate the function and extent of GABAergic signaling in immune cells, in roles as diverse as regulation of hypoxia and nitric oxide, both of which are key cellular messengers. This study has also identified critical therapeutic targets which correlate with autistic like behavior. TNFR1 knockout mediated protection against interneuron loss indicates the viability of TNF inhibition to prevent ASD post-infection.</p> |                                       |
| <b>Summary Statement</b><br>I identified a novel source of neural-immune synergy in macrophages and applied my findings to characterize and develop a preventative strategy against maternal infection induced interneuron dysfunction, a leading cause of autism.   |                                       |
| <b>Help Received</b><br>Used lab equipment at the Palmer Lab and HTBC of Stanford University. Palmer Lab provided behavioral data for analysis. ADRF funded cell culture studies. BABEC, Invitrogen, NanoEnTek, Cayman Chemical, Bio-Rad, Cell Signaling Technology, Jr Scientific, eBioscience, and R&D donated reagents.   |                                       |



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| <b>Name(s)</b><br><b>Emily To</b>  | <b>Project Number</b><br><b>S0533</b> |
| <b>Project Title</b><br><b>Intracellular Ion Channel Drug Potency Assay: Ensemble Mitochondrial Measurements Demonstrate Bilayer Platform Potential</b>  |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>Ion channels and transmembrane proteins are crucial in regulating many physiological processes. Scientific and sensing measurements of ion channel conductance depicting activity often utilize lipid bilayers and Patch Clamp, which have great shortcomings in application. This experiment uses a bilayer formation system that integrates the measurement electronics within the fluidic controls. The system enables commercial operation of the platform, a step toward applications of ion channel measurements for remote sensing and pharmacological studies requiring minimal operator involvement.<br><b>Methods/Materials</b><br>Novel lipid bilayer platforms were created from several acrylic plates following a modified stencil of a previously developed platform being studied at the time. A gravity-propelled pin-tool mechanism was used to form the bilayers and to read ionic flux. Results of flux obtained reflect the efficacy of the platform as a suitable alternative to Patch Clamp in its ability to read, characterize, and isolate multiple ion channels with ease. The experiment was conducted with mitochondrial ion channels as a model channel with identifiable characteristics.<br><b>Results</b><br>After a series of homogenizing and centrifuging, the cells were obtained. The resulting membrane fragments containing the ion channels were incorporated into the lipid bilayer and currents were run through the electrode to receive conductance levels. The levels accumulated were then used to study characteristics of ion channels found in the mitochondria. Blockers were added, ionic flux was successfully acquired, and the platform was proven highly effective.<br><b>Conclusions/Discussion</b><br>Information accumulated could provide additional insight into the functions and potentials of ion channels, as well as a new mechanism to study ion channels, drug-screening, and cell characterization. |                                       |
| <b>Summary Statement</b><br>Novel lipid bilayer platforms are designed to replace Patch Clamp technology in order to efficiently study intracellular ion channels commonly found in mitochondria.  |                                       |
| <b>Help Received</b><br>Used lab equipment under supervision of Dr. Jacob J Schmidt; Graduate student/mentor Ahmad El-Arabi at UCLA  |                                       |



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| <b>Name(s)</b><br><b>Sailee Yadav</b>   | <b>Project Number</b><br><b>S0534</b> |
| <b>Project Title</b><br><b>Targeting ER Stress in Type 2 Diabetes: A Bittersweet Battle</b>   |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>Type 2 diabetes and obesity are major public health priorities because of their high prevalence and incidence in the U.S. population. Although the mechanisms underlying type 2 diabetes are incompletely understood, research has shown that exposure of pancreatic beta-cells to the environment and dietary stress induces endoplasmic reticulum (ER) stress that can ultimately lead to cell death and cause diabetes. In this project, we tested our hypothesis that bitter melon protects the cells from free fatty acid induced ER stress and hence is a potential anti-diabetic drug for the prevention and cure of type 2 diabetes.<br><b>Methods/Materials</b><br>We profiled how bioactives from bitter melon counter ER stress by analyzing the signaling involved in ER stress (HAC1 splicing) and exploring its downstream apoptotic pathways using baker's yeast ( <i>Saccharomyces cerevisiae</i> ) as a model. The dietary effects of omega-6 (gamma-linoleic acid) and omega-3 (alpha-linolenic acid) free fatty acids and 4-hydroxynonenal (4HNE), one of the stable byproducts of omega-6 fatty acid metabolism, were checked upon the induction of ER stress. We also tested whether bitter melon counteracted the ER stress caused by these dietary factors. The mean and standard deviation of three independent experiments was calculated.<br><b>Results</b><br>Our results show that bitter melon protects yeast cells from fatty acid induced ER stress and hence has the potential to be anti-diabetic therapy.<br><b>Conclusions/Discussion</b><br>These studies show that bitter melon target the endoplasmic reticulum and may help the cells to overcome ER stress. It can be a potential drug for cure and prevention of type 2 diabetes. Our ultimate goal is to develop a drug incorporating the "superfood" properties of bitter melon that will win the bittersweet battle against type 2 diabetes and other metabolic diseases. |                                       |
| <b>Summary Statement</b><br>In this project, we explored the effects of bitter melon on fatty-acid induced endoplasmic reticulum stress as a mechanism for preventative drug therapy for type 2 diabetics.  |                                       |
| <b>Help Received</b><br>AP Biology teacher, Mr. Craig Monden, helped brainstorm techniques and ideas; Trained with lab procedures and equipment at City of Hope National Medical Center under the supervision of Dr. Sushma Yadav   |                                       |



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| <b>Name(s)</b><br><b>Kelly X. Zhang</b>  | <b>Project Number</b><br><b>S0535</b> |
| <b>Project Title</b><br><b>Fluorescent Imaging for Nano-Detection (FIND) of Cancer Cells for Future Surgery</b>  |                                       |
| <p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b><br/>Currently, surgeons operate blindly on tumor patients: many tumor cells escape surgery and spread. The purpose of this project is to introduce a biodegradable fluorescent nanoparticle for imaging tumors to aid cancer surgery. The three main goals of this year's study are: 1) to optimize the procedure for making fluorescent nanoparticles to achieve a particle under 200 nm, 2) to study the uptake of the nanoparticles with a variety of tumor cell lines, and 3) to determine the mechanism of nanoparticle uptake by tumor cell lines.</p> <p><b>Methods/Materials</b><br/>The fluorescent nanoparticles were made with albumin protein and FITC fluorescent dye using a method called desolvation. In order to optimize the protocol to achieve the smallest sized particle, certain conditions were varied while all other variable were controlled, and the average size of each sample was measured. In order to study the uptake of the nanoparticles by different types of cancer cells and demonstrate the general utility of this imaging method, the nanoparticles were incubated with eight cancer cell lines. A study of competition was designed to determine the mechanism of nanoparticle uptake. Increasing concentrations of unlabeled albumin were added to the cells along with the nanoparticles and the numbers of fluorescent cells were counted.</p> <p><b>Results</b><br/>The optimized protocol yielded nanoparticles under 200 nm, right inside the target range. The study of the nanoparticle uptake by cancer cell lines showed a positive uptake of the nanoparticles by all eight tumor cell lines. The uptake mechanism study revealed a decrease in the number of fluorescent cells with an increasing concentration of additional albumin.</p> <p><b>Conclusions/Discussion</b><br/>The optimization of the protocol achieved a particle size that has the highest chance of escaping the blood vessel to the tumor tissues. The study of the nanoparticle uptake by cancer cell lines shows that the fluorescent nanoparticles can be uptaken by a variety of tumor cell lines, demonstrating that this imaging system can be used for a variety of different cancers. Finally, the results from the mechanism of particle uptake indicates that the mechanism is receptor mediated and not by general endocytosis. In future surgery, the nanoparticles can be injected to a patient's circulatory system, escape to the tumor, attach, and glow. This would allow surgeons to completely remove the tumors, extending the lifetime of many patients.</p> |                                       |
| <b>Summary Statement</b><br>The purpose of this project is to introduce a biodegradable fluorescent nanoparticle for imaging tumors to aid cancer surgery.   |                                       |
| <b>Help Received</b><br>Used lab equipment at the University of the Pacific under the supervision of a graduate student, Poonam Saraf.   |                                       |



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| <b>Name(s)</b><br><b>Larry Zhang</b>   | <b>Project Number</b><br><b>S0536</b> |
| <b>Project Title</b><br><b>HMM Model Usage to Determine Gene-Specific CpG Ratios and Location of Genes</b>   |                                       |
| <b>Abstract</b><br><b>Objectives/Goals</b><br>1. To locate introns and exons within a sequence of DNA using dinucleotide frequencies that differ between the two<br>2. Calculate gene-specific CpG dinucleotide ratios between introns and exons and compare between related genes<br><b>Methods/Materials</b><br>Lenovo Computer<br>Sample Hidden Markov program<br>DNA data from various databases (GenBank, fruitfly.org, etc.)<br><b>Results</b><br>The HMM was not able to successfully locate introns and exons on the DNA, though it did calculate specific CpG dinucleotide frequencies for each gene, which allowed me to calculate a CpG ratio. However, these CpG ratios ranged from 3-7, with no obvious or direct correlation between related genes such as BRCA1/BRCA2 within humans/mice, which both cause breast cancer, or OCA2 between humans, flies, and mice, which codes for eye color.<br><b>Conclusions/Discussion</b><br>The HMM program did not successfully locate introns and exons, probably due to the small size of training data to tell it the frequencies of dinucleotides between introns and exons. A successful training set would probably have around 200 genes, with introns and exons labeled. Additionally, this experiment used various genes from various locations in the genome of humans, mice, and flies, which brings in the complication that frequencies of dinucleotides simply are not constant between different regions of the genome. Additionally, the CpG ratios found had no real correlation between related genes, showing that dinucleotide frequencies (or more specifically, the CpG dinucleotide) have no relationship to the gene's function. |                                       |
| <b>Summary Statement</b><br>This project uses dinucleotide frequencies to calculate locations of introns vs exons and calculated unique frequencies for various species.   |                                       |
| <b>Help Received</b><br>Obtained sample HMM program from Dr. Li of Avid Academy  |                                       |



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| <b>Name(s)</b><br><b>Maria D. Zumkeller</b>   | <b>Project Number</b><br><b>S0537</b> |
| <b>Project Title</b><br><b>Comparing Iron Concentration of Organic and Non-Organic Vegetables</b>   |                                       |
| <b>Objectives/Goals</b><br>The purpose of my investigation is to compare and determine the relationship between iron concentration of organic and non organic spinach and potatoes.   |                                       |
| <b>Abstract</b><br><b>Methods/Materials</b><br>To determine the unknown concentration of iron in the food samples, I prepared a series of standard solutions in which the analyte concentration (iron) is precisely known. I then measured the transmittance and absorbance of the standard solutions using a spectrophotometer. The absorbance measurement of each standard solution was then used to create a calibration curve which demonstrated how the experimental observable (the absorbance) varies with the concentration. The unknown solution was then analyzed using the same method as employed to create the standard solutions yet in this case the analyte is the food sample. The absorbance and transmittance levels of the food sample solutions were then measured. In order to calculate the iron concentration of the food samples, the average absorbance of the unknown solution trial was used with the slope and intercept from the calibration curve through a specific equation. |                                       |
| <b>Results</b><br>There was not a significant amount of change of iron concentration between the organic and non organic vegetables thus proving my hypotheses. When plotted, the average amount of iron in the organic and non organic vegetables (spinach and potatoes) did not demonstrate a drastic variation.  |                                       |
| <b>Conclusions/Discussion</b><br>Iron is an essential component to the proper function of the human body. It is crucial to the creation of hemoglobin that carries oxygen to all parts of the body and regulating cell function throughout the body. A lack of the mineral can lead to susceptibility of illnesses and conditions such as anemia. Thus, the amount of iron intake one receives can determine overall health. Many americans have accepted the fallacy that organically grown vegetables provide more nutrients than non organically grown vegetables and possess an overall higher nutritional value. However, my experiment has refuted this belief.   |                                       |
| <b>Summary Statement</b><br>Comparison of the iron concentration within organic and non organic vegetables using the method of spectrophotometry.   |                                       |
| <b>Help Received</b><br>Used lab equipment at Sanger High School under the supervision of Nathan Whittington, science teacher.  |                                       |