



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Anshu R. Abhat	Project Number S0401
Project Title Effect of Neurotrophin-3 on Suppression of Tumor Vascularization	
Abstract Objectives/Goals Investigations were performed on two sets of glioma tumors that were generated earlier in the absence or presence of Neurotrophin-3 (NT-3) with a significant difference in size. The objective of my study was to evaluate and analyse the disparity between the growth of the two tumors. Methods/Materials Immunohistochemistry was performed on cryostat sections using antibodies specific for brain cell types and protein markers expressed on blood vessels. I further carried out double immunofluorescence imaging using different conjugated fluorochromes (red and green) with an epifluorescence microscope. Results The evaluation of digital images of AT and ATNT-3 glioma tumors revealed an elaborate network of vascularization (angiogenesis) of AT tumor and a total lack of angiogenesis in ATNT-3 tumor. The analysis further showed that progression of AT tumor growth appears to be directly related to the supply of growth nutrients in the core of the tumor mass. Conclusions/Discussion The present study suggests an inhibitory role of NT-3 in angiogenesis and suppression of glioma growth. NT-3 appears promising in tumor therapy.	
Summary Statement The comparative study of the AT tumor and the ATNT-3 tumor suggests that NT-3 has the potential to prevent vascularization.	
Help Received Mentor at UCLA (Dr. Shalini Kumar) provided the tissue samples, antibodies, and microscope for investigation. Lab facilities at UCLA were used. Father helped arrange poster board.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Sukhjeet S. Batth	Project Number S0402
Project Title Protein Profile Analysis: A New Application May Streamline Structural Predictions	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals The ability to predict protein structure from amino acid sequence alone would be invaluable to scientists everywhere. A pharmacologist working on creating a new drug, for example, could analyze a protein sequence with his computer and find its structure in seconds. Computational biology is far more efficient than the long, tedious analyses of structural biologists which include x-ray crystallography and nuclear magnetic resonance. The present study approaches this challenge from a very specific position. A method of protein sequence analysis, Profile Analysis, was applied to create a structural prediction for alpha-helix packing.</p> <p>Methods/Materials Profile Analysis was used to predict initial contact residues (i's) in packing diamonds of 4-3 alpha-helix packing. Position-specific scoring matrices (profiles) were made from a structure correlated scoring matrix and amino acid sequences from either +3 or +4 packing alpha-helices. Different helix lengths were used to create profiles which helped establish the most accurate method of applying Profile Analysis. Reliability was also found with the use of leave-one-out and z-score analyses of predictions in cholecystokinin.</p> <p>Results Profiles made from the helix range consisting of i to i+11 were determined to be the most accurate for the prediction of both +3 and +4 packing alpha-helices among sequences of known structure. In addition, leave-one-out and z-score analyses confirmed that predictions in cholecystokinin by profiles created from the range i to i+11 were within the range of accuracy. These profiles predicted Leucine-13 and Arginine-31 as the initial contact residues of a +3 and +4 helix, respectively, in human cholecystokinin.</p> <p>Conclusions/Discussion Based on accuracy of predictions for known helices and further supported by reliable predictions within the range of accuracy for cholecystokinin, it seems that 4-3 alpha-helix packing relies heavily enough on sequence to be predictable by this method. With further study, this application of Profile Analysis could eventually replace the long, tedious, and costly analyses of structural biology for prediction of structures which rely as heavily on sequence as 4-3 alpha-helix packing.</p>	
Summary Statement The present study attempted to predict a specific protein structure, 4-3 alpha-helix packing, using a computer protein sequence analysis, Profile Analysis.	
Help Received	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Max R. Biessmann	Project Number S0403
Project Title PCR: How Much DNA Do You Need?	
Abstract Objectives/Goals How little DNA is necessary to amplify a gene by polymerase chain reaction (PCR)? My objective was to determine the optimal conditions for PCR to amplify the tubulin gene from the smallest amount of fruitfly DNA. I believe that DNA from one fly will be enough, but perhaps I can use even less. Methods/Materials I used programmable PCR amplifier machine, Taq polymerase, nucleotide primers, buffers and nucleotides, gel apparatus, eagle eye detector for fluorescent DNA products. I used DNA from fruitflies to amplify a piece of the tubulin gene with two specific DNA primers. I determined the optimal conditions for a PCR reaction by varying several parameters such as annealing temperature and the number of amplification cycles. Results Using these optimal conditions I showed that PCR was indeed able to amplify DNA from a single fly. Experiments were repeated several times, and by diluting the DNA from a single fly up to 1000 fold, I calculated that I could amplify the tubulin gene from as little as 25 picograms of total fly DNA or 10-12 grams of total fly DNA. Conclusions/Discussion The polymerase chain reaction, PCR, is a very powerful method for amplifying small amounts of DNA if one knows the optimal conditions. I was able to show that only a very small amount of DNA is needed to "xerox" many copies of the targeted DNA for further biological studies. That is why PCR is also used in forensics and in criminal investigations	
Summary Statement To find the optimal conditions at which PCR reactions occur and to find the minimal amount of fruitfly DNA needed to amplify a single copy gene.	
Help Received Used lab equipment at UCI under the supervision of Dr. Harald Biessmann	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Matt E. Bowman	Project Number S0404
Project Title Hfr-1 and the Hessian Fly	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals The goal of my project was to determine how the Hfr-1 resistance gene is affected by the Hessian Fly.</p> <p>Methods/Materials Using TRIzol rna preps I was able to quantify wheat tissue that had been infested with Hessian Flies. Then I ran Northern blots to move the rna onto a Hybrid membrane. I then hybridized with a radioactive probe and exposed the membrane to film.</p> <p>TRIzol, chloroform, ethanol, isopropanol, rna precipitation solution, agarose/formaldehyde gel: various pipettes, sterilized tubes, gel boxes</p> <p>Results The results of my three experiments were as follows: Exp. 1- I infested three pots with three male flies and three female flies, three pots with six of each sex, three pots with twelve of each sex, and three pots with twenty-four of each sex. Each group has its own uninfested control. I harvested the pots five days later. I performed TRIzol rna preps and Northern blots to come up with my results. The Hfr-1 resistance gene is not affected by higher levels of Hessian Flies. Exp. 2 I wanted to see in which part of the wheat plant Hfr-1 was most induced. I infested ten pots with six flies of each sex. I then took parts of the plant. I determined that Hfr-1 is most induced 2cm above the ground. Exp. 3 I wanted to determine if as long as Hessian Flies were present that Hfr-1 would stay induced. I infested eight pots with six flies of each sex. I then harvested one group and reinfested the others. I repeated this process until I finished them all. My results were that Hfr-1 is not affected by the amount of time the flies are present.</p> <p>Conclusions/Discussion Exp.1 The Hfr-1 resistance gene is not affected by large amounts of Hessian Flies. Exp.2 Hfr-1 is more heavily induced 2cm above the ground Exp. 3 Hfr-1 is not affected any differently as long as Hessian Flies are present.</p>	
Summary Statement To determine how the Hfr-1 resistance gene in wheat is affected by the Hessian Fly	
Help Received My aunt helped me with the research, my mom helped me with the board	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Kenny H. Chow	Project Number S0405
Project Title Age and Lesion-Induced Changes in Protease Nexin 1 Expression in the Brain	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals The blood brain barrier is an important structure in the maintenance of brain homeostasis. Protease nexin 1 (PN1) is a 43-kDa serine protease inhibitor (serpin) found predominantly in the brain. Secreted by a variety of glial cells, PN1 concentrates around blood vessels and capillaries to inhibit blood-borne thrombin. Extravasation of thrombin due to blood brain barrier compromise has been shown to be remediated by potent PN1 inhibition. PN1 activity clears thrombin, and promotes neuritic outgrowth vital in nervous system development and repair processes. The present study was designed to test the hypothesis that PN1 concentrations after injury are increased, while decreased with brain aging.</p> <p>Methods/Materials For these experiments, two methods of analysis will be employed. In the first experiment, immunocytochemistry will be used to compare the expression of PN1 in brains with ibotenic acid lesions that simulate brain injury. In the second experiment, immunocytochemistry will be used to compare intensity of PN1 staining in aged vs. young rats. In the third experiment, Western Blot analysis will be used to quantitatively compare expression of PN1 in aged vs. young rats.</p> <p>Results In the first study, rat brains with ibotenic acid lesions simulating injury in the brain were stained to verify the expression of PN1. Results of this immunocytochemical staining illustrated strong differences in PN1 expression at sites of injury, revealing an increase in response to brain damage. In the second study, PN1 concentrations were quantified in aged (over 24 months) and young (3 months) rat brains. Protein quantification assays carried out by Western blot revealed a decline in PN1 expression relative to age.</p> <p>Conclusions/Discussion These results suggest that PN1 levels could be tied to the decline in neuronal protection that accompanies memory loss, senile dementia, and neurodegeneration. PN1 has already been evidenced to contribute to the pathology of Alzheimer's disease, and it may be of interest to further explore its role in neuronal protection. The possibility of its use in therapeutics is also worth pursuing.</p>	
Summary Statement This project hopes to elucidate the roles of protease nexin 1 in aging and protecting the brain against injury that may arise from trauma or neurodegenerative disorders.	
Help Received Worked under the supervision of Sonsoles de Lacalle at the California State University, Los Angeles.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Michael R. Davis	Project Number S0406
Project Title The Effect of Temperature on the Enzyme Peroxidase	
Abstract Objectives/Goals I tested the effects of temperature on the enzyme peroxidase. Methods/Materials In the presence of hydrogen peroxide, peroxidase reacts to produce visible gas bubbles. I used prepared slices of potato, with uniform size and shape, because they are a peroxidase source. The potatoes were heated/cooled to temperatures that ranged from one to one-hundred degrees Celsius. I used a pipette to apply 1 mL of hydrogen peroxide to the surface of the potato. Then I measured the area of peroxidase that reacted with the potato. Results I found that the amount of foam increased relative to the temperature, until it reached 62° C, at which point it dropped off to 0%. Conclusions/Discussion As the temperature increased, so did the amount of foam. The kinetic energy of the enzyme increased, enhancing the interaction with the substrate, causing a larger reaction. However, when the enzyme reached 62° C., it became denatured and useless.	
Summary Statement I tested the effects of temperature on the enzyme peroxidase.	
Help Received I received no help on my project.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Jonathan R. Deans	Project Number S0407
Project Title Characterization of the Arabidopsis Auxin-Response Mutant 4(4,2) and an ARM Gene	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals Professor Mary Williams at Harvey Mudd College has identified a mutant in the plant Arabidopsis, and as a result of past experiments has concluded that it contains an F-Box motif and is similar in its phenotypical mutations to the f-box containing mutant TIR1. Both of these genes, when mutated show reduced shoot growth, and reduced lateral root growth. I plan to further research this plant mutation to give it an identity and know its purpose in the plant, and also to locate it on the chromosome.</p> <p>Methods/Materials To reach this goal i used the methods of a new field of science called Bioinformatics to compare and contrast this mutation with other genes in the Arabidopsis genome. This field of bioinformatics is done mostly on the computer using internet sites with databases of genomes. All of the lab work consisted of extracting DNA through buffers and washes, and using a PCR to duplicate the region of interest.</p> <p>Results My results showed that, from the plants whose DNA registered on the gel, all but one had the DNA of the mutant plant. The one other plant showed a normal plant. My results from comparing the gene sequence of the mutant gene to others in the genome were impressive. I only found one other gene that was closely related, but it was located on chromosome 2, not the chromosome in focus.</p> <p>Conclusions/Discussion From my results, it may have seemed as if the one plant whose results were not expected would turn my hypothesis of the region where the gene is encoded around, but after running plant 16 again the results were the same. What this tells me is that the gene does not lie south of where my specific primers ended, which only improves hypotheses about the genes locations. My results from the genome searches showed that there are other f-box proteins in the genome, and only a few are similar to the mutant that was found.</p>	
Summary Statement My project was the work in further identifying a mutant which nothing is known about in the plant Arabidopsis	
Help Received Mother proof-read project and helped assemble board. Used lab equipment at Harvey Mudd Colled under the supervision of Dr. Mary Williams	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Stephanie T. Do	Project Number S0408
Project Title Gene Regulation: The Use of Antibiotics to Control the Kinetics of Lactose Operon Induction	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals To investigate the effect of ampicillin and kanamycin on the lactose operon induction of Escherichia coli through enzyme assays.</p> <p>Methods/Materials In the experiment, the production of beta-galactosidase (the enzyme produced during induction) was monitored for three different treatments (the control and the two antibiotic solutions) within a 60-minute time period. There were two important chemical processes used in this experiment: IPTG and ONPG. The compound IPTG has a similar structure to lactose; it starts lactose operon induction without being metabolized. The enzyme produced is measured in the chemical reaction between ONPG and the beta-galactosidase. The nitrophenyl group that is broken off the galactose by the enzyme has a yellow color. A UV/visible spectrophotometer was used to measure the intensity of the yellow color at a wavelength of 405 nm. The absorbances of the cultured tubes were graphed versus reaction time to show the enzyme activities.</p> <p>Results After tracking the enzyme production for sixty minutes, the amount of beta-galactosidase being produced slows down drastically in the kanamycin solution after only twenty minutes. However, the ampicillin did not affect the induction of the lactose operon. When compared with the control, enzyme production in this treatment is normal.</p> <p>Conclusions/Discussion The kanamycin did have a lactose operon induction effect as predicted. Within an hour, the amount of enzyme production leveled off. However, the ampicillin did not seem to have any effect. The antibiotic solution probably did not have enough time to induce cell wall destruction. If data was collected to make a growth chart versus time for the antibiotic solution spiked with bacteria, then the effect each of the antibiotics had on the bacteria as a whole could have been observed. It would also enhance the investigation because the amount of cells growing could be compared along with the amount of enzyme produced. Further investigation could include the effect of antibiotic dosage.</p>	
Summary Statement The purpose of this investigation is to study the effect two types of antibiotics will have on the production of beta-galactosidase in Escherichia coli.	
Help Received Used laboratory equipment at La Sierra University under the supervision of Dr. James Wilson.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Katie Dunbar; Stanford Tran	Project Number S0409
Project Title Will the Recessive Allele of a Fruit Fly's Eye Color Be Exhibited in the Presence of a Dominant Allele?	
Objectives/Goals The objective is to determine if a recessive allele will show in the phenotype of a <i>Drosophila melanogaster</i> eye color.	
Abstract	
Methods/Materials Sepia flies, which has recessive brown eyes, was crossbred with Wild flies, which had dominant red eyes. Their offspring's eyes, looking exactly like the Wild, were compared to the parents by the process of paper chromatography. The chromatograms were only visible under a fluorescent black light.	
Results There was slightly less drospterin (orange) pigment in the Heterozygous compared to the Wild. The xanthopterin (green-blue) pigment is very dark in the Sepia whereas very light in the Wild. The data was quantified by measuring the Rf value, pigment length, and using a spectrophotometer, which all measured the amount of pigment. There was a noticable difference between the Homozygous and Heterozygous eye pigment in the spectrophotometer at 4000 angstrom units, right at the lower edge of ultraviolet radiation. Sepia had the lowest Rf value, followed by the Heterozygous and Wild. The Heterozygous almost had the exact same Rf value as Wild, but the Wild was slightly higher.	
Conclusions/Discussion The Wild's eye color isn't exactly the same as the Heterozygous's, as shown in the Rf values, pigment lengths, and spectrophotometer readings. This experiment proves that the dominant trait doesn't override recessive trait completely, as stated in many textbooks.	
Summary Statement In a <i>Drosophila melanogaster</i> (fruit fly's) eye color, the Heterozygous condition does NOT have the exact same phenotype as the Homozygous condition.	
Help Received Used lab equipment at school; Did all experiments under Mr. Dowling supervision	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Sonia Gupta	Project Number S0410
Project Title Original Mutagenesis Strategy Reveals Novel Activities in a Drosophila Gene and Potentially Any Gene of Interest	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals Just like Dr. Frankenstein and his accomplice, Igor, I created mutants. Now, my mutants are a bit different from Frankenstein, but nonetheless they are paving a new path for science. NOVA, Novel OVexpression Activity is a mutagenesis scheme that I developed to use a mutagen, try to mutate rhomboid and Star transgene, and overexpress them. The NOVA mutagenesis approach is used to analyze two genes, rhomboid and Star, which play an essential role in regulation of a signaling pathway during the development of multi-cellular organisms, including everything from flies to humans. The idea of NOVA analysis is to expose a transgene to mutagenesis, express the mutated transgene at high levels, and then screen for novel phenotypes in the wings. After the mutants are found, their DNA is analyzed to figure out the exact spot at which the molecular lesion occurs.</p> <p>Methods/Materials Using Drosophila fruit flies, I crossed the flies and used a strong driver, GAL4, to overexpress the gene rhomboid. After a few generations, I screened the offspring for mutants. Once the mutants were found, the DNA was extracted, put through a Polymerase Chain Reaction (PCR), run through a gel, and finally, sequenced. The data is then analyzed. A construct is created, and then it is injected into the fly and generations are crossed to be certain that the original phenotype is present in the fly.</p> <p>Results I was able to isolate novel phenotypes, called Dominant Negative and Neomorphic. Additionally, I was able to analyze the molecular lesion that was responsible for the novel phenotypes.</p> <p>Conclusions/Discussion My conclusion is that the NOVA strategy can be applied to two genes in Drosophila. In principle, it can be used as an effective tool for generating dominant mutations in genes of unknown functions. Diseases, such as genetics ones and cancer, are caused by a deregulation of endogenous proteins. Since most of cancer is caused by the overproduction, the use of Dominant Negative mutations can help. The use of Dominant Negative forms of these mutations may be used to control these components that may be a great utility of curing such diseases. This may be done through gene therapy by introduction of Dominant Negative. Drosophila is merely a tools to find information on forms of Dominant Negative mutations. Then, the information can be applied to human genes in something such as gene therapy.</p>	
Summary Statement Using NOVA, I was able to generate novel phenotypes in Drosophila mutants and analyze the exact molecular lesion where this occurred.	
Help Received Used the lab equipment at University of California, San Diego under the supervision of Annabel Guichard; My supervisor, Annabel Guichard, also helped me learn how to use computer programs such as Adobe Illustrator, Excel, Photoshop, etc.; My sister, Monica, helped me chose the colors for my board;	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Rehana R. Hassan	Project Number S0411
Project Title The Effect of Various Household Chemicals on Hemoglobin Testing with Luminol	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals The project objective was to determine if household chemicals other than detergent or bleach would interfere with the reaction of luminol and hemoglobin in a hypothetical crime scene atmosphere.</p> <p>Methods/Materials For this experiment, I needed human blood (blood donor was tested and cleared for diseases, Hepatitis, and HIV), carpeting, luminol, water, detergent (control), ammonia (var.1), nail polish remover (var.2), mouthwash (var.3), a light meter, measuring utensils, and a dark room. I created a hypothetical crime scene by cutting carpet into 3" by 3" squares and pouring 10cc of human blood on to each square. To prove that the luminol reacted w/blood, I let the blood dry without interference, sprayed the luminol on, and measured the intensity of the glow using a light meter. Then I began my experiment. I cut up the same number of squares for the 4 groups and poured 10cc of blood onto the center of each square, letting the blood dry 3 days. I then scrubbed out the blood using the appropriate solutions and rinsed them with water to remove any residue, let dry for 3 days. After dried, I went into a dark room and sprayed each square with luminol and measured the intensity of the glow - each square was sprayed one at a time at identical conditions.</p> <p>Results The control and unwashed sample (to prove the luminol worked) both had about the same reading on the light meter, 4.1. The detergent had an average reading of 4 on the light meter. The mouthwash had an average of 3.76; the least affect on the reaction. The nail polish remover average is 3.28, it was slightly more potent than the mouthwash. The ammonia group had the strongest affect, the light meter read a mere 1.604.</p> <p>Conclusions/Discussion I have concluded that criminals are extremely stupid to begin with and most likely will only used detergent to clean up their bloody trails, so it will be fairly easy to catch them. Moreover, if a criminal uses something other than detergent to remove blood, it really will not do them any good; but using ammonia may help a little. However, I don't believe anyone would really be able to bear the stench of ammonia. Ammonia is still is not potent enough to full erase all DNA evidence; there was still a reaction during my small scale experiment. Also, forensic science is an extremely advanced field of science, and is at least one-step ahead of criminals.</p>	
Summary Statement In the field of forensic science, detectives use luminol to detect blood that has been washed out, this experiment is testing to find out if any household chemicals will interfere with luminol's reaction with the hemoglobin in blood.	
Help Received	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Kyle R. Hyman	Project Number S0412
Project Title Running with DNA	
Abstract Objectives/Goals The objective of my project was to see if the pH of a porous gel had an effect on the the structure of DNA molecules that are running through it. Methods/Materials Gels were prepared with electrophoresis running buffers of different pH?s (5, 8.3, 9, and 13). Three types of DNA molecules were used in each of the gels. One type of DNA was a 100 base pair ladder, a set of linear double helixes that differ in size by 100 base pairs; another is lambda Hind III, DNA from a bacterial virus cut into linear double stranded pieces of different sizes by an enzyme; the third is a set circular DNAs, also double helices. Results DNA in the pH 8.3 environment ran perfectly. The electrophoretic mobility of DNA in the gel of pH 5 was retarded and the quality was not as great as the pH 8.3 gel. The reason for this observation is that in such acidic environments, the H ⁺ ions neutralize the negatively charged phosphates that help link the nucleotides together in each chain. The DNA is less negatively charged and slows in its migration towards the positively charged electrode that represents the far end of the gel. The gel with the pH of 9 revealed that DNA ran even worse at high pH levels. The only type of DNA that ran at pH 9 was the circular DNA and that ran too fast to obtain proper results. The linear DNA in that gel did not even run at all. At such alkaline extremes, the hydrogen bonds holding the DNA double helix break and the two strands separate, thus nullifying the use of running the gel. However, the strands of circular DNA stays together because the strands are braided together even though the bonds break. The fourth gel with the extreme pH of 13 did not properly solidify. Therefore, I was unable to run the DNA through it to observe how it to observe how DNA would react at such an extreme pH. Conclusions/Discussion The results of my experiment support my hypothesis in that the pH of a gel has a major effect on DNA structures and thus the way the DNA runs through it. In order to obtain proper results in the most efficient manner, the pH of an electrophoresis gel should be 8.3. This pH allows for DNA to travel through the gel in a way that shows the data clearly and in the most timely manner.	
Summary Statement My project is about how pH affects the structure of DNA.	
Help Received I used lab equipment at the University of California Riverside under the supervision of Dr. Hyman	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Mira Lalchandani; Denton Sato	Project Number S0413
Project Title A Cellular and Molecular Investigation of the Downregulation of Wild-Type p53 by a Dominant-Negative Mutant p53 Allele	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals The purpose of this study was to investigate the effects a dominant-negative p53 protein has on the cellular and molecular response of prostate cancer cells to radiation. The hypothesis of this experiment is that cell cycle arrest after radiation is decreased by the downregulation of wild-type p53 by a dominant-negative mutant p53 allele in prostate cancer cells.</p> <p>Methods/Materials This experiment used vector/parental and R273H mutant cells of the CWR22R cell line to address whether ionizing radiation of prostate cancer is a good form of treatment by analyzing the status of p53 using Western blotting and flow cytometry methods to observe the gene on a molecular and cellular level. A method called mitotic trapping was also used with the CWR22R cells to differentiate between cells in G2 and M phases of mitosis.</p> <p>Results Although the Western blots and flow cytometry did not present sound evidence of the mutant R273H dominant-negative effect, there was some evidence that introduction of the mutant R273H p53 allele had the expected effect of reducing p53 function following radiation in the mitotic trapping data.</p> <p>Conclusions/Discussion The research emphasizes the importance of predicting the different cell cycle responses to cancer treatment, such as irradiation of cells, and how genetics can play an important part on the effectiveness of such treatment.</p>	
Summary Statement This project looks at the impact of a genetic mutation on cell destruction after exposure to radiation.	
Help Received Used lab equipment at the University of California, Davis Med Center under the supervision of Ms. Nancy Nesslinger and Ms. Susan Scott.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) James Lew; Michael Schapira	Project Number S0415
Project Title Monitoring Gene Expression in Pulmonary Carcinoma Using Oligonucleotide Arrays	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals Our purpose is to monitor the differences in gene expression between a cell line of pulmonary carcinoma (HTB174), a normal cell line (HTB175), and normal tissue using oligonucleotide arrays. We hope to target the specific involvement of individual genes and gene combinations that are involved in transforming normal tissue into carcinogenic tissue. By chance, we may also discover some genes not previously associated with the development of carcinogenic tissue. On top of this, we are looking to check any differences in gene expression in the normal tissue and the normal lab-produced cell line.</p> <p>Methods/Materials We first extract total RNA from the samples and isolate the messenger RNA. We then perform a reverse transcription reaction with the mRNA to make complimentary DNA, which is then made into double stranded cDNA in an effort to clean it. Once the cDNA is cleaned, we perform an in-vitro transcription to obtain a single stranded cRNA strand. Once this cRNA strand is cleaned, it is fragmented into smaller pieces, each containing a specific gene. These small segments containing individual genes are then hybridized on oligonucleotide arrays. Once hybridized, we scan the arrays and analyze the data using special software.</p> <p>Results Upon preliminary analysis, we have found that a few oncogenes and cell-cycle related genes are overexpressed in the cancerous cell line relative to the normal tissue sample. We are noticing that tumor suppressor related genes are underexposed in the HTB174 sample relative to the same normal tissue sample. Furthermore, we are noticing a number of genetic differences between the normal cell line, which is produced in a lab, and the tissue. However, these differences are not major and are not cancer-related. Due to some unpredicted "errors" in the processing of the data, we have not yet completed our analysis. Nevertheless, we will have comprehensive results by the time of the fair.</p> <p>Conclusions/Discussion Our results thus far are pointing us in the direction of conventional thought pertaining to pulmonary carcinoma. In terms of comparing normal cell lines to normal tissue, our results so far reaffirm the notion that synthetic cells are not exactly the same as naturally occurring ones - in a lab, these cell lines can multiply more, thus expanding the possibilities of genetic mutations. However, we still need more time to analyze the final results to make absolute conclusions.</p>	
Summary Statement We are monitoring and attempting to explain the differences in the over-expression and under-expression of certain genes in carcinogenic lung cells and normal cells.	
Help Received Used lab equipment and basic protocols at Affymetrix, Inc. under the direction of Ms. Angelyn Tracy and Dr. Garry Miyada.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Wing Y. Li	Project Number S0416
Project Title Interaction of Integrin alpha-3/beta-1 and Laminin-5 Modulates Alveolar Epithelial Barrier Formation	
Abstract Objectives/Goals This study investigated the interaction between integrin $\alpha 3/\beta 1$ and its ligand, laminin-5 in modulating alveolar epithelial barrier. Methods/Materials Cell Culture and preparation of slides- Primary type II cells were harvested from male Sprague Dawley rats. Procedures for cell extraction and animal sacrifice were pre-approved by review board. Culture media was changed every day. Slides that had an Ln-5 rich matrix were coated in Ln-5 for two hours and cells placed thereafter. Slides with extracellular matrix protein coating were also coated for two hours and cells placed thereafter. Western Blot- Western blots were done on SDS-page using a 7.5% stacking gel. The protein concentration of samples was determined through Bio Rad DC protein assay kit. Standard Western Blot procedure was followed. Immunofluorescence- Standard immunofluorescence procedure was followed. Results Cells plated on Ln-5 developed confluence at <24 h, those plated on plastic without Ln-5 developed confluence at 24-48 h, those plated on anti-integrin antibodies didn't develop confluence. Uniform scratch made with needle on confluent monolayers of AECs. Wound closure measured serially +/- anti-integrin $\alpha 3$ mAb, or either anti-Ln-5 mAb with/without blocking activity. Closure faster on Ln-5-coated (at <48 h) to uncoated plastic (50% at 48 h). Presence of anti-integrin $\alpha 3$ mAb reduced rate of closure for cells plated on Ln-5, and prevented closure for cells on plastic Conclusions/Discussion . Interaction is likely important for alveolar epithelial cell migration and spreading during re-epithelialization of alveolar surface following lung injury in vivo. Cells coated on Laminin-5 had higher levels of Integrin $\alpha 3-\beta 1$, which was probably attributed to the surface affinity of Laminin-5 or Laminin-5's ability to up regulate levels of Integrin $\alpha 3-\beta 1$. Interaction is likely to be important for epithelial barrier formation, which consists of cell adhesion, monolayer formation, migration and wound healing. Cell adhesion and spreading either induces Integrin $\alpha 3-\beta 1$, is promoted by Integrin $\alpha 3-\beta 1$ or both. The Laminin-5 coated filters bind cells at their $\alpha 3\beta 1$ receptors, which accelerates attachment and spreading, and probably induces the expression of more $\alpha 3\beta 1$, which further hastens the development of monolayers.	
Summary Statement Two proteins in the lungs are essential for the normal function and development of the lungs.	
Help Received Dr. Lubman and Dr. Hua Zheng gave me support and advice for this study. Parents gave me ride to and from lab.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Sean R. McCreery	Project Number S0417
Project Title Total DNA and Organism Complexity	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals The object of my project was to determine if I could extract pure DNA by a simple kitchen technique from several diverse plant and animal species, quantify the amount, and compare the results to answer the question: Do complex organisms such as mammals have more DNA than simpler organisms?</p> <p>Methods/Materials Six diverse plant and animal species had their total DNA extracted by a simple kitchen technique involving homogenization of the specimen in salt solution, cell degradation with papain and soap, and final extraction of the DNA with 99% isopropyl alcohol. The extracts were then poured through coffee filters and the dry weight of the DNA was determined. Purity was measured by UV Spectrophotometry.</p> <p>Results DNA weight for the specimens in grams were banana 0.28, onion 0.16, wheat 0.20, beef liver 0.28, chicken liver 0.27, and squid 0.33 (n = 9 for all with standard deviation calculated). DNA purity of the specimens was excellent with a mean A260/A280 of 1.9 (1.8 - 2.0 very pure). DNA weight in grams per 100 grams of specimen were banana 0.51, onion 0.13, wheat 2.05, beef liver 0.69, chicken liver 0.62, and squid 1.81.</p> <p>Conclusions/Discussion These results imply that some simpler organisms may have more DNA than more complex ones. Particularly in the case of beef and chicken liver, the specimens were quite comparable and the amounts of DNA were similar. This suggests that a bird and mammalian species may have similar amounts of DNA. However, there was poor correlation with the known C-values (DNA haploid weight), probably because of several specimen variables in weight, homogenization, cell size and ploidy. Finally, the fact that the absolute DNA extract weights were so similar between all species suggests a design problem that could limit the total amount of DNA extracted, making quantitative comparison between species undependable.</p>	
Summary Statement Total DNA was extracted from six diverse plant and animal species by a simple kitchen technique, tested for purity, quantified, and compared to determine if the amounts correlated with organism complexity.	
Help Received DNA purity was tested by UV Spectrophotometry by Tom Lum and Daniel Green at the Veterans Administration Campus, Martinez, CA., in the J.A. Green Laboratory of Immunology and Infectious Diseases.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Lalit R. Patel	Project Number S0418
Project Title Urbanization of Coastal Waters and Concocting Hormonal Chaos	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals The populace of Greater Los Angeles, a highly urbanized metropolis, use an abundance of chemicals every day. As a result, increased concentrations of endocrine disruptive contaminants are likely to occur in the area's environment, where they may hinder and alter healthy endocrine function in many organisms, including humans and wildlife. Consequently it is critical that the endocrine disruptive potential of this environment be assessed.</p> <p>Methods/Materials To assess the area's endocrine disruptive potential, a study involving an environmental sampling of 20 male <i>Pleuronichtys verticalis</i> was conducted at five trawling sites (n=4 at each site) in the coastal waters off Greater Los Angeles. Four male <i>Pleuronichtys verticalis</i>, acquired from Dana Point, CA and acclimated in the lab, served as controls. All 24 <i>Pleuronichtys verticalis</i> were blood sampled and assayed for vitellogenin, an egg yolk protein not typically produced in males, but inducible in the presence of endocrine disruptive estrogenic stimuli.</p> <p>Results The results show that: ·Environmental exposure to the urbanized coastal waters off Greater Los Angeles results in endocrine disruption. ·The endocrine disruptive potential is uniformly distributed over the coastal waters.</p> <p>Conclusions/Discussion From this it can be concluded that there is an elevation of endocrine disruptive activity in the environment surrounding Greater Los Angeles. Possible errors limiting the accuracy of these conclusions include: ·Inadequate sample size. ·Inadequately quantifiable data ·Questionable sourcing of controls</p>	
Summary Statement An analysis of the endocrine disruptive potential of the coastal waters off Greater Los Angeles.	
Help Received Steven M. Bay of Southern California Coastal Water Research Project for providing laboratory equipment and space to house and complete project.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Maria I. Rangel	Project Number S0419
Project Title What Is the Effect of Heat on the Extraction of DNA from Fruit Flies (Drosophila) Larvae Cells?	
Objectives/Goals What affect dosetemperature variation has on fruit flies (Drosophila) Larva Cells, so that DNA could be extracted from them? How many times will be necessary to isolate the DNA from the Gel and cut it with restiction enzymes, in order to get clear DNa bounding pattern?	
Abstract Methods/Materials You swatch ~80 larva fruilit flies until you get a soup. Put the substance in a beaker. Place another beaker in top of the first add water and a thermometer, and test the six different temperatures. Centrifuge 2 ml of the substance. Keep centriguge it in until you have layesr. Remove the aproppriate layer then add 10% of SDS and 80 ul of NaCl. Also add 567 ul of TE Buffer. Centrifuge it, and add 70% of Ethanol and 96% of Phenol. Performe the electrophoresis and the DNA fingerprints to determine if DNA was present or not in the DNA spource..	
Results I tested six different temperatures. At 168F DNAwas not present. At 173F DNA is not present seen before and after the electrophoresis. At 178 DNA was not present. At 183 F DNA was present. At 188 F DNA was present. At 193 F DNA was present. In this temperature there was a large particle formed in the bottom and middle of the centrifuge. This test was the test that I used to perform the restiction Digest. When I added the Eco RI to the DNA I saw that the molecule was separating in to smaller fragments	
Conclusions/Discussion Heating the DNA at hiht temperatures increases the posibility to extract DNA more efficiently. From the information that I have aquire at high temperatures the DNa molecule becomes denatured. The bonds between molecules bases are broken and the DNA ladder fall apart. This in fact has been my hypothesis, in which it was supported by trhe results in the isolation of the six different temperatures. It was necessary to cut the DNA with the restiction enzymes two times.	
Summary Statement My purposes of my experiment was to extract the DNA out of fruit flies larvalcells, and test the DNA at different temperatures, to see what was the effect.	
Help Received Mrs. Duran with research	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Oksana A. Sergeeva	Project Number S0420
Project Title Effect of Thiols on Transformation of dUMP into dTMP	
Abstract Objectives/Goals Thymidylate synthase (TS) catalyzes the reaction transforming dUMP into dTMP in cells. In cancer cells, TS is expressed at an increased level. While usual drugs treat cancer by inhibiting TS, the NewBiotics# drugs are substrates of TS that produce toxic products upon TS catalyzed reaction. At NewBiotics different thiols were added to the reaction of (E)-5-(2-Bromovinyl)-2'-deoxy-5'-uridyl monophosphate (BVdUMP) catalyzed by TS. The two non-natural ones, mercapotoethanol (ME) and dithiothreitol (DTT), worked to give desired toxic products. The natural thiol found in cells, glutathione (GSH), did not support the reaction. In the current work I studied the natural reaction of the conversion of dUMP into dTMP catalyzed by TS in the presence of different thiols to explain their effect on the BVdUMP reaction. Methods/Materials To evaluate the effect of thiols on the enzyme I studied the natural TS reaction converting dUMP into dTMP in the presence of six thiols: mercapotoethanol (ME), dithiothreitol (DTT), glutathione (GSH), N-Ac-cysteine (Ac-Cys), cysteine (Cys), and cysteine methyl ester (Cys-OMe). I used High Performance Liquid Chromatography to monitor the reaction. Results Four of the thiols (ME and DTT, and # to a lesser extent # Cys, and Cys-OMe) supported the reaction, and in the presence of the other two thiols (GSH and Ac-Cys) there was no reaction. After identifying the thiols that worked, I changed the enzyme concentrations to get more accurate results. I then chose to study Cys-OMe further, because it was natural and supported the reaction, and tested this thiol at different concentrations in the reaction. I found out that the higher concentration of the thiol, the less amount of dTMP was formed. I also preincubated the TS and Cys-OMe, and I found out the amount of dTMP formed was decreased. Conclusions/Discussion I concluded that all of the thiols I tested with the exception of DTT and ME inactivate TS.	
Summary Statement The TS catalyzed reaction of the NewBiotics' drug did not occur in the presence of GSH because certain thiols, including GSH, inactivate the enzyme.	
Help Received Used lab equipment at NewBiotics, Inc under the supervision of Dr. Maria Sergeeva	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Kedar Shah	Project Number S0421
Project Title Identification of Single Amino Acid Mutations in the p53 Gene of Drosophila melanogaster	
Abstract Objectives/Goals The goal of this study is to identify single amino acid mutations in the p53 gene of Drosophila melanogaster. Methods/Materials DNA from several fly strains, some with dominant negative p53 gene cDNA genomic insertions, was extracted. Primers were used to selectively amplify the gene of interest through PCR. After verifying the success of the reaction through gel electrophoresis, the next step was to clone the gene into a vector. Using vector-specific primers, the gene from various strains was sequenced and mutations were identified through computer analysis. Results The gels for each of the five attempts at DNA extraction and amplification either contained smeared bands or no bands at all. Therefore, recombination of the gene of interest into vectors was not attempted. Instead, plasmids containing the gene of interest were transformed into cells, which grew and multiplied. Large quantities of the plasmids were extracted from the cells using a Qiagen midi-preparation. A gel of the plasmids after the midi-preparation showed bands at the correct size. PCR with a new set of primers was then run on the plasmids containing the gene of interest. A gel for this reaction showed multiple bands at 1025 base pairs for each sequence. Results of the sequencing are still pending. Conclusions/Discussion Five attempts to extract and amplify the mutated cDNA p53 sequences from the Drosophila genome were unsuccessful because the primers used were designed for a region outside the Dmp53 cDNA sequence engineered into the flies. A new set of primers was used for the PCR of the gene of interest contained in plasmids. The success of this step verified that the same gene-specific primers could potentially be used to sequence the midi-prepared plasmids. If successfully found, the mutated sequences of the Dmp53 cDNA can be compared to the standard wild type sequence to identify single amino acid mutations. These single amino acid mutations cause dysfunction in Drosophila melanogaster. Because Dmp53 is homologous to human p53, a gene whose mutations are the single most frequent cause of human cancer, understanding the cause and effect of single amino acid mutations in Dmp53 is instrumental in understanding the nuances of human p53.	
Summary Statement My project focuses on finding mutations in a strain of fruit flies that are genetically engineered with the p53 gene, which functions as a tumor suppressor in both fruit flies and humans.	
Help Received Received guidance from teacher Tanya Buxton and mentor Vikash Bhagawandin; Obtained fly strains from Mike Olmann of Exilixis Inc.; Obtained primers from Parkash Jhurani of Genentech Inc.; Sequencing performed by Dhaya Seshasayee at Genentech Inc. and Vikash Bhagawandin at UCSF.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Leslie L. Sheu	Project Number S0422
Project Title BDNF Expression in the Aged and AD Brain and Its Expression in Relation to Cytoskeletal Proteins	
<p style="text-align: center;">Abstract</p> <p>Objectives/Goals My objective is to better understand Alzheimer's disease through fluorescent immunohistochemistry and double labeling. 1) Establish the distribution of BDNF (brain-derived neurotrophic factor) in the control (normal) aged brains and late AD brains. 2) Show changes in cytoskeletal aspects from normal to AD brains. 3) Find a correlation between BDNF and cytoskeletal proteins. I hypothesize that both BDNF and cytoskeletal labeling will decrease from aged to AD brains.</p> <p>Methods/Materials Major materials used include: human brain tissues (frontal cortex), vibratome, vacuum, orbit shaker, fluoroboxes, microscope slides and coverslips, fluorescent microscope, mercury lamp, permafluor, and primary and secondary antibodies. Completion of my experiment involved 4 basic steps. 1) Obtain brain cases (6) from the tissue repository at UCI and cut them into thin slices using a vibratome. 2) Use fluorescent immunohistochemistry and double labeling to label BDNF and cytoskeletal structures (including washes, primary antibody, and secondary antibody) 3) Mount tissues on slides (8 tissues per case x 6 cases = 48 tissue samples) and coverslip them. 4) View all slides under a fluorescent microscope and determine levels of BDNF and cytoskeletal proteins.</p> <p>Results Trends are exhibited. The amount of BDNF decreased slightly from normal to AD brains while the amount of cytoskeletal proteins increased more dramatically. There was a negative correlation between BDNF and cytoskeletal proteins.</p> <p>Conclusions/Discussion BDNF and cytoskeletal proteins both play crucial roles in AD. BDNF is lost with aging, but those who lose too much of this neurotrophin develop AD. Cytoskeletal proteins are increased because as AD attacks, more proteins are required to maintain structure and rigidity. My hypothesis was only partially correct, as I predicted correctly that BDNF levels would decrease but while I thought cytoskeletal amounts would decrease, the results show that they increased.</p>	
Summary Statement My project is about differences between normal and AD (Alzheimer disease) brains, focusing on the changes in BDNF (brain-derived neurotrophic factor) and its relationship to cytoskeletal aspects.	
Help Received Used equipment at the Institute of Brain Aging and Dementia of the University of California, Irvine, under the supervision of Dr. Paul Adlard	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Ellen J. Shilgevorkian	Project Number S0423
Project Title Confirming the Identity of an Unknown Piece of DNA by Restriction Mapping	
Abstract Objectives/Goals The purpose of the experiment is to show the basic and at times advanced functions of DNA such as replication, translation, transcription, through the study of DNA sections, that have been catalyzed with DNA restriction enzymes (a process called DNA restriction). The purpose of this project is to bring a better understanding of the universal hereditary material along with the benefits of contemplating such a unique macromolecule and its effects on human life, culture, and knowledge. By studying small sections of the material, this project hopes to bring some sense of resolution. Methods/Materials 1 micro centrifuge tube of lambda DNA; 1 micro centrifuge tube of HindIII; 1 micro centrifuge tube of EcoRI; 1 micro centrifuge tube of KpnI; 1 micro centrifuge tube of BamHI; 1 micro centrifuge tube of Bovine Serum Albumin; 1 bottle of TBE running buffer; 1 microcentrifuge tube of Buffer E; 1 microcentrifuge tube of Buffer C; 1 bottle of 1% Agarose; 1 bottle of H ₂ O; 6X Loading Dye; Wax Paper; Ethidium Bromide; Electrophoresis Chamber; Power Supply; UV Box and Camera; Micropipets; Micro centrifuge tubes; Rubber gloves; Lab coat; plastic cover; sharpie markers. Results After the digestion process, the double digests resulted in what was expected. The DNA digested EcoRI and KpnI resulted in a bp fragment in between the 100-200 markers. The DNA digested with KpnI and HindIII resulted in a bp fragment a little over the 400 markers. The DNA digested with EcoRI and HindIII resulted in a bp fragment in between the 500 and 600 markers. Conclusions/Discussion The hypothesis was proven to be correct. The experiment resulted with the appropriate base pairing observed in the picture. With the appropriate resulting of base pairs, it can be concluded that the piece of DNA was a 3BHS clone. The KpnI site divided the insert unevenly into ~170 bp and ~400bp regions. By this, my assumption about the insertion with orientation from 5' to 3' was correct also. Thus, showing that the KpnI site was closer to the EcoRI site.	
Summary Statement To figure out the unknown.	
Help Received used lab equipment at California State University Los Angeles under the supervision of Charly.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) David T. Vu	Project Number S0424
Project Title Can Paper Chromatography be Used to Identify Different Species of Plants?	
Abstract Objectives/Goals The objective is to determine if plant pigments were distinctive for each species. Methods/Materials Leaves of different plants were obtained. Residue from leaves was applied onto strips of chromatography paper by placing the leaf on top of the strip of paper and rolling a coin back and forth on top of the leaf. This step was repeated around ten times for each leaf. Once the strips were prepared, chromatographs were run with a solvent containing 9 parts ether to 1 part acetone. The test ran for 30 minutes on the shorter strips and up to an hour and a half on the longer strips. Results The results on the same kind of leaves did not remain constant. Multiple test were performed and the pigments did not deposit at the same spot. Conclusions/Discussion Using chromatography to recognize plant pigments is not possible. The test produced differnt results every time the same leaf was run.	
Summary Statement Using paper chromatography on plant leaves to see if a recognizable pattern would appear every time.	
Help Received Mr. Jones gave me the name of the speices of plants that I used.	



**CALIFORNIA STATE SCIENCE FAIR
2002 PROJECT SUMMARY**

Name(s) Kevin R. Yackle	Project Number S0425
Project Title The Effect of Directing Ribozymes to Attack Proliferating Cell Nuclear Antigens and Inhibit the Growth of Cancer Cells	
Abstract Objectives/Goals My project was to determine if by specifically directing a ribozyme to attack the Proliferating Cell Nuclear Antigen (PCNA mRNA), will the growth of a cancer cell be inhibited and thus kill the cell; and if the cells died, to determine the origin of their death. Methods/Materials A total of eight rat and human brain cancer cell cultures were treated with either ribozyme, ribozyme and lipid, dysfunctional ribozyme and lipid, c2 cerimide, or nothing. The treatments were cultured and WST-1 dye was added to be metabolized in the cells. The metabolic rate was determined by an ELISA plate reader, which read how much dye was in the cells. A different culture of cells had PI dye and ANNEXIN-5 dye added to the treatments. These were then read by flow cytometry to determine whether the cells died through necrosis or apoptosis. Results When compared to the control cells, the cancer cells which had the ribozymes and a specific lipid added seemed to have a reduction in growth, as much as 24%. However, the cells that had a dysfunctional ribozyme and lipid also had a reduction in growth, up to 18% when compared to the normal cells. In the second assay, when the mode of death was determined, our control worked. The cells with the c2 cerimide, a chemical know to induce apoptosis, did in fact die through apoptosis. The cells that had no treatment were still living, however the cells with all other treatments died through necrosis and not apoptosis. Conclusions/Discussion Although the first assay appeared to have succeeded in reducing the cancer cell count, the treatments that had the largest death rate were due to the toxic lipid that was added to them. Also, the naked ribozyme treatment did reduce the growth of the cancer cells, which means that although the RNA piece is small, it will be taken into the cell. Less exciting was the death of the brain cancer cells, which appeared to be mainly through necrosis. This means the cell lyses or bursts, which can cause severe swelling. However, the control c2 cerimide treatment induced apoptosis assuring me that the procedure I went through was correct and can be repeated in later experiments.	
Summary Statement My project involved using a particular known catalyst, called ribozyme, and directing it to attack PCNA, a transcription factor necessary for polymerase to proofread the mRNA, thus creating shorter mRNA fragments and cancer cell death.	
Help Received Dr. Joan Robbins and Mr. Eric Alspaugh, Immusol, Inc. for direction and assistance; Dr. Carol Kruse at Univ. of Colorado for consultation.	