



**CALIFORNIA STATE SCIENCE FAIR  
2004 PROJECT SUMMARY**

<b>Name(s)</b> <b>Sumail S. Bhogal</b>	<b>Project Number</b> <b>J0401</b>
<b>Project Title</b> <b>The Effects of pH upon Living Tissues</b>	
<b>Objectives/Goals</b> Problem: To determine the resistance of homogenates to pH fluctuations at various temperatures. Hypothesis: My hypothesis is that the most resistant buffer will probably be the liver homogenate at cold temperature.	
<b>Abstract</b> <b>Methods/Materials</b> Materials Needed: 1.Beef Liver Tub; 2.Two Potatoes; 3.Paring Knives; 4.Weighing Balance; 5.Commercial Electrical Blender; 6.Deionized Water; 7.Two 200 mL Beakers; 8.Two 50 mL Beakers; 9.Eight 10 mL Beakers; 10.Eight Graduated Cylinders; 11.Droppers; 12.0.1M NaOH; 13.0.1M HCL; 14.Digital pH Meter; 15.Water Bed; 16.Styrofoam Ice Bath; 17.Cubed or Granulated Ice; 18.Commercial Buffer Solution, pH 7; 19.Non- Lint Wipes; 20.Bulb Thermometers. Procedure: 1. Actual procedure: a.Took the 200 mL beaker in which the potato homogenate was poured and used the graduated cylinder to measure out exactly 10 mL of homogenate. b.Poured out a beakerful of 0.1M NaOH and 0.1M HCL. c.Using a dropper, poured drop by drop of NaOH and HCL into the solution and used the pH meter to measure the pH of each drop added. Recorded results for each and every drop. d.After the tenth drop, put 5 drops instead of one and recorded results three times. e.After recording results with HCL, calibrated the meter by washing it in water, dipping it in buffer solution pH 7 and then resetting the pH to standard 7. f.Resumed the procedure with NaOH and then redid the entire procedure with liver after re-calibrating. g.Again did the procedure, just with different variables such as having the solution in water at the freezing point and also water at body temperature. h.Later on, compared all of the results and came to the conclusion that the most effective buffer was liver in cold water.	
<b>Results</b> As I had hypothesized, the most effective buffer that kept the pH from fluctuating greatly was the liver homogenate at the freezing point. I have come to the analysis that the colder the temperature is, the less the fluctuation there is in the pH. Then second most effective was the liver at the room temperature. After that were the potato at the freezing point and then the potato at room temperature.	
<b>Conclusions/Discussion</b> From the beginning, my hypothesis was that the liver in the freezing point would be the most effective and my results have proved my hypothesis correct, the most effective buffer is liver homogenate at the freezing point.	
<b>Summary Statement</b> In my project I attempted to determine which animal or plant homogenate was the most effective in various temperatures to pH changes.	
<b>Help Received</b> Used lab equipment under supervision of Dr. Allan Tannabi at Cal State University of Bakersfield.	



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<b>Name(s)</b> Sean E. Browne	<b>Project Number</b> <b>J0402</b>
<b>Project Title</b> <b>Fungus Among Us: How Yeast-Powered Balloons Reveal Secrets of Fermentation</b>	
<p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b> The objective is to learn more about fermentation by observing how yeast, a common fungus used in the fermentation process, reacts with different liquids.</p> <p><b>Methods/Materials</b> When fermentation occurs, carbon dioxide is released. The experiment was designed to measure the amount of carbon dioxide released by eight different liquids that were mixed with active dry yeast and put into bottles. Balloons were attached to the tops of the bottles. As carbon dioxide was released from each mixture, the balloons expanded and the circumference was measured.</p> <p><b>Results</b> Apple juice released the most carbon dioxide in 80% of the experiments. On average, substances containing sugar or fructose also released higher levels of carbon dioxide</p> <p><b>Conclusions/Discussion</b> My hypothesis was that previously fermented substances would release more carbon dioxide when combined with yeast. However, the results suggest that sugar plays a much more important role in the process of fermentation. The experiment demonstrates that yeast alone does not result in fermentation, but must react with sugar in order to begin the fermentation process.</p>	
<b>Summary Statement</b> To determine how yeast reacts with various substances by measuring the release of carbon dioxide.	
<b>Help Received</b> Science teacher advised on process and timelines. Mother took me to buy materials for experiments. . Father helped me glue my materials to the board.	



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<b>Name(s)</b> <b>Derick Choi; Neal Kawas; Tal Lorberbaum</b>	<b>Project Number</b> <b>J0403</b>
<b>Project Title</b> <b>Screening for Strong Gene Promoters</b>	
<p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b> The objective of our project is to screen for strong gene promoters from the natural environment that will help protein production in Streptomyces. We are doing this experiment in hopes of finding a new industrial or pharmaceutical use for our newly discovered DNA sequence. Hypothesis: Genes need to have promoters to eventually be translated into proteins. We believe that nature has promoters that will increase protein production in Steptomcyces. We think that we can find one of these promoters and isolate it from nature.</p> <p><b>Methods/Materials</b> Materials and Equipment: Soil samples from Adobe Creek, Stanford hills, and Genecor lawn; Plastic bags, spatulas, flasks, spreader, tips, petri dishes, test tubes, gel blocks, pipette man, gloves, protection glasses, microscope, UV camera, electrophoresis apparatus, ventilation hood, refrigerator, water bath, centrifuge; Streptomyces, restriction enzymes, ligase, vector DNA; Bacteria transformation solutions, DNA isolation kit. Procedure: We collected soil samples from three different environments. Then we grew the bacteria in a rich medium overnight and harvested it for the isolation of bacterial DNA by using the centrifuge. The DNA was digested into small pieces with restriction enzymes. We inserted them into our vector containing a promoterless reporter gene, but have an activator gene for producing red pigment, a type of antibiotics, on Streptomyces colonies. We transformed the recombinant DNA into Streptomyces and grew them on antibiotic agar plates. We did a negative control that had no DNA in the vector. We isolated the vector from the red colony containing the promoters. We sequenced the foreign DNA and did a BLAST search to find out what organisms and genes these promoters come from.</p> <p><b>Results</b> When we finished our experiments we saw one red colony and one pink colony. The sample was from the water environment. The dark red colony contained a stronger promoter than the one with a pink colony. We then did a BLAST search on the red colony, which revealed that out of our 846 base pair sequence, our promoter was approximately 450 bp long. The promoter was undiscovered.</p> <p><b>Conclusions/Discussion</b> We can conclude that strong promoters do exist in nature. Our results showed that colonies did have red which meant that the antibiotic was produced. This tells us that a relatively strong promoter was present that can work in Streptomcyces.</p>	
<b>Summary Statement</b> The objective of our project is to screen for strong gene promoters from the natural environment that will help protein production in Streptomyces.	
<b>Help Received</b> Used lab at Genecor Int., Inc. under the supervision of Dr. Wei Liu	



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<b>Name(s)</b> <b>Lauren E. Christensen</b>	<b>Project Number</b> <b>J0404</b>
<b>Project Title</b> <b>Your Kitchen Is Your Lab: Extracting Onion DNA</b>	
<b>Objectives/Goals</b> My project objective was to extract a DNA sample from an onion in my kitchen using materials commonly found at home. The goal of my project was to perform the experiment, extract the sample and then to get confirmation that I had been successful.	
<b>Abstract</b> <b>Methods/Materials</b> an onion, 1tsp salt, 1/4c of liquid dish-washing soap, 1/8tsp of meat tenderizer (available at supermarkets), 1 16oz bottle of chilled rubbing alcohol, 1/4c warm water, 1 coffee filter paper, wooden toothpicks, knife and chopping board, mesh strainer, handheld blender, small container, glass measuring cup, basic microscope (I used a Meade model 9260), glass slides, kitchen timer, measuring spoons and cups, rubber spatula. Method: Peel and cut 1/2 of the onion into small pieces. In glass measuring cup dissolve measured salt into measured tap water. Add chopped onion. Using hand blender, blend for 3 seconds. Put bottle of alcohol into refrigerator. Transfer onion mixture into small container. Add measured amount of dish soap. Using a toothpick, stir this mixture for 5 minutes (set timer or have someone help you). Line mesh strainer with the coffee filter and set over glass container. Pour onion mixture into filter to strain. This will take about 30 minutes. Remove filter with remaining onion, discard. Add meat tenderizer to the strained/filtered onion goop in a clean glass measuring cup. Set timer and stir with clean toothpick for 5 minutes. Note the amount of goop in the measuring cup. Remove chilled alcohol from refrigerator and slowly, without stirring, pour in an amount equal to onion goop. DNA will float up through alcohol. Swirl DNA onto clean toothpick. It will look like clear snot. Smear sample on clean glass slide. Look at sample with microscope or high powered magnifying glass.	
<b>Results</b> Using the procedure set forth on the web site, I was able to remove what seemed to be a DNA sample.	
<b>Conclusions/Discussion</b> I sent out several emails to various professionals asking for help in determining if my experiment really worked. Dr Linda Walling, Associate Dean of Biological Science from UCR-Riverside responded and agreed to look at what I had. During our meeting I explained my experiment and showed her pictures I took through my microscope of my slides. She confirmed that my sample really was DNA. My conclusion and what I learned from this experiment is that you can extract DNA from an onion using common items found at home in your own kitchen.	
<b>Summary Statement</b> How to extract DNA from an onion in your kitchen.	
<b>Help Received</b> Mother helped type and photograph me while I did experiment, Professional opinion Dr. Linda Walling, Professor UCR Riverside	



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<b>Name(s)</b> <b>David L. Darbonne</b>	<b>Project Number</b> <b>J0405</b>
<b>Project Title</b> <b>Apoptosis: The Other Way Cells Die</b>	
<b>Abstract</b> <b>Objectives/Goals</b> The purpose of my project is to see how much Apo2L and Fas Ligand protein it takes to kill Jurkat cells by apoptosis in culture. If the Jurkat cells are not very sensitive to Apo2L or Fas Ligand, then to see if cross-linking each protein will help it kill the cells. <b>Methods/Materials</b> I added Apo2L and Fas Ligand protein, at different concentrations, to Jurkat cells in culture. I also added different amounts of an antibody that can connect or cross-link the protein molecules together. After incubating the cells with Apo2L, Fas Ligand and the antibody for 18 hours, I added alamar blue dye to the cells. Four hours later, I measured fluorescence from the alamar blue dye with the cells using a fluorescence plate reader. This let me know if the cells died or were still alive. <b>Results</b> I found that Apo2L or Fas Ligand alone could not kill the Jurkat cells, but when I added the cross-linking antibody with Apo2L or Fas Ligand, the cells died. The cells died the most when I added 6.25 ng/ml or more of either Fas Ligand or Apo2L with the cross-linking antibody. <b>Conclusions/Discussion</b> Apo2L, Fas Ligand or the crosslinking antibody alone did not kill the Jurkat cells. A combination of the antibody with Apo2L or Fas Ligand did kill the Jurkat cells. From this I learned that it is not only the amount of Fas Ligand or Apo2L that is needed to kill the Jurkat cells, but it is also how the Fas Ligand or Apo2L is presented to the cells. My project conclusions can be useful for scientists exploring the use of apoptosis proteins for cancer treatments.	
<b>Summary Statement</b> My experiments helped me find out how much Apo2L or Fas Ligand it takes to kill Jurkat cells by apoptosis in culture, and if this apoptosis needs each ligand to be cross-linked to kill the cells.	
<b>Help Received</b> My father taught me how to culture the Jurkat cells and make dilutions of the proteins I used. He also showed me how to use the plate reader. Genentech, inc. provided the cells, proteins and equipment. I performed all experiments for my project.	



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<b>Name(s)</b> <b>James A. Fraser</b>	<b>Project Number</b> <b>J0406</b>
<b>Project Title</b> <b>Digestion Is the Question</b>	
<b>Objectives/Goals</b> For my project, I recreated the digestive system and digestive enzymes. I wanted to see if starch could break down and combine with glucose. If this does happen then it proves that not only glucose, but starch travels through your bloodstream and gives vitamins and minerals to your cells. I was also trying to find out if both starch and glucose could break down without without digestive juices and acids. I also decided to test real foods with various levels of starch we as humans eat daily (potatoes, corn, Cheerios, pasta, and white bread).	
<b>Abstract</b> <b>Methods/Materials</b> Cornstarch, two slices of white bread, half of a cup of any type of pasta, half of a cup of corn kernels, half of a brown potato, half of a cup of Cheerios, distilled water, 1-pint jar with a lid, Glucose enzymatic strips (glucose test strips), starch enzymaticstrips (starch test strips), sugarless apple juice, and 6-foot long (30 cm) sausage casings.  First collect all of your materials. After you have done that, assemble your experiment. First fill your sausage casing with 1/4 of a cup of the starch solution, 1/4 of a cup of sugarless apple juice, and 1/4 of cup of distilled water. Fill it until only one inch is not filled, and then tie the end very tightly. Fill the jar with your distilled and place the sausage casing softly on the bottom of the jar and leave it. After ten minutes, check the water for glucose, and starch. Repeat this procedure for the next hour. Repeat this test with each of the six foods(pasta, white bread, Cheerios, potatoes, and corn).	
<b>Results</b> My results turned out to show that all of the six tests released glucose and starch during the one hour period. The tests released these substances at different times because of their textures.	
<b>Conclusions/Discussion</b> For all of the six tests my hypothesis was wrong. The starch was shown to be "digested" and broken down. The glucose, starch, and distilled water were all able to escape the semi-permeable membrane. This lead to shrinkage in the sausage casing because like your intestines it gave the glucose and starch to the body fluids to be transported to your cells for energy.	
<b>Summary Statement</b> Can starch break down small enough and fast enough to exit a semi-permeable membrane in the second stage of digestion?	
<b>Help Received</b>	



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<b>Name(s)</b> <b>Rebecca L. Kaspar</b>	<b>Project Number</b> <b>J0407</b>
<b>Project Title</b> <b>GENEalogy: Tracing a Mutant Gene through My Family Line</b>	
<b>Abstract</b> <b>Objectives/Goals</b> The purpose of this study was to find out how many of my relatives have a prothrombin gene mutation that my father carries. Do my relatives and I also carry the mutant gene, and if so, is the mutation passed on from parent to child? I hypothesized that 50 percent of my family members would have the prothrombin mutant gene. <b>Methods/Materials</b> Materials: 50 ml and 1.5 ml tubes, Cheek cells, PBS, dNTPs for Taq polymerase, Taq polymerase, Thermocycler, Agarose gel, Buffer with salt, DNA standards, HindIII enzyme, Centrifuge, DNA size markers, Ethidium bromide, Electrophoresis apparatus, Ultraviolet light, DNA oligonucleotides, Loading dyes. Methods: Swish mouth with PBS. Isolate the DNA. Make the agarose gel. Set up Polymerase Chain Reaction (PCR). Cut with HindIII. Separate the DNA fragments on the agarose gel until the dye is almost to the bottom. The gel showed one or two bands, depending on whether the piece of DNA had been cut once (wildtype) or twice (mutant); this showed that the person had the mutant gene or if both genes were wildtype. <b>Results</b> The results showed that my grandfather, 4 of 6 aunts and uncles, and 5 of 13 cousins all had the prothrombin gene mutation. My fathers mutation was not a spontaneous mutation and was a dominant gene. <b>Conclusions/Discussion</b> In conclusion, I discovered that 48 percent of my family carry the prothrombin mutation, which proved my hypothesis.	
<b>Summary Statement</b> I traced a prothrombin gene mutation through my family line.	
<b>Help Received</b> Roger Kaspar drove me to the lab many times and taught me the correct use of the equipment. Brian Johnston provided the equipment. BioRad and SomaGenics donated some of the materials.	





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<b>Name(s)</b> Genevieve (Jenny) H. Kromm	<b>Project Number</b> <b>J0408</b>
<b>Project Title</b> <b>Bread Battle: Wild Yeast vs. Commercial Yeast</b>	
<b>Abstract</b> <b>Objectives/Goals</b> The purpose of my project is to determine whether wild yeast starter contributes to a better loaf of homemade bread, comparing the results to a homemade loaf of bread made with commercial yeast. My hypothesis was that the wild yeast starter would result in overall better bread results. I decided to measure height, weight, porosity, taste, texture, and smell of the wild and commercial yeast breads to test my hypothesis. <b>Methods/Materials</b> My materials and the procedures consisted of four (4) parts: Part I: Growing the Wild Yeast; Part II: Testing the Wild Yeast; Part III: Baking the Wild Yeast Bread; and Part IV: Baking the Commercial Yeast Bread. <b>Results</b> My hypothesis was not correct. The commercial yeast bread was more successful than the wild yeast bread. The average height of the commercial yeast bread was higher than the wild yeast bread. The average porosity was also greater in the commercial yeast breads than in the wild yeast breads. The weight of the wild yeast bread was heavier than the weight of the commercial yeast bread, and I think this connects to the wild yeast bread's low porosity. The commercial yeast bread kept a consistent 908 grams through all three tests. The wild yeast bread's average was 1210.6 grams, making wild yeast bread's score on weight greater. Finally the taste, texture and smell were better from the commercial yeast bread. <b>Conclusions/Discussion</b> I figured out why my first 2 tests of wild yeast starter didn't rise well. In Part II, the balloon did not rise like it is supposed to, and I couldn't understand why until I left the bottle with the balloon overnight. In the morning, the balloon was fully inflated, which led me to conclude that wild yeast starter takes longer to digest food and produce gases than commercial yeast! My research showed the purpose of commercial yeast was not for better bread, but faster rising. At the time, I assumed that both yeasts took the same amount of time to produce gas. Now I know wild yeast needs 24-72 hours! That's one reason why the wild yeast starter did not rise as well. My conclusion is that wild yeast starter did not give as successful bread results as commercial yeast. If I changed my bread machine to a different time frame that would allow the starter to take time to produce gas, then maybe the results would have leaned more toward the wild yeast starter. Thank you for reading about my project!	
<b>Summary Statement</b> I grew wild yeast starter and tested its rising/baking results in bread against rising/baking results in bread made with commercial yeast.	
<b>Help Received</b> Mother showed how to use bread machines.	





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<b>Name(s)</b> <b>Iris J. Liu</b>	<b>Project Number</b> <b>J0409</b>
<b>Project Title</b> <b>The Best Amount of Yeast for Dough Rising</b>	
<b>Abstract</b> <b>Objectives/Goals</b> The objective is to determine the best quantity of yeast for dough rising. I hypothesize that more yeast makes the dough rise larger and quicker. <b>Methods/Materials</b> Six pieces of dough were made at 70 degrees Fahrenheit in each experiment. A total of five experiments were conducted. Each piece of dough contained the same ingredients (8 tablespoons of sugar, $\frac{1}{4}$ teaspoon of salt, 2 cups of flour, and $\frac{1}{2}$ cup of warm water) but variable amounts of yeast. The first piece of dough had no yeast (the control), the second had one teaspoon of yeast, the third had two, and so on. The volume of each piece of dough was measured at every hour for four hours. The relation of different amounts of yeast, dough volume change and time was plotted. <b>Results</b> The dough with increasing amount of yeast rose larger and quicker in four out of five experiments, until a best quantity (four teaspoons) of yeast was reached. <b>Conclusions/Discussion</b> The best quantity of yeast for dough rising was four teaspoons per piece of dough. Before reaching the best quantity, adding more yeast made the dough rise better. Excess yeast did not appear to help dough rising; in contrast, it impeded the process, probably by exhausting nutrients and producing ethanol.	
<b>Summary Statement</b> This project focuses on identifying the best amount of yeast for dough rising.	
<b>Help Received</b> My parents helped with data analysis and science fair board.	



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<b>Name(s)</b> <b>Brizet Morales</b>	<b>Project Number</b> <b>J0410</b>
<b>Project Title</b> <b>Enteric Mystery</b>	
<b>Objectives/Goals</b> My goal was to find out whether enteric bacteria could be differentiated by biochemical tests. Since enteric bacteria cannot be differentiated by their physical characteristics or their morphology, I thought that they could maybe be differentiated by their chemical characteristics.	
<b>Abstract</b> I used four different types of enteric bacteria. These were <i>C. freundii</i> , <i>S. liquefaciens</i> , <i>E. coli</i> , <i>E. aerogenes</i> , and an unknown bacteria. My teacher/advisor provided me with these. The unknown bacteria was one of the other four cultures of bacteria. I wanted to see if I could find out what the unknown bacteria was by using four tests. These were the citrate utilization test, the hydrogen sulfide production test, the Vogues-Prosauker test, and the Methyl red test.	
<b>Methods/Materials</b> I used four different types of enteric bacteria. These were <i>C. freundii</i> , <i>S. liquefaciens</i> , <i>E. coli</i> , <i>E. aerogenes</i> , and an unknown bacteria. My teacher/advisor provided me with these. The unknown bacteria was one of the other four cultures of bacteria. I wanted to see if I could find out what the unknown bacteria was by using four tests. These were the citrate utilization test, the hydrogen sulfide production test, the Vogues-Prosauker test, and the Methyl red test.	
<b>Results</b> After reading the tests, I found that the unknown bacteria was <i>E. coli</i> . I found that enteric bacteria can be told apart by biochemical testing.	
<b>Conclusions/Discussion</b> I learned that biochemical testing is very important when trying to identify a new species of bacteria. If a new type of bacteria is found and it cannot be classified by its morphology or the Gram-stain Method, it can most likely be classified by its chemical characteristics.	
<b>Summary Statement</b> I wanted to see whether enteric bacteria could be differentiated by biochemical tests.	
<b>Help Received</b> My teacher provided me with the bacteria	



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<b>Name(s)</b> <b>Alexandra G. Moyzis</b>	<b>Project Number</b> <b>J0411</b>
<b>Project Title</b> <b>Swimming Backstroke in the Gene Pool</b>	
<p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b> Scientists have found a gene that may play a part in whether an athlete is a sprinter or excels at endurance events. This gene has been named ACTN3 and it makes a protein called actinin-3 that helps muscle cells re-power quickly. A change (mutation of C to T) in ACTN3 can cause the cell to stop building the actinin-3 protein, leaving it useless to the muscle cell. Without actinin-3, however, the athlete may be better at long distance events. Genes that are important to our survival as a species are also seen in animals related to us through evolution. Scientists have found the ACTN3 gene in other animals, but not the change (mutation of C to T) that makes its protein absent from the muscle cell. The goal of this science project is to see if the C to T change in ACTN3 is found only in humans or if it is found in other primates as well.</p> <p><b>Methods/Materials</b> Polymerase Chain Reaction (PCR), which makes many copies of the DNA of interest, coupled with DNA sequencing, was used to tell if the C to T change was found in the ACTN3 gene. General Materials: micropipets, pipettors, microcentrifuge tubes, test tube racks, centrifuge, ice bucket/ice. PCR: DNA, primers, DNA polymerase, deoxynucleotides, tris buffer, water, PE 9700 PCR machine. Gel Electrophoresis: agarose, tracking dye, gel apparatus, power supply. Gel Staining: pyrex dishes, ethidium bromide stain, water. DNA Sequencing: PCR product, deoxynucleotides, terminator nucleotides, DNA polymerase, water, ABI 3100 DNA Sequencer</p> <p><b>Results</b> Out of 5 human samples, three have both C and T at ACTN3, and two have only T. Out of 25 primate samples, 15 have only C, seven have both C and T, and three have only T.</p> <p><b>Conclusions/Discussion</b> The mutation (C to T) in the ACTN3 gene is found in primates as well as humans. I conclude that this mutation is not specific to humans, and likely originated millions of years ago. Usually, when a mutation causes the cell to stop building a protein the result is disease or even death. This protein stopping mutation in ACTN3, however, seems to be good, since many primates (including humans) have kept it.</p>	
<b>Summary Statement</b> I determined that a mutation that may play a part in whether an athlete excels at endurance events originated millions of years ago.	
<b>Help Received</b> This research was conducted in my mother and father's laboratory at the University of California, Irvine Medical School. Simin Hakim helped me run my PCR and DNA sequencing reactions.	



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<b>Name(s)</b> <b>Armen R. Perian</b>	<b>Project Number</b> <b>J0412</b>
<b>Project Title</b> <b>Who Done It? DNA Fingerprinting and Forensics</b>	
<p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b> Can DNA fingerprinting with the help of restriction enzymes help solve a crime scene mystery? If there are several suspects, how can the different DNAs be matched to that of the crime scene DNA? Also, can increasing the voltage in the electrophoresis chamber cause the DNA fragments to move faster and further on the agarose gel?</p> <p><b>Methods/Materials</b> Rehydrate DNA/buffer samples with 200 ul sterile water. Rehydrate EcoRI/PstI enzyme mix with 750 sterile water. Prepare electrophoresis buffer by adding 60ml of 50X concentrate to 2.94 liters of distilled water. Prepare 1% agarose gel by adding 1 gram of agarose powder to 100 ml of buffer. Pour agarose gels enough to cover the gel com teeth (0.5-0.75cm). On day 1 prepare DNA samples by adding 10ul of DNA to 10ul of enzyme mix. Incubate tubes overnight at 37C. On day 2 add 5 ul loading dye to DNA samples. Place agarose gel in chamber with 275 ml of buffer. Inject 10ul of DNA size marker (standard) into lane one well. Inject 20 ul of Crime Scene DNA into lane 2. Do same as CS for suspects. Electrophorese for 40 minutes. Place gel into staining tray, add 60 ml of DNA stain and let stay overnight covered at room temperature. Day 3; Pour off DNA stain and add 60 ml water and destain for 15 minutes. Pour off water and analyze.</p> <p><b>Conclusions/Discussion</b> Suspect 3 had the same DNA fragments as the Crime Scene DNA pattern for every trial. It was easier to figure this out by looking at the gels, than by comparing the base pair sizes on graphs which slightly overlapped with the other suspects. Some of the lighter base pairs traveled further than the last band of the marker, but I was still able to match the suspect to the crime scene. I did contaminate all of the DNA samples with suspect 3 DNA for trials 2-6, but that did not make a difference since the number of bands and distance is close to trial 1. Trial 2 at 200V gave the best results and band separation with identical distances traveled by CS and S3. Trial 2 worked best probably because all of the samples and reagents were fresh on first day. Increasing the voltage (IV) made the bands travel further (DV), but did not change the patterns, so the same suspect could be matched to the CS.</p>	
<b>Summary Statement</b> Restriction enzymes cut DNA of crime suspects into smaller fragments, which by electrophoresis can be matched to DNA found at a crime scene.	
<b>Help Received</b> My aunt let me test my experiment at her clinical lab. My mom helped me understand the biotechnology of DNA fingerprinting. My dad helped me with my graphs and excel.	



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<b>Name(s)</b> Michelle K. Reed	<b>Project Number</b> <b>J0413</b>
<b>Project Title</b> <b>Purified Antioxidants Protect DNA and Life from Oxygen Free Radicals</b>	
<b>Abstract</b> <b>Objectives/Goals</b> Oxygen naturally creates free radicals in our bodies and in water. Oxygen free radicals damage DNA and cause disease, cancer and aging. I tested such free radical DNA damage and planarian worm lifetime effects. I used a related series of pure antioxidants (Tannic, Ellagic and Gallic Acid) to test if they would block free radical DNA damage and protect the worms lifetime. <b>Methods/Materials</b> To copy free radical generation, speed up and control it, I am using the Fenton reaction, in which adding certain amounts of copper sulfate and hydrogen peroxide in water creates free radicals. I exposed DNA that I made in the lab using PCR and planarian worms to free radicals made in the Fenton reaction. I measured the oxidative damage to DNA by gels and worms by scoring. I used many Dilutions of fenton and antioxidant chemicals to find out their toxicity to worms. I used the lowest toxic concentrations of each Fenton chemical. I tested non-toxic concentrations of Gallic, Tannic, Ellagic acid, and Pomegranate juice. All antioxidants were tested to see if they could protect the DNA from damage and worms lifetime. <b>Results</b> Both Tannic and Ellagic acid protected both the worms lifetime and DNA from damage, but Tannic acid itself is toxic to worms. Gallic Acid protected the worms but not the DNA. <b>Conclusions/Discussion</b> Gallic , Ellagic and Tannic acid are a Family of antioxidants. Gallic is the single unit. Ellagic is two Gallics. Tannic is five or more Gallics. If More is better, tannic should be the best antioxidant. Tannic is toxic, maybe for the same reason it TANS leather. Ellagic is a very strong antioxidant protects the worms and DNA and is not toxic. Gallic is a good antioxidant by protecting the worms, but not the DNA. Gallic acid may be complementing antioxidant genes in the Worm.	
<b>Summary Statement</b> I am trying to find out which pure antioxidant protects DNA and Planarian worms from oxygen free radicals made in a Fenton reaction.	
<b>Help Received</b> Mother- Planarian worm care and note book format. Father- PCR, DNA, Computers, poster, Lab tools and supplies, practice speaking.	



**CALIFORNIA STATE SCIENCE FAIR  
2004 PROJECT SUMMARY**

<b>Name(s)</b> <b>Cameron B. Seebach</b>	<b>Project Number</b> <b>J0414</b>
<b>Project Title</b> <b>Do Snakes Have Legs? Electrophoresis of Leg Muscles in Lizards and Possible Leg Muscle Regions in Snakes</b>	
<p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b> The purpose of my experiment is to determine whether snakes have detectable leg muscle regions still existent from past legged ancestors.</p> <p><b>Methods/Materials</b> I took tissue samples from four dead snakes and two dead lizards. From the two lizards, I took front and back leg and jaw tissue samples. From the snakes, I took tissue from where front legs and back legs would be on a snake and from the jaws. Then I prepared the samples to be used in Tris-HCL (protein) Electrophoresis gels. I loaded my gels and ran them.</p> <p><b>Results</b> Once the gels had been run and stained, the banding patterns were very similar between the front and back leg gels of both the snakes and the lizards, but were completely different from the jaw gels.</p> <p><b>Conclusions/Discussion</b> Protein electrophoresis can never establish a genetic relationship between two species because proteins, unlike DNA have multiple configurations. For example, a protein could have an Amino acid sequence of UAG, but could also be UAU. Thus, proteins can never be used to prove directly that the snakes are related to the lizards. However, protein allows us to infer things about the muscles in a a designated sample. The banding patterns of the leg regions on the snakes being very similar to the banding patterns of the legs on the reptiles proves that there is still some muscle left over from past legs in an area that needs no more muscle than areas on other parts of the snake, but that area has more anyway. I believe this can be attributed to the previous bone structure of a leg that may have been there in an ancestor.</p>	
<b>Summary Statement</b> Have snakes evolved from a past legged ancestor?	
<b>Help Received</b> Used equipment at school lab under supervision of Mr. Mark Michail.	