Issue 4
May 2012
Sponsored by the HHCEF

The Hen Hud Hub Science Research Newsletter

Hendrick Hudson High School
2166 Albany Post Road, Montrose, NY 10548.

Special points of interest:

- Behavioral Sciences
- Neuroscience
- Environmental science
- Nutritional Sciences
- Biology and Medicine
- Genetics
- Engineering

Achievements of our Science Research Students, by Dr. C. Rogers

This year has seen a very talented group of students finishing the Science Research three years program. These students earned up to 12 SUNY Albany credits for their work, which was recognized 'significant and determining' by colleges reviewing their applications.

Students have demonstrated the ability to conduct independent research in a genuine professional environment, interacted with scientists, developed and conducted their own experiments, organized their findings, learned how to most effectively present and write–up their results and conclusions.

To qualify for competitions, each student wrote a full scientific paper with their results and conclusions, which was entered to the INTEL Science Talent Search and the Siemens competitions.

Tiffany Porras worked on the molecular pathways involved in the generation of cancerous tumors using a model of mammary stem cells in mice. She placed first as a speaker presenter in the Biology category at the Junior Science and Humanities Symposium (JSHS) for Westchester, Putnam and Rockland counties. and hereby qualified to compete at the New York State JSHS, which took place in Albany in March. Tiffany also competed with a poster at WESEF, Westchester Science and Engineering Fair, an Intel ISEF qualifying fair. She was awarded the Philips Award for Exceptional Research, awarded to projects that focus on improving the quality of people's lives.

Cassandra Rogers worked on identifying genetic markers for Schizophrenia using Induced Pluripotent Stem derived from Schizophrenia patients and allowed to differentiate into neurons in culture. Cassandra placed 2nd at NYSSEF, New York State Science and Engineering Fair, and 4th at WESEF, in the Cellular and Molecular Biology category.

Alexander Alves worked on identifying the habitat preferences of tropical fishes, which are brought by the Gulf Stream along the Long Island shore. Alex placed 2nd at JSHS with his poster presentation in the Environmental Science category. Alex received the "Visual Impact Award" for projects with the best visual representation of the research at WESEF.

Shivanee Shah worked on evaluating how taste perception for sweetness changes with estrogen levels in women, using Miraculin a protein found in Miracle fruit, a fruit which modifies perception of sour and sweetness. Shivanee received the Fisher Scientific Award, for outstanding projects in the field of behavioral sciences at WESEF.

Genetic association signals in schizophrenia map to long non-coding RNAs expressed in differentiating human neurons, by Cassandra Rogers (Intel paper excerpt)

Schizophrenia is a severe mental disorder affecting about one percent of the population. It is characterized by paranoia, hallucinations, disjointed behavior, and social aversion, and is diagnosed using criteria from the DSM-IV manual; however, it is an umbrella term and manifestations can range from social aversion to catatonia (Xu et al 2010). Onset is typically during adolescence and early adulthood.

A host of genetic, epigenetic, and environmental factors have been identified in its pathogenesis. Because of this heterogeneity and the influence of epigenetic and environmental factors, identifying candidate genes is difficult and complex. However, dozens of genes and chromosomal loci have been implicated using modern genetic tools such as Genome-Wide Association Studies (GWAS). These candidate genes include, but are not limited to, DISC1, ZNF804A, NRXN1, SMARCA2, and NRG1 (Millar et al. 2000; Stark et al. 2008; Donohoe et al. 2011; Koga et al. 2009;

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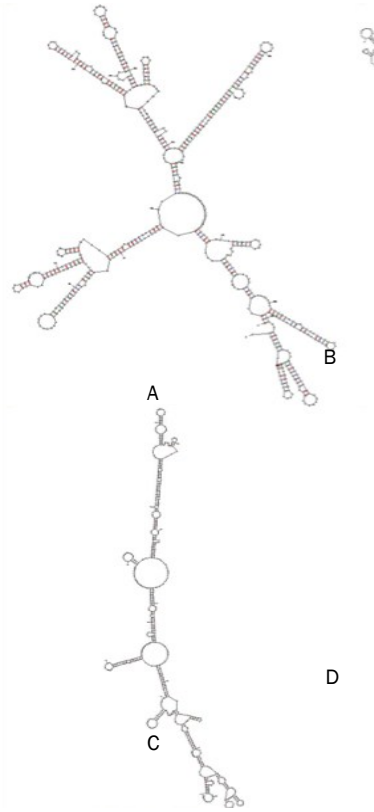


Images A, B, C, and D show MFOLD secondary structures of various splice variants of the long non-coding RNA RP11-319G6. Images [B] and [C] may have structures similar to that of some pre-miRNAs.

It is impossible to predict exactly what the secondary structure of these lncRNAs will be, and different programs produce a variety of results.

Pedrosa et al. 2010). A number of these candidate genes code for transcription factors. Due to the complexity of neuropsychiatric illness, however, many of these genes are characteristic in only a subset of patients, indicating that disruptions in a variety of genes may produce the same symptoms.

The means by which gene expression is analyzed in the developing brain, and more specifically, the brains of patients, are complex, expensive, and time-consuming, making the development of a genetic model of schizophrenia difficult. They also present many scientific limitations that make it difficult to draw specific conclusions regarding their molecular basis. For example, analyzing autopsied brain samples is confounded by cause of death, post-mortem delay, and the use of prescribed, legal and illicit drugs. There is increasing evidence that gene regulation may be a significant factor in the development of neuropsychiatric disease. Gene regulation is the result of a variety of contributing factors, including DNA methylation, histone modification, transcription factor expression, chromatin remodeling complexes, and expression of regulatory noncoding RNAs (Hsieh and Eisch 2010).

Genetic studies indicate that genes involved in neuronal differentiation may play a role in the development of a variety of neuropsychiatric disorders. Studying neuronal differentiation in neuropsychiatric disorders can be carried out using live human neurons grown in vitro. In the past, cell line and mouse models have to be used to study candidate genes involved in neuropsychiatric disorders. However, it would be extremely valuable to analyze expression in human neurons, in particular, neurons derived from patients with schizophrenia or other neuropsychiatric disorders. This has recently been made possible with the 2006 discovery of a technique to produce iPSCs. These cells are derived from terminally differentiated somatic cells, such as skin fibroblasts, that have been reverted back to a pluripotent state when transformed with viruses and plasmids containing the transcription factors Oct4, Sox2, Klf4, and c-Myc (Takahashi and Yamanaka 2006). iPSCs resemble embryonic stem cells, but offer the added benefit of having the genotype of the person whose fibroblasts were used. This provides the means to analyze patient-specific neurons, an extraordinary development in the field of neuropsychiatric genetics. Fibroblasts can be obtained from a patient with schizophrenia and reverted into the pluripotent state. iPSCs can then be induced to differentiate into neurons using a variety of growth factors (Brennand et al. 2011). Using iPSCs, it is now possible to track gene expression and epigenetic changes occurring during neural differentiation in vitro.

Regulatory noncoding RNAs, which include microRNAs (miRNA), small nucleolar RNAs (snoRNA), and long non-coding RNAs (lncRNA), are thought to be responsible for transcriptional and post-transcriptional regulation of a variety of genes, including those involved in neuronal development (Mehler and Mattick 2006). Approximately 20% of lncRNAs are known to interact with chromatin, indicating what could be an integral mechanism in regulating gene expression. Two novel lncRNAs, RP11-319G6.1 and RP11-586K2.1 were identified by this lab (Lin et al., 2011). Not only do they increase dramatically in differentiating neurons, but also contain single nucleotide polymorphisms (SNPs), rs893703 and rs12527359, respectively, that were identified in recent GWAS carried out in patients with schizophrenia. These SNPs are far from coding genes. RP11-319G6.1, which contains the SNP rs893703, maps to chromosome 3 within an intron of Retinol Binding Protein 1 (RBP-1) (Lin et al., 2011). This lncRNA increases significantly in differentiating neurons.

RP11-586K2.1, which contains the SNP rs12527359, maps to chromosome 8. Similar to RP11-319G6.1, its SNP is associated with schizophrenia and bipolar disorder (Sun and Kawaguchi, 2011; van Neerven et al., 2008).

The findings suggest that both RP11-586K2.1 and RP11-319G6.1 are involved in the development of a subset of patients with schizophrenia. This study examines the possible links between neuronal differentiation, RP11-319G6.1 and RP11586K2.1 expression, and schizophrenia. (...)

Discussion

Some 20% of all lncRNAs are known to regulate the expression of chromatin (Hung and Chang, 2010). However, the mechanisms through which they do this are mostly unclear. It is very possible that RP11-319G6.1 and RP11-586K2.1 function as chromatin modifiers. It is also possible that they are preliminary miRNAs (pre-miRNAs), which mature to become miRNAs, in which case they would function post-transcriptionally.

miRNAs influence gene expression by reducing mRNA levels of targeted genes. Primary miRNAs (pri-miRNA), which are lncRNAs, are processed by the Drosha/DGCR8 complex, after which they are exported from the nucleus. There, Dicer cleaves the pre-miRNA into a functional miRNA, which is approximately 22bp in length and contains a characteristic hairpin-loop structure (Murchison and Hannon, 2010).

The mature miRNA is then loaded into RISC (RNA-induced silencing complex), which binds to specific mRNAs, inducing their degradation or translational suppression (Murchison and Hannon 2004; Cai et al. 2010).

It is important to note that a significantly large subset of schizophrenic patients (approximately 1%) has microdeletions at the 22q11.2 locus. This region codes for DiGeorge Critical Region 8 (DGCR8), which associates with Drosha to guide the maturation of pri-miRNA into pre-miRNA (Drew et al. 2010). It is believed that loss of this critical component of the miRNA-processing complex may contribute to abnormalities in neuronal development and may lead to the onset of schizophrenia (Kim et al. 2010).

The data shown in the results section suggest that expression of both lncRNAs is decreased in iPS15-2 cells, which are known to have 22q11.2 microdeletions. The qPCR findings indicate a possible connection between the microdeletions at the 22q11.2 locus and the decreased expression of RP11-586K2.1. RP11-586K2.1 may be a pre-miRNA with significantly decreased expression in cells lacking the complex that processes them from the pri-miRNA stage to the pre-miRNA stage. An integral part of a complex that matures pri-miRNAs to pre-miRNAs is deficient in the iPS15-2 cells; without this complex, the pre-miRNAs it would normally produce are reduced. However, it is not yet known how reductions in RP11586K2.1 affect neuronal differentiation. Based on the results of this study, a grant proposal has been submitted by the lab to the NIH to study the effects of these two lncRNAs on neuronal differentiation.

The SZ39-1 line showed a highly increased level of expression of RP11-586K2.1 in day 14 neurons. The genetic factors that contributed to the development of schizophrenia in this subject are unknown. Unlike the iPS15-2 sample, SZ39-1 showed an increased level of expression of RP11-586K2.1. Genetic studies show that gene deletions and gene duplications in the same region can cause schizophrenia and autism. In other words, gene dosage—either over-expression or under-expression—can adversely influence brain development. Thus, the differences in the induction of the lncRNAs in this study seen in different schizophrenia samples are not necessarily contradictory. However, additional studies will obviously be necessary since the sample size in this preliminary study was small.

In the RP11-319G6.1 experiment, day 0 SZ97-7 neurons were compared to day 0 iPS15-2 neurons, and it was found that day 0 iPS15-2 neurons had a significantly lower level of expression of RP11-319G6.1 than did the SZ97-7 day 0 neurons. These data further highlight the lower expression of both lncRNAs in iPS15-2, the cell line with a 22q11.2 microdeletion.

Overall, these findings lend some support that these two lncRNAs, which map to regions of the genome identified in two GWAS studies are involved in a subset of patients with schizophrenia.

Conclusion

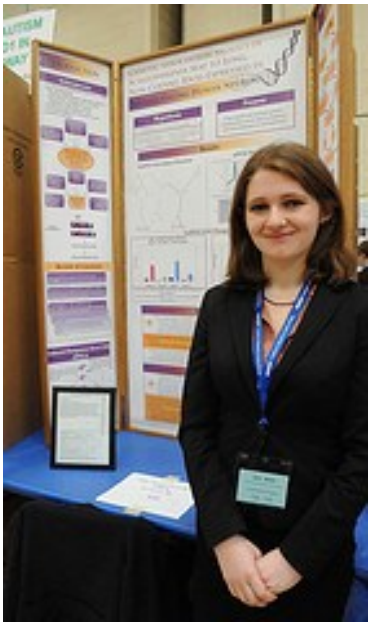
Research on neuropsychiatric disease has been hindered in the past by the incapacity to do effective research on differentiating human neurons. The controversy regarding embryonic stem cells has delayed their use as a means to study human neurogenesis. However, with the advent of iPSCs, neuropsychiatric disease studies examining neuronal differentiation are now possible, as skin cell biopsies of diagnosed adult patients that are reverted to iPSCs and further differentiated into neurons can be analyzed.

There are some problems limiting the use of iPSCs for disease modeling in schizophrenia and other diseases. Producing these cells is very difficult and tedious. The cells are slow-growing and heterogeneity in their capacity to differentiate is a confounding factor in assessing patient versus control differences. Workers in the field are trying to overcome these obstacles.

The findings of this study are not conclusive regarding the definite function of RP11-319G6.1 and RP11-586K2.1. However, it leads to a wealth of further questions that will be analyzed in the lab.

Additional iPSC samples from other cell lines containing with the 22q11.2 microdeletion would further validate the findings of this experiment. Another experiment of interest would be

Mentor: Dr. Herbert Lachman, Albert Einstein College of Medicine



Cassandra Rogers presents her poster at WESEF

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to carry out a gene-knockdown study, in which the expression of RP11-319G6.1 and RP11-586K2.1 are reduced using so-called shRNA vectors. This would allow us to study the effect of these lncRNAs on gene expression and neuronal differentiation, which in turn would provide insight into schizophrenia pathogenesis. Further experimentation could attempt to determine whether these lncRNAs affect chromatin.

Finally, another interesting experiment would be to determine the effects of lithium, a drug commonly used to treat bipolar disorder and some patients with schizophrenia, on the expression of these lncRNAs.

A potential source of error came from the small sample size used in these experiments. Given the opportunity and time to perform experiments on more lines results might have been more definitive. The lab is currently creating more cell lines for this reason.

While genetics once focused on the genes that code for specific proteins, the view of the human genome is now expanding to include regions once viewed as simply "junk." The long stretches of DNA between genes, and the introns within genes, were once thought to have little, if any, functional role. Only later was it understood that these regions are transcribed into various non-coding RNAs, including lncRNA, miRNA, snoRNAs, and other functional RNAs.

These non-coding RNAs contribute to transcriptional and translational regulation, influencing the expression of coding genes. The vast network of gene regulation by way of non-coding RNA and epigenetic factors plays a significant role in neuronal differentiation and growth, as well as the onset of neuropsychiatric disorders, including schizophrenia.

The diagnosis of schizophrenia is defined by a series of criteria, but genetic models and genetic subsets of schizophrenic patients could provide a new mechanism for diagnosis. Moving forward, more genetic research and studying the effect of non-coding RNAs on brain function will improve our understanding and treatment of neuropsychiatric disorders.

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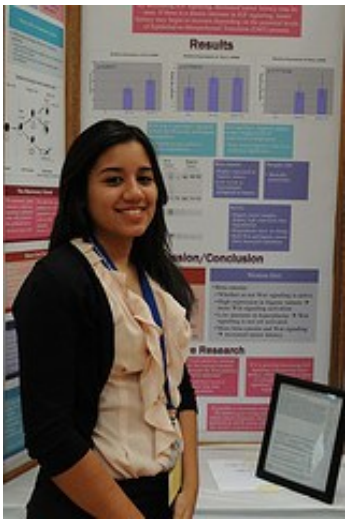
The Role of IGF Signaling in Modulating WNT Signaling Effectors in Mammary Gland Tumorigenesis in Mice, by Tiffany Porras. (Intel Paper Excerpt)

Cells that have yet to be differentiated are known as stem cells. They are different from mature differentiated cells in that stem cells still have the ability to divide symmetrically or asymmetrically, albeit infrequently (Charafe-Jauffret et al 2008). These "undifferentiated" cells divide rarely, compared to cancerous cells, so they always maintain this constant undifferentiated state that allows them to develop into a mature cell in the future (Weissman 2000). Weissman states how within a given stem cell population there are a small subset of stem cells that self-renew for a defined interval, meaning that only certain stem cells self-renew at a cer-

tain time, for a specific amount of time. In adult tissue, stem cells are characterized by their ability of multilineage differentiation (Weissman 2000). Multilineage differentiation is classified as the ability to be long lived and produce many cell types in a tissue (Smith & Chepko 2001). The earliest types of stem cells are totipotent, which extend from the zygote to the inner area of cell mass of the blastocyst (Weissman 2000). These cells then give rise to somatic stem and progenitor cells, as well as primitive germline stem cells (Weissman 2000). Adult stem cells begin as pluripotent cells, able to develop into multiple, but not all, cell types (Campbell & Reece 2005). They then turn into multipotent stem cells where they are specialized in a distinct area. Stem cells are capable of constructing non-self-renewing differentiated progenitors, which then specify into a more restricted differentiating potential and finally become mature cells (Weissman 2000). In order to specialize, stem cells continue to follow pathways that increase in lineage commitments (Weissman 2000). With the ability to specialize in various locations of an organism (such as the breast, brain, blood), stem cells can dictate how these cells differentiate in a particular area.

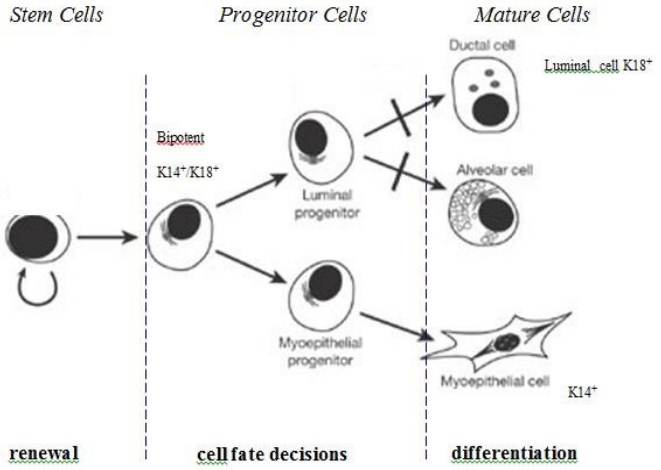
The mammary gland is said to contain a subset of mammary epithelial stem cells found in the epithelial area of the structure (Smith & Chepko 2001). The mammary gland contains two cell populations once the cells are fully matured (Bouras et al 2008). In one direction, the stem cells pursue the fate of myoepithelial progenitors, which then mature into terminally differentiated myoepithelial/basal cells. In the other direction, the stem cells commit to becoming luminal progenitors, which then differentiate into the luminal population that contains both ductal and, under the influence of pregnancy hormones, alveolar cells (Visvader et al 2006). Both cell populations contain a distinct amount of cells in the self-renewal-like phase. According to Smith and Chepko, if cap cells (multipotent progenitor cells, only around during puberty) enter the epithelial cell population near the body of the end bud in the mammary gland, these cells will become part of the luminal cell population. These end buds, better known as terminal mammary end buds, begin to develop in virgin females around the age of approximately 3 weeks and continue to grow until approximately 12 weeks (Smith & Chepko 2001). However, both cap cells and terminal end buds begin to disappear when the mammary fat pad is filled by the growth of the mammary ductal tree (Smith & Chepko 2001). If the cap cells migrate horizontally along the outermost layer of the duct that is in contact with the "fibrous stroma," then the fate of the cells lies with the myoepithelial population (Smith & Chepko 2001). This is said to be the result of the action of extrinsic factors on the cap cells (Smith & Chepko 2001). Already stated by Weissman, stem cells differentiate into specific cell types through the influence of signaling pathways. Cell specialization may also be said to be the result of gene action that directs the development of the fate of individual cells (Artavanis-Tsakonas 1999). Signaling pathways are activated by extracellular hormones and growth factors binding to cell surface receptors, which influence cell fate decisions of the mammary epithelium as shown in many previous studies (S (mith & Chepko 2001).

The development of the mammary gland is regulated by certain factors that affect how the gland grows. One of these factors is growth hormone (GH). GH is produced by the pituitary gland, which is located right at the base of the hypothalamus in the brain. GH increases protein synthesis in the body, incites the breakdown of fats, and aids in the body's absorption of glucose from the blood (Campbell & Reece 2005). GH induces and its function is mediated by insulin-like growth factor I (IGF-I) (Wiseman & Werb 2002). IGF-I is known to have an effect on the body's growth and development, whether being normal or abnormal (Campbell & Reece 2005). This growth factor is a requisite factor identified in mammary gland development (Wiseman & Werb 2002). GH and IGF-I have a direct relationship, implicating that when there are high levels of growth hormone, there will be high levels of IGF-I. In addition to



Tiffany Porras presents her poster at WESEF

Epithelial cell lineage in mouse mammary gland



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Mentor: Dr. Teresa Wood, Professor, New Jersey Medical School and Cancer Center.

circulating IGF-I, the mammary gland expresses IGF-I both in the stroma compartment (induced also by GH) and in the epithelial compartment in TEBs during development (Richert & Wood 1999). In addition to normal development, the GH/IGF-I axis has a significant role in the "cancer-cell process," especially in breast cancer, where GH and IGF-I bodily processes influence certain patterns in cancer occurrences that can affect the breast area (Pollak & Schernhammer 2004). The risk of developing cancerous cells increases when there are high levels of IGF-I, which also plays in part of the metastasis of the cancer (Cohen & Clemmons 2000).

IGF-I signals through a receptor called insulin-like growth factor-type 1 receptor (IGF-IR). IGF-IR is required for the growth of an organism, as it mediates the action of IGF-I. The IGF-I receptor (IGF-IR) is required in mammary epithelium for proper development of the ducts (Wiseman & Werb 2002). As well as the ducts, IGF-IR is responsible for the formation of the terminal end buds (Wiseman & Werb 2002). During pregnancy and postpubertal stages, IGF-IR continues to be expressed in the epithelium of the ducts (Richert & Wood 1999). The spatial patterns for the receptor's expression have been identified to have significance in determining the precise roles for these factors in the regular growth and development of the mammary gland (Richert & Wood 1999).

Two major signaling pathways that regulate stem cell number and self-renewal are the Notch and Wnt pathways. Notch is known to be involved in normal development of the mammary gland (Charafe-Jauffret et al 2008). Notch signaling in this pathway is a regulator of multiple cellular processes, including stem cell maintenance, cell-fate specification and differentiation (Artavanis-Tsakonas et al 1999). Notch's relevance in breast cancer development is its role in regulating self-renewal pathways, which when deregulated can lead to excess proliferation in the stem and or progenitor cells potentially leading to breast tumorigenesis (Charafe-Jauffret et al 2008). Previous research involving the Notch pathway in a DNhIGF-IR transgenic mouse used specific markers that can locate both myoepithelial and luminal cell populations to determine what was going on with cancer development and the cell populations when affected by Notch signaling. What was seen in these studies was a decrease in the myoepithelial cell population and a slight increase in the luminal population. More recent research has focused on another possible cell signaling pathway affecting the formation of tumors in the breast.

Wingless Related Proteins (Wnts) represents a family of secreted glycoproteins that have various expressive functions and patterns (Roarty & Rosen 2010). The best characterized Wnt pathway is known as Wnt/β-catenin-dependent signaling (Roarty & Rosen 2010). It plays a significant role in multiple adult stem cells, as well as in proper mammary gland development (Roarty & Rosen 2010). It has been noted recently though that the Wnt/β-Catenin pathway activation is affecting poor outcome in certain types of breast cancers (Kramtsov et al 2010). It is possible that excessive Wnt activation can be contributing to the pathogenesis of breast cancer (Roarty & Rosen 2010).

The first transgenic mouse model used in the laboratory was a dominant-negative human IGF-IR (DNhIGF-IR) expressed from the mammary-specific mouse mammary tumor virus (MMTV) promoter where the transgene has a defective kinase in the beta subunit of the IGF-IR and thus cannot transduce any further downstream phosphorylations of the IGF-IR. The data that resulted from using this model displayed a shift in lineage in the dominant negative mammary epithelial cells that lead to an increase in luminal progenitors and a decrease in myoepithelial stem/progenitor cells. The next tumor model involved was a MM MM TV–Wnt-1 tumor model that is associated with mammary gland hyperplasias developing within 4-5 months of age. Since the MMTV-DNhIGF-IR transgenic model reduces stem-like cells but amplifies luminal progenitors and the MMTV-Wnt-1 transgenic model shows an increase in the "stem cell" population as well as luminal progenitors during formation of mammary tumors, a bigenic tumor model was created in order to see what would occur in terms of tumor formation and tumor latency when both transgenic models were combined. It was believed that by combining both transgenic models, the tumor latency would be increased because there were less stem/progenitor cells in the DNhIGF-IR to begin with. After combining both tumor models, it was seen that when IGF-IR signaling was decreased, tumor latency was also being decreased. Lineage markers were used to see if the bigenic model illustrated a shift in lineage that could be making the mammary epithelial cells (MECs) more susceptible to tumorigenesis. Epithelial to mesenchymal transition (EMT) markers were also used to track the cells because EMT can cause tumor cells to become more motile, and hence, more invasive. Based on these studies, the question being asked is whether loss in IGF receptor signaling increases Wnt or Notch signaling and EMT dur-

ing Wnt-induced tumorigenesis.

We are looking at certain end points in Wnt hyperplasias, Wnt tumors, bigenic hyperplasias, and bigenic tumors to observe a change in Wnt signaling expression and if targeting this pathway in the future may be of therapeutic help in breast cancer treatment.
(...)

Discussion:

Although the RT-PCR appeared similar between the bigenic and Wnt-only hyperplasias, there still was an important result. What is being seen is that compared to the Wnt alone hyperplastic profile, the bigenics seem to be occurring 2 to 3 months sooner. This verifies the observation that tumors and hyperplasias are occurring earlier than expected. As the stem cell population is being altered, it appears that the Notch target is also being altered in the Wnt tumors. So far this supports the hypothesis that Wnt signaling is positively correlated to latency of the tumors and the hyperplasias and that decreased IGF-IR enhances the Wnt tumor formation.

As for the Western blot, the beta-catenin symbolizes whether or not Wnt signaling is active in any of the samples. With relatively high expression of beta-catenin levels in the bigenic tumors, it implies that there is more Wnt signaling activation in these tumors. Low amounts of beta-catenin in the hyperplasias still dictates that Wnt signaling is in the process of being activated, and that as it develops into a tumor, the activation only increases and gets stronger. With more beta-catenin and more Wnt signaling, this means that the tumors will start forming sooner, and develop and grow at a faster rate.

Conclusions:

The way that mammary tumors progress and develop in the body is a mystery to all, not only because there are hundreds of types of malignancies, but because there also follows a number of factors involved in the progression of cancer. Signaling pathways are major factors behind the evolving and ever changing world of breast cancer, as more questions are being asked and fewer questions are being answered. It is important to look through as many perspectives as possible when it comes to a topic as serious as this so that the answer to the question will become easier to find.

Looking first at the Notch pathway that was mostly known for its involvement in the growth and development of the mammary gland, target genes were identified and ligands were studied in order to have a better meaning of the subject. Pointed then towards the Wnt pathway, more factors appeared as RNA expression was analyzed and protein levels were examined. The importance of studying these pathways and their level of activation in mice using transgenic models is to determine whether or not there is a factor that can be acted upon as a sort of therapeutic treatment. If it is seen that decreasing IGF signaling is decreasing tumor latency, what will happen if there is a drastic increase in IGF signaling? Will tumor latency begin to increase?

It is critical to expand upon the results that have already been made public as that will lead to a clearer understanding of the answer. Feeding off of this experiment, since there was an increase in protein levels of beta-catenin, it might be appropriate to look at beta-catenin at an RT-PCR level to determine its relative expression and fold change. Also, because EMT is significant in the progression and metastasis potential of a tumor, SLUG should continue to be observed in both Wnt and bigenic hyperplasias and tumors via RT-PCR. If it is possible to determine where the pattern goes wrong and the cancer originates, it can aid in decreasing the rate and level at which cancer affects the world's population today.

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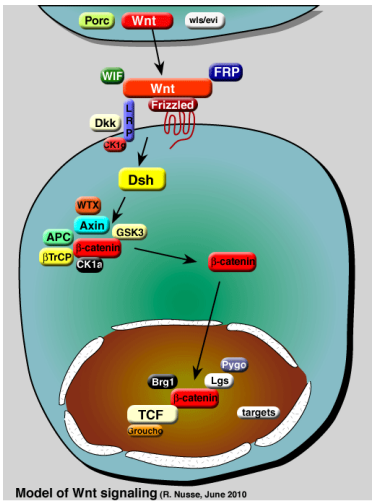
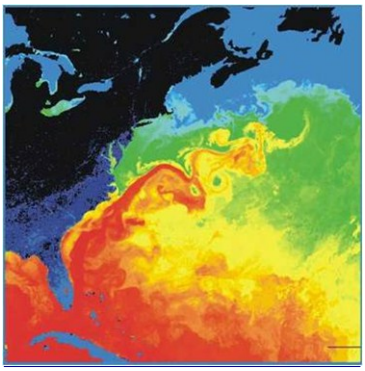
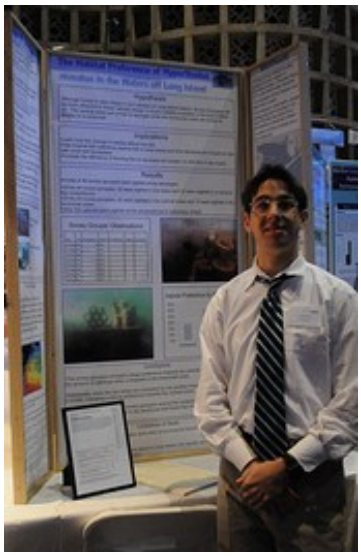


Model of Wnt signaling (R. Nusse, June 2010

Senior Projects



Sea Surface Temperature picture of Gulf Stream by NOAA from Paparo's article



Alex Alves presents his poster at NYSSEF

The Habitat Preference of Hyporthodus niveatus In the Waters off Long Island , by Alex Alves (Intel Paper excerpt)

The waters off long island are host to many tropical fish (Paparo 2007, Galiano 1996, Cohen 2007). Although most people believe that the tropical fish can only survive around Florida, this is far from being the case. (Whitefield 2002) The gulfstream helps transport these fish to long island. Once there, the shallow bays provide a location for the fish to survive into the latter months of summer and fall until the water becomes too cold. The Hyporthodus niveatus commonly known as the Snowy Grouper is one such fish and during its stay it requires a place of shelter (Paparo 2007, Galiano 1996).

The gulfstream itself is sole responsible for transporting these juvenile fish (Paparo 2007, Galiano 1996). It is a warm water current which transports the water north from tropical waters to the colder waters of the Atlantic as far up the Arctic Circle (Whitefield 2002).

To understand better how the Gulf Stream may transport these tropical fish, once must understand the lifecycle of these fish. The adult species reproduce in the waters in the Gulf of Mexico in the spring, creating Microscopic eggs and larvae, which are then sucked into the Gulf Stream. (Paparo 2007, Galiano 1996). The larvae fish are too small to fight the current and, thus, are carried north along with it. As they travel north, the larvae fish grow and become juveniles, but they are still forced to remain in the Gulf Stream as the pelagic water surrounding the current is far too cold. (Paparo 2007, Galiano 1996, Whitefield 2002) As the summer progresses, the juvenile fish, transported in the Gulf Stream, move closer to long island, being around 200 miles off shore (Whitefield 2002). With the help of even a small storm, pockets of water can be pushed into long island waters, closer to the shore. Once at the shores of long island, the fish seek out the warm shallow waters of the bays. The shallow bays are able to be heated by the sun to the point where the water is warm enough for the fish to survive and thrive. Unfortunately for the majority of the species, this time is only limited to the summer as the winter months will bring colder water, and they still will not be old enough to swim south and will not survive (Paparo 2007, Galiano 1996).

Although the water temperature may be similar to the fish as in their normal range, there can be many differences between the environment in their normal range and the environment off Long Island (Paparo 2007, Galiano 1996). The largest difference between the two environments is that in visibility (Rich Galiano 1996, Cohen 2007). The waters off Long Island are fairly cold and thus allow many different species of algae to develop (Paparo 2007, Galiano 1996). These algae species are able to thrive in the warmer waters. The extremely high algae count is a stark opposite to the low algae count in places such as Florida (Paparo 2007, Galiano 1996). From personal experience, I can say that the waters in Long Island can sometimes be filled with so many algae that you can't see your hand in front of you. The increases in algae also have many effects on the ecosystem. The first simple one being that the algae decreases the visibility in water. This means that in green to brown tinted water the neon colored tropical fish will stand out. Another affect of the algae is that it changes the dissolved minerals on other things in the water. Another big difference between the normal range of these fish and Long Island is that the temperature in Long Island fluctuates much more. The reasons that the bays are warm enough to allow the tropical fish to survive is that they are shallow and that the sun heats them up, however due to the natural tide cycle in an incoming tide cold water from the deeper ocean comes into the bay, decreasing the temperature. Fluctuations such as this are not as large in the native range of these fish. I predict that despite all these differences that the fish will instinctively tend to favor habitats that are similar to these which they favor in their normal range (Wilson 2010).

By placing various artificial structures in the waters off long island, it is possible to learn about the habitat preferences of juvenile Hyporthodus niveatus in this situation. Such data hasn't been gathered for Hyporthodus niveatus outside of their normal habitat range. (...)

Discussion and Conclusion

The number of fish that preferred the cinderblocks could be explained by the fact that I posed no threat to the fish. As a result, these fish would not feel threatened and would have no need to hide. Staying around the cinderblock gave the fish the option of hiding if it became necessary, yet it did not back the fish into a corner.

The vast majority of the fish found in and around the structure were Snowy Groupers. Snowy groupers are a common tropical fish in the area and thus their large population would lead to more fish around the structure. Another possible reason for the lack of diversity of other fish is that Snowy Groupers are predators. They could have eaten or scared

away fish of other species.

The results of the experiment from the first year are not enough to prove or disprove the hypothesis. The sample size was far too small to decide anything definitively. This can be the consequence of many different parameters, but does not indicate a failure in the project itself. I believe that the main reason for the small sample size is the year itself. The number of tropical fish found in Long Island every year is slightly different, with extreme variations: In some years there will be thousands and in others practically none. Atlantis Marine World also had little success in tropical fish collection that year, indicating that it was an overall bad year for tropical fish in Long Island. The Gulf Stream can alone be responsible for the lack of tropical fish. In 2010, there could have been another factor. The BP oil spill in the Gulf of Mexico has had some devastating effects on the local environment, many of which are still unknown. It is plausible that the oil affected the spawning of the fish in the Gulf of Mexico and in the south Atlantic. If this was the case, the number of Larva in the gulf steam would decrease substantially.(NAS 1975)

The reasons stated above are based off the lack of tropical fish as a whole, however in addition there are other factors that could have added to the lack of a sample size the first year. Although the locations were picked carefully, it is still possible for the locations themselves to be an issue. Parameters such as the flow of water in the bay, the distance to docks and other factors could influence the results. I believe that the reason the first location failed to give any results could be because of a sandbar redirecting the water from inlet around the location. The second and third locations are close to a large dock, it is possible that the tropical fish took refuge in the dock before they had an opportunity to go to my project. Another possible downfall of the second and third locations could be the current. At certain times of day the current in the location moves extremely fast. Although this would make the need for shelter increase, it could also make it more difficult for certain fish to get to the shelter once spotted. One thing that could have affected both locations would be the presence of large predatory fish such as a striped bass, bluefish, or large spotted grouper at the location of the shelter. If one of these fish knew the location of the shelter they would use the shelters as a mean of lure fish there to better hunt them. If this was the case, only the smallest fish would be able to hide in the large vertical tubes such as the snowy grouper did. The low number of spotted fish at the location may be the result of many of these factors combined.

The number of snowy groupers found in the structure in the summer of 2011 corresponds with the number of sightings in general. During the summer of 2011 many different species of tropical fish where sighted, even some rarer species. Personally, I saw seals, rays, and other tropical species, all of which are extremely rare generally in this location. I have not seen such diversity in the waters off Long Island in years. Evidence towards this could be the frequent storms towards the end of the summer climaxing with hurricane Irene. Storms are pockets of warm air from the gulf, and they help carry warm water as well, as storm is a strong evidence of the flow of the Gulf of Mexico.

The results from the second year are much more conclusive and I base my conclusion on the data from that year. In total I was able to count 44 snowy groupers in or around the structure. Of the 44, 12 chose to stay around the structure and in the cinderblock indicating that the fish had the option of whether or not they chose to hide in the PVC structure. Of the 32 Snowy Groupers who decided to go into the PVC tubes, 22 of the fish chose to go into the vertical orientated tubes compared to the 10 that where in the horizontal tubes. The fact that 22 of the fish decided to enter the tubes indicate that the tubes were a viable habitat for the Snowy Groupers. The PVC tubes were identical in size and shape, but difference in orientation. If we assume for a second that the orientation of the tubes have no impact on fishes preferences then we should see an evenly distributed preference of fish. Given this assumption we get a p value of 0.03389 so we reject the null hypothesis of the test and say that there is evidence that the orientation of the tubes influence the preference of fish. Just comparing the two tubes, the Snowy Groupers showed a strong preference for the vertically oriented tubes.

This study showed the preference of the fish in the waters off Long Island. To discover if the preference of the fish changed when compared to the fish in their natural habitat it would be necessary to compare the results with a similar experiment in the normal range of their habitat.

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Example of structure after a year of submersion. Picture taken by student.

Mentor: Dr. Todd Gardner, Atlantis Marine World Aquarium, Riverhead.

Example of Hyporthodus niveatus in the structure

Senior Papers

The Effect of Estrogen on the Perceived Taste of Miraculin, by Shivanee Shah (Intel Paper excerpt)

Shivanee Shah presents her poster at NYSSEF

Imagine being able to eat lemons and perceive them as sweet, even without adding any sugar. This may be possible with the addition of miracle fruit. Miracle fruit is a tiny red berry with a large seed covered by a thin layer of pulp that contains Miraculin (Bartoshuk et al., 1969). Miraculin is a taste modifying protein that causes the suppression of sour taste and makes it seem sweet. To activate the effect of miracle fruit, one must hold the pulp of the berry in their mouth for a few minutes. After swallowing the pulp, the effect of Miraculin lasts for about thirty to sixty minutes (Bartoshuk et al., 1974). The berry itself has no taste. Even though the sour taste of a food seems suppressed, the acids continue to work as acids and thus can be harmful if consumed in large quantities (Kurihara and Beidler 1969).

Miracle is native to West Africa and for hundreds of years miracle fruit was used by native Africans to suppress the bitterness and sourness of palm wine and maize dishes to make them palatable (Daniell 1852). Miracle fruit is difficult to cultivate outside of its natural environment because the plants need to be in a tropical environment with no frost and need a certain pH level to grow successfully. Presently, these plants have been cultivated in Florida (Sun et al., 2006). Also, Miraculin has been successfully transferred into genetically modified lettuce, tomatoes, and strawberries. The effect of Miraculin continues to work in all three plants. (Sun et al., 2006, Sun et al., 2007, Sugaya et al., 2008). The concentration of Miraculin protein found in miracle fruit can be as high as 10% (Sun et al., 2006). It is unknown specifically how the protein works on taste cells to suppress taste, but it is known that the protein does not work by blocking sour taste receptors (Bartoshuk et al., 1974).

With its effect to suppress sour taste and make it seem sweet, Miraculin could be very useful in everyday life. Patients undergoing chemotherapy often complain that food is unpalatable because of a metallic taste lingering in their mouth. With the addition of miracle fruit to their diet, the patients can experience food without the metallic taste. It could also be beneficial for nausea caused by bitter tastes commonly experienced by pregnant women in early pregnancy because miracle fruit can prevent or minimize this bitterness. Diabetics can use the miracle fruit to increase their perception of the sweetness of foods, allowing them to keep their blood sugar in balance by reducing intake of highly sweetened foods. In addition, it can be used as a weight loss aid; people can add miracle fruit to their diet and reduce their use of sugar and artificial sweeteners. In addition to pregnancy, these conditions are likely to be disproportionately experienced by women, as rates of breast cancer are increasing, and women are more likely to be obese and diabetic (Bartoshuk et al., 1974). Thus, Miraculin may lead to useful advances for women's health.

Genetic variation in the ability to taste bitter has been known since 1932. A chemist from Dupont named Arthur Fox was synthesizing phenylthiocarbamide (PTC) in his lab when some dispersed in the air. His lab mate complained of a foul, bitter smell while Fox was unable to detect the compound (Fox 1932). Family studies followed and discovered that humans vary widely in their ability to detect not only PTC, but also a related compound 6-n-propylthiouracuil (PROP). Approximately 70% of individuals in the United States are considered "tasters," while the remainder of people are "nontasters" of these compounds. The primary difference between tasters and nontasters is the number and density of fungiform papillae that contain active taste cells. The ability to taste PTC and PROP are conferred by genetic variation at a bitter taste receptor, TAS2R38 (Kim et al., 2003). In addition to bitter, tasters also experience many other basic tastes (e.g. sweet) and textures (e.g. oral burn and oral touch) at intensified levels. This may impact dietary preferences, as tasters tend to dislike bitter foods, such as black coffee, grapefruit, and Brussels sprouts (Duffy and Bartoshuk 2000).

Another biological variation that could impact Miraculin function is hormone variation. Hormone levels of women change throughout life. Fluctuations in reproductive hormones, particularly estrogen, have been shown to influence sensory functions, including visual, auditory, and olfactory thresholds for women (Doty et al., 2009, Parlee 1983, Simerly et al., 1989). Sex differences in taste perception have been reported in numerous species, including rats and humans. The effect depends on a variety of factors, including the taste quality, taste concentration, and the species. In humans, the threshold for sweetness is the highest during preovulation, when estrogen levels are rising. Taste thresholds refer to the lowest concentration of a tastant that can be reliably distinguished from plain

water. In addition, during the early stages of pregnancy, when estrogen levels are high, women are more sensitive to bitter taste. This is theorized to prevent the pregnant mother from ingesting toxins that may harm the fetus. Many women continue to have heightened taste sensitivity to bitter after pregnancy (Than et al., 1994).

The objective of this work was to determine how biological variations in the genetic sensitivity to bitter taste and hormonal fluctuations influence the taste suppression effects of Miraculin. Specifically, the following aims and hypotheses were tested. First, the correlation between perception of citric acid before and after Miraculin and PROP taster status will be positive. Second, the correlation between perception of citric acid before and after Miraculin and days since last period, a marker of estrogen levels, was calculated to test the hypothesis that the correlation between the days since the last period and the perception of citric acid after Miraculin will be positive.

(...)

Discussion:

Although this study had inconclusive results, it still is important to study further the effects of Miraculin. One of the hypotheses was that the correlation between perception of citric acid before and after Miraculin and PROP taster status would be positive. The second hypothesis was that the correlation between the perception of citric acid before and after Miraculin and days since last period, a marker of estrogen levels, also would be positive. Unfortunately, due to the limited number of participants enrolled in the study, the data was not significantly different and led to statistically inconclusive results. A controlled estrogen level is critical for this study. While a large number of young women in our school enrolled for this study, the majority of these volunteers had to be turned down as they were taking some form of hormonal contraception or had very irregular cycles, making them ineligible for this study. Additionally, vegan students, which consume large level of soy, also had to be eliminated due to the phytoestrogenic effects of soy (Xu et al., 1998) on endogenous levels of estrogen. Only 18 participants had cycles regular enough to be considered, as it was critical that their estrogen level be controlled. The lack of significance of the results certainly stems from the limited number of participants. Some uncontrolled parameters, such as the food intake on the day of the test, stress level, time of testing during the day, lack of sleep, possible infections with the common cold may have created additional variability confounding the data. Such parameters likely would cancel each other in a larger group, but with a limited number of eligible subjects, they may have created uncontrolled effects.

These insignificant findings, with a rather small experimental group, may, therefore, seem unimportant; however, they bring us closer to understanding the relationship between estrogen levels and the perceived taste of citric acid before and after Miraculin, as well as between taster status and perceived taste of citric acid before and after Miraculin. Certainly, it will be necessary to add more subjects to allow more conclusive findings. At present, however, some insignificant trends suggest that the hypothesis about taster status and the effects of Miraculin may be correct, while the hypothesis about estrogen levels and the effects of Miraculin may not be correct. This needs to be studied further in order to examine the effectiveness of miracle fruit and Miraculin in woman's health issues, for example bitterness induced nausea. The ability to taste sucrose varies with the estrogen level (Than et al., 1994). In our study, the students also rated their perceived taste of sucrose in parallel to the Miraculin study hereby presented. Our testing did not replicate the results found in the literature. This shows that the present methodology had flaws which prevented a significant result from emerging. Even perceived tastes of sucrose had been shown to be affected by estrogen levels. A major flaw could have been the small size of the group, as mentioned earlier. Additionally, the age of the subjects, between 15 and 17 years old, and their life styles may have generated increase variability and additional parameters. The hormonal level of teenagers can also vary drastically and their estrogen cycle may not be completely established at this age, further confusing the results.

Future studies may produce a better understanding of how Miraculin works. This will be essential to make a sweetener supplement out of it. With the knowledge of how Miraculin works, we can more effectively use it in daily life. Another intriguing question for this field is to determine whether Miraculin has any adverse effects. To date, there have been no reported adverse effects; however, it will be key to make certain that there are no adverse effects if this product has any potential to be used by the general public. These are some of the questions that will dominate research in the field of Miraculin.

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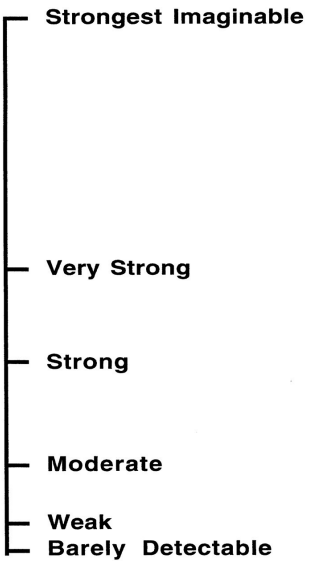

Example of tasting scale used by subjects in the study.

Mentors: Dr. Kathleen Keller, New York Obesity Research Center, Columbia University. Dr. Kathleen Curtis, Oklahoma State University

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Junior Projects

Replacing platinum as the catalyst in PEM fuel cells, by Alexis Mangual

Mentor: Dr. Steven Suib, University of Connecticut

The modern world needs to replace oil as its major source of energy in the near future if society is to thrive for many years to come. Today, there are two main contenders: Fuel cells and Nuclear Energy. However, because Nuclear Energy can be dangerous, fuel cells are considered the better choice. Polymer Electrolyte Membrane Fuel cells or PEM fuel cells may be the silver bullet in replacing oil as a necessity to fueling transportation, a major component of global oil consumption. Polymer Electrolyte Membrane or PEM fuel cells operate around the electrons released by oxidizing hydrogen and then the recombination of the ions with electrons and oxygen to form water. Hydrogen molecules are oxidized at the anode side of the fuel cell and the released electrons are used to generate a current. The hydrogen ions pass through the electrolyte which facilitates the transport of hydrogen ions and combine with oxygen at the cathode side forming water. As a result, Fuel cells are an extremely clean and efficient system having only pure water and heat as byproducts in the reaction making fuel cells a viable candidate for replacing gasoline as the number one energy source. (Wang et al). However, on the anode and cathode layer, there is a catalyst coating which presents a major problem for this technology. The catalyst is a major part of the fuel cell as it facilitates the reactions that take place and is a major factor in the efficiency and power of the system (Yan and Wu, 2007). The only commercially available catalyst for Fuel cells is Platinum. Platinum, being so expensive, is a contributing factor to the significant price difference between fuel cell engines and gasoline engines (Sun, Ogden, and Delucchi, 2010). Although the initial cost is high, the overall lifetime cost of a fuel cell vehicle is relatively similar to that of a standard internal combustion engine (Sun, Ogden, and Delucchi, 2010). Platinum is subject to multiple types of degradation that decrease the electrochemically active surface area available for the fuel cell reaction (Aindow, Haug, and Jayne, 2011). Replacing platinum as the catalyst in PEM fuel cells as well improving the support used for the catalyst has become a major issue as a new alternative catalyst is needed that is not only less expensive, but also just as efficient and more durable.

There are a variety of potential replacements for Platinum catalyst. One major possibility are platinum based alloys which take the advantages of Platinum and combine them with the positive effects of alloy interaction on durability. (Mercado and Popov, 2005). The other major category is non-platinum catalyst which although less expensive, can be significantly less powerful then Platinum as a catalyst for fuel cell reactions (Olson, Chapman, and Atanassov, 2008).

One such catalyst considered to replace Pt was PtNiZr which was made adding Zr to PtNi. The alloy is a viable alternative to Platinum in PEM fuel cells while also having increased resistance to corrosion as compared to Platinum (Whitacre, Valdez, and Narayanan, 2007). Increasing the overall amount of Zr in the alloy over 5% found a decrease in over-all power density possibly due to the occupation of potential Pt sites on the support by Zr. However in the configuration of Pt59Ni39Zr2, the alloy provided an increase in current density when compared to pure Pt meaning, was less expensive than pure platinum, and did not suffer from degradation that high Zr content created (Whitacre, Valdez, and Narayanan, 2007). These ternary (trimetallic) alloy catalyst are excellent replacements to pure platinum in part due to the ability for the metals to act synergistically to enhance the effects each metal has (Cigdem Guldur

et al.). Another such alloy was the PtxNi/C con-figuration where x could be anywhere from 1 to 3 defining the ratio of Pt to Ni (Wu, Wexler, and Wang, 2009). The objective of this catalyst was to reduce Pt loading while maintaining or even increasing the overall catalytic activity in the ORR (oxygen reduction reaction). Through multiple test, they found that the overall catalytic reactivity of the Pt alloy was on par or even greater than that of pure Platinum by partially replacing the Platinum with Nickel (Wu, Wexler, and Wang, 2009). The configuration of Pt3Ni1 which was shown to be efficient was studied for its overall durability. The results found that the configuration also showed high resistance to sintering increasing its overall durability (Mercado, Kim, and Popov, 2004). This catalyst not only exhibited an exceptionally high power output as shown by Wu, Wexler, and Wang but also was durable and more cost effective than pure platinum. This catalyst showed clear signs of being a viable option as a Platinum based alloy catalyst for future PEM fuel cells.

A different study conducted on the PtNi configuration evaluated the catalysts for performance and resistance. The study conducted looked at Pt1Ni1 as a potential configuration when compared to regular Platinum. In this PtxNi configuration, they found a decrease in overall power output due to the decreased reactivity caused by the corrosion of the Ni (Cho et al). Although the PtxNi/C configuration shows a lot of promise, in the Pt1Ni configuration, the lower ratio of platinum to nickel causes a decrease in durability and overall performance (Cho et al).

Platinum can be paired with a variety of different metals to form a catalyst to produce a catalyst with enhanced electrochemical characteristics and lower cost. Another such Platinum alloy catalyst examined was PtPd. This catalyst configuration was tested at atomic ratios of anywhere between 1:2 and 1:8. It was found that the catalyst demonstrated increased corrosion resistance with lower ratios and increased performance with higher ratios (Thanasilp et al.). Palladium combined with Platinum in a catalyst gave the catalyst special properties not present in pure platinum catalyst in much the same way that Nickel and Zirconium gave Platinum catalysts special properties and positive properties in moderation.

Another first row transition metal, Cobalt, can also be paired with Platinum to produce an alloy catalyst that is both comparable to pure platinum and less expensive. PtCo was tested at differnt alloy ratios and it was found that as long as Platinum content was greater than Cobalt content, for instance Pt0.55Co0.45, the catalyst had greater power output then Platinum. Cobalt exhibited two special properties in the catalyst: It increased desorption of oxygenate species of the platinum surface and induced a contraction of the Pt lattice leading to a change in the electrochemical properties of Pt (Wu et al.). Like the other previously mentioned metals and transition metals, Cobalt again provided a special quality when combined with Platinum to form an alloy catalyst that would make it a viable candidate for a component of a catalyst superior to pure platinum.

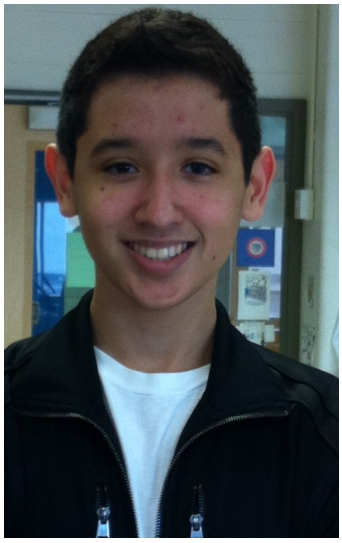
First row transition metals all appear to be viable candidates as components of an alloy catalyst that can beat Platinum as examples such as Cobalt and Zirconium prove to increase stability while retaining electrochemical performance (Antolini et al.).

Amongst other metals that stand out for being paired with Platinum to form a superior alloy catalyst is Rhenium. When Rhenium was added to a PtSn catalyst, the alloy catalyst exhibited higher current density than either PtSn or PtRe alone. Rhenium has the special properties of helping to break C-C bonds in an ethanol fuel cell (Basu et al.) If Rhenium has a special property in an ethanol fuel cell, the likely hood is it would have some sort of special property in a PEMFC.

A major possibility comes up when discussing replacing pure Platinum in that an alloy catalyst can be made from other metals exhibiting decent performance in a fuel cell and then adding Platinum to the alloy using its special property of immense catalytic performance. A catalyst was tested consisting of Palladium, Tin, and Platinum with an alloy ratio of Pd0.45Pt0.5Sn0.5. The catalyst exhibited a catalytic activity around half that of commercial E-Tek Platinum. The components of this alloy catalyst acted synergistically having positive effects on catalytic performance.

Another possibility is to create alloy catalysts that go beyond ternary. In a study testing Pt6CuFe, Pt6AgCu, and Pt6AgFe, research highlighted the fact that each component of the catalyst had its own special impacts on the performance of the catalyst and together, the components of the alloy had a final unexpected positive impact on performance (Güldür et al.).

On the other hand, there are the non-platinum catalyst which have their own advantages but also provide their own disadvantages. One such catalyst was Tantalum Oxide (Ta2O5)



Alexis Mangual

Junior Projects

which was carbon supported by a thin carbon paper (Kim et al, 2010). The Tantalum Oxide was found to be chemically stable under the pH conditions inside the ORR on the cathode side however it did not exhibit the power output that Platinum or Platinum based alloys could produce (Kim et al, 2010). The problem with Tantalum Oxide is that it tends to have poor electrochemical performance due impart to its poor conductivity; The Tantalum Oxide was found to have a 9% current density to that of Platinum at best providing a 1/3 the power output when carbon supported (Kim et al, 2010). The fact was that although the Tantalum Oxide is a solid competitor to Platinum as the catalyst for fuel cells, its catalytic reactivity had to be drastically improved before it could even be considered as a replacement for Platinum or Platinum based alloys (Kim et al, 2010).

Tantalum Oxide as a catalyst can be significantly improved by addition of tungsten oxide to the mix. Adding tungsten oxide to the standard tantalum oxide catalyst significantly improves electrochemical activity although the results are still lower than platinum and platinum based alloy catalyst (Jin Yong Kim et al.). At increased loadings of tantalum oxide enhanced with tungsten oxide, the catalyst becomes a viable alternative to platinum while still maintaining a far lower cost to platinum.

Ultimately, Metal alloy catalyst that include Platinum prove better to non-Platinum based catalyst because they currently exhibit greater performance values as catalysts while still retaining greater stability and significantly lower costs than those of commercial Platinum.

If using a Platinum based alloy catalyst in a PEMFC , it is necessary to choose the best catalyst support available. Standard carbon supports such as XC-72 vulcan carbon prove less efficient as supports compared to NMCCs or Nano-silicon carbide.

Both pure platinum and a platinum based alloy catalyst were tested on an NMCC support to examine the effectiveness of NMCCs as carbon supports. PtPd/NMCC and even the Pt/NMCC (Nitrogen Modified Carbon Composite). The results showed that when Pt3Pd and even pure Platinum are supported by NMCC, they possess a much higher stability than regular carbon supported Platinum and also have an increase in power due to the an increase in electrochemically active surface area (Li, Park, and Popov, 2009). The catalyst supported with NMCCs demonstrated increased performance than had the NMCCs been replaced with standard XC-72.

Platinum supported with SiC (Nano-Silicon Carbide) after 4000 testing cycles, had a noticeably greater electrochemically active surface area than the Platinum on a standard XC-72 carbon support. The power output of the SiC supported Platinum was near that of standard carbon supports and the SiC supported Platinum demonstrated increased resistance to degradation in the fuel cell (Shichun Mu et al.).

Even the preparation of the catalyst for fuel cell usage is an important matter when researching ways to improve catalyst to reduce the need for pure platinum. One such new preparation technique as opposed to the standard deposition on the support is Supercritical deposition. Supercritical deposition involves using a supercritical fluid to more evenly disperse catalyst particles on the support. Catalyst prepared by SCF demonstrated drastic improvements in activity over standard techniques as a result of the more homogenous distribution of active particles over the carbon support (Can Erkey et al.).

I propose a metal alloy catalyst that combines low Platinum loading (below 10 wt. %) with multiple metals such as Nickel, Zirconium, Iron, and Palladium, creating a multi-metal alloy catalyst that has performance comparable to that of Platinum along with increased stability and significantly lower cost. The research would consist of finding the best metals for the catalyst evaluating their special properties in a catalyst then testing a wide variety of metal loadings in the alloy to find the catalyst configuration that would be best suited to replace Platinum as the catalyst in Polymer Electrolyte Membrane Fuel Cells.

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Molecular Characterization of the Dopamine Transporter and its Cocaine binding site, using a bacterial homologue, the Leucine Transporter, by Audrey Hecht.

Sophomores Projects

The interactions between nerve cells or neurons in the brain play a central role in human behavior as they control emotions and cognitive responses to environmental factors. Sensory neurons transmit information from receptors located throughout the body that detect external stimuli such as the smells, sights, sounds, textures and taste, as well as internal stimuli such as muscle tension, carbon dioxide levels, and blood pressure. These sensory stimuli 'travel' along specialized extensions of neurons, called axons, as electrochemical signals, i.e., action potentials, toward the central nervous system; near the axonal end, the neuron splits into several branches. Located at the extreme end of each branch is the presynaptic terminal that, together with the postsynaptic terminal of another neuron these structures, builds the so-called synapse (Fig. 1). At the synapse, nerve impulses are carried from one neuron to another by means of chemical messengers called neurotransmitters. These neurotransmitters pass signals between neurons in the brain, thereby forming networks that regulate emotion and behavior (Campbell & Reece, 2005).

Upon the release of neurotransmitters from synaptic vesicles located in the presynaptic terminals of the transmitting cell into the synapse, they bind to receptors on the postsynaptic membrane, located on the receiving cell (see Fig. 1 for a schematic representation). Binding of the neurotransmitter to the receptors leads to a cascade of events at the postsynaptic nerve ending that is responsible for the propagation of a nerve impulse. This sequence of molecular events is terminated by the reuptake of the neurotransmitter back into the presynaptic terminal, thereby preventing the continued interaction of the neurotransmitter with the postsynaptic receptors. Different neurotransmitters, e.g. the monoamines (dopamine, serotonin, or norepinephrine) bind to particular receptors (Campell & Reece, 2005). For example, the neurotransmission of dopamine (DA) influences behavioral, interactive, cognitive, physical, and emotional reactions. Dopaminergic neurotransmission ends with the removal of the neurotransmitter dopamine from the synapse by the reuptake of dopamine by the dopamine transporter, DAT. DAT together with other transporters such as SERT, NET, and GAT (transporters for serotonin, norepinephrine, and GABA, respectively) belong to the SLC6 gene family, encompassing the human members of the neurotransmitter: sodium symporter (NSS) family (Chen et al., 2004). These symporters use the electrochemical Na+ gradient across the presynaptic membrane as source of energy to transport their respective substrate into the cell (Campell & Reece, 2005).

Physiological effects of dopamine in the synapse:

Studies performed on knockout mice in which the DNA required to synthesize DAT was removed from the cell showed that the reuptake of DA from extracellular space was 300-fold times slower than average, hereby increasing the concentration of DA on the exterior of the cell. The mice were hyperactive, possessed cognitive deficits, disrupted sensormotor gating, and sleep dysregulation. Pyschoactive drugs, such as cocaine and amphetamine (e.g. ecstasy) have been shown to cause similar effects on behavior and it appeared feasible to assume that psychoactive drugs act on DAT in a similar manner (Chen et al., 2004).

The action of psychoactive drugs :

Compounds with addictive properties, like cocaine or amphetamines inhibit DA reuptake and target the mesolimbic dopamine system. This process prolongs the sensation one experiences when using inhibitor drugs since elevated local concentrations of DA in the synapse result in continued stimulation of the postsynaptic receptors. This observation led to the hypothesis that the inhibition of DA reuptake is the primary action of cocaine. However, cocaine may also be capable of blocking other monoamine transporters other than DAT (Amara & Sonders, 1998).

Audrey Hecht

Mentor: Dr. Matthias Quick, Assistant Professor of Clinical Neurobiology, Columbia University, College of Physicians and Surgeons.

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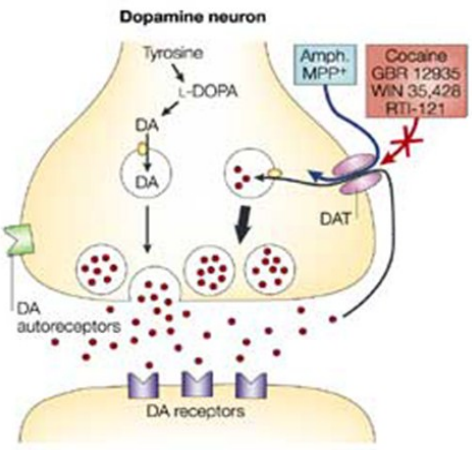
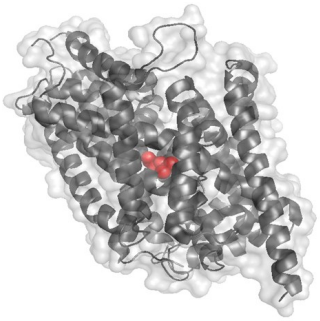


Figure 1: The dopamine neuron. Dopamine (DA) is accumulated in vesicles in the presynaptic neuron and released in the synaptic cleft where it can bind to DA receptors located in the postsynaptic neuron. Clearance of DA from the synapse is achieved by re-uptake of DA into the presynaptic neuron by the dopamine transporter (DAT). Psychoactive drugs, e.g. cocaine or amphetamines, inhibit the re-uptake leading to increased levels of DA in the synapse.

Figure 2: Model of the LeuT Protein



Other psychostimulants target the same group of transporters by inhibiting the reuptake of neurotransmitters from the synapse. SSRIs (Serotonin Reuptake Inhibitors), such as Prozac, act on the serotonin and norepinephrine transporters and are used as antidepressants to treat depression. Similar to the inhibitory efficacy of cocaine, their action leads to a high level of free neurotransmitters left in the synapse, resulting in prolonged interactions with postsynaptic receptors. As a result, the continuation of neurotransmission acts against the clinical manifestation of depression (Kristenson et al., 2011)

Structure-function relationship of SLC6 transporters:

The human transporters that mediate the presynaptic reuptake of monoamine neurotransmitters such as DA, serotonin and norepinephrine belong to the SLC6 gene family, a subgroup of the Neurotransmitter: Sodium Symporter (NSS) family. SLC6 proteins regulate the levels of extracellular concentrations outside of the cell by translocating neurotransmitters across the cell membrane. The SLC6 transporters mediate the cotransport of neurotransmitters in a sodium and chloride dependent mechanism (Chen, et al., 2004). Structural similarities of helices and residues exist between the SLC6 transporters. Proteins have hydrophobic α-helices spanning the membrane. The properties of a protein also differ based on amino acid residues; however, members of the SLC6 family share a high degree of similarity in their amino acid sequence. In particular, it is anticipated that the neurotransmitter translocation pathway within the different NSS members is formed by the similar positioning of α-helices in the proteins. Subtle differences in the residue arrangement are responsible for the specific transport of a particular neurotransmitter (Anderson Wang & Lewis, 2010).

Biogenetic studies on SLC6 transporters using LeuT :

An innovative breakthrough in the understanding of the structure-function relationship of NSS members came from the Gouaux laboratory by determining a high resolution crystal structure of the Aquifex aeolicus leucine transporter LeuT (Yamashita et al., 2005). LeuT (Fig. 2) has become the primary template for studying SLC6 transporters due to its high sequence similarity with its human counterparts. The transporter is a bacterial homolog of the SLC6 family and thereby amenable to genetic manipulation in order to produce large amounts of pure protein required for crystallization. Many detailed biochemical studies have been carried out to elucidate the molecular mechanism of NSS proteins using LeuT as a model system. LeuT co-crystallized with TCAs (tricyclic antidepressants) provide a glimpse of how drugs act on NSS at the molecular level. Together with functional studies, their mode of action could be explained (Shi et al., 2008).

Conclusion

Structural studies of transporters play a growing significance in future drug discovery and innovation. Expanding our knowledge of the structure/function relationship of the transporters involved in these central human processes will enable studies that are designed to develop new enhanced drug treatments (Anderson Wang & Lewis, 2010). Further pharmacological profiling of illicit psychostimulant (e.g. cocaine and amphetamine) abuse by means of observing and testing DAT can aide in specific drug addiction prevention (Chen et al., 2004).

Proposed project:

By applying the knowledge gained from the work with LeuT, the goal of my proposed research project is to identify the cocaine binding site in DAT and how the interaction of cocaine with the transporter affects the transport of dopamine by DAT.

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The Relationship Between Fine Particulate Matter and Diabetes Type II, by Dylan Gambardella

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Dylan Gambardella

According to the American Diabetes Association, more than twenty five million Americans (approximately one out of every twelve people) are directly affected by diabetes mellitus each year. Diabetes is a set of diseases, identified by high blood glucose levels, that result from the body's inability to produce and/or use insulin. The disease can be broken down into two main groups: Type I and Type II. These two variations of the disease differ greatly, each having distinct characteristics, such that they are almost not the same disease (American Diabetes Association, 2011).

Diabetes Type I is often known by the name "Juvenile Onset" for its occurrence in young children. The likelihood of being diagnosed with Type I diabetes is influenced mainly by one's genetic makeup and their immune system. Patients with this form of diabetes do not naturally produce the amount of insulin necessary to maintain normal blood sugar levels. This is caused by the patient's immune system manufacturing antibodies that destroy the insulin producing beta cells in the pancreas (Khardori, 2011). Therefore, these cases must be treated with insulin injections. All ethnic groups are equally susceptible to the development of Type I diabetes. Symptoms of a Type I diabetic include increased thirst and urination, constant hunger, weight loss, blurred vision, and extreme tiredness. Five to ten percent of all diabetes cases in the United States are classified as Type I (American Diabetes Association, 2011).

Diabetes Type II is often called "Adult Onset Diabetes," as it is mainly found in adults. This type of diabetes is known as “insulin resistant," and can be caused by genetic factors (as in Type I), and by inadequate insulin secretion in the pancreas. Insulin resistance is attributed to elevated levels of free fatty acids in plasma, and often leads to decreased rates of glucose transport into muscle cells. This allows glucose to remain in the blood to a degree of hyperglycemia sufficient to cause pathologic and functional changes in various target tissues (American Diabetes Association, 2004). Insulin resistance can also cause hepatic glucose production to elevate and an increased breakdown of fat (Khardori, 2011). Additional to the predisposing genetic factors, this type of diabetes can be attributed to an unhealthy diet, a lack of physical activity, and surprisingly pollution from the environment (American Diabetes Association, 2004). All ethnic groups are prone to the development of Type II diabetes, though the disease is more common in African Americans, Hispanics, Native Americans, and Asian or Pacific Islanders. Like Type I, patients with Type II can experience a loss of energy, the constant need to urinate, excess thirst, weight loss, blurred vision, frequent infections and slow wound healing. Type II accounts for over ninety percent of Diabetes cases in the United States (American Diabetes Association, 2011).

Air pollution is a known but unexpected factor in the prevalence of Type II Diabetes, as correlation but not causation between the two has been established (Pearson, 2010). A specific type of air pollution, fine particulate matter, is considered to be especially dangerous to humans. Fine particulate matter (PM) can be made up of toxic organic compounds, dust, dirt, soot, smoke, or heavy metals. PM is formed by burning plants, processing metals, and through automobile exhaust. Dr. W.E. Wilson describes the formation of particulate matter in detail, "The particle formation process includes nucleation of particles from low-vapor pressure gases emitted from sources or formed in the atmosphere, condensation of low-vapor pressure gasses on existing particles, and coagulation of particles," (W.E. Wilson, 2002).

One kind of particulate matter, PM2.5, categorizes all pollutants under 2.5 micrometers, and is considered to be especially harmful (Environmental Protection Agency, 2010). In a study conducted by M S O'Neill in 2006, long term exposure to PM2.5 was found to be associated with a higher risk of relative mortality among people with diabetes, when compared with the general population. Research from 2010 showed that ambient fine particulate matter was found to induce insulin resistance in mice, causing symptoms similar to Type II diabetes (Krämer, 2010). The Environmental Protection Agency credits the great health risks posed by PM2.5 to its ability to lodge deep within one's lungs and circulatory system. This is due to its minute width (approximately 1/30 the average width of a human hair).

Air pollution has long been considered detrimental to one's overall health (Brook, 2004). Recent studies have shown the effects of particulate matter on various bodily functions. In 2004, it was concluded by the American Heart Association that short term exposure to PM led to an increased number of hospital admissions and cardiovascular mortality (Brook, 2004). In addition, PM expo-

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sure has been observed to alter the platelet activation process in humans, leading to homeostatic irregularities (Emily Cozzi, 2007). Fine particulate matter particles can also reach the circulatory system, causing obstructions and effecting blood flow (O'Neill, 2006). This type of blockage can lead to the formation of blood clots, which are known to be the leading cause of strokes (American Stroke Association, 2012). Fine particulate matter is capable of interfering with the lungs, as well, by depositing particles in the lower airways (gas exchanging portions of the lung), effectively disrupting the respiratory system and leading to numerous breathing problems (O'Neill, 2006).

Though scientific research has demonstrated a relationship between Diabetes Mellitus and air pollution (more specifically fine particulate matter), experts have yet to find the underlying cause. Epigenetic changes could possibly be a reason for the association between diabetes prevalence and air pollution. Epigenetic traits result from changes in the chromosome, but not the DNA sequence (Salam, 2012). Exposure to particulate matter may affect one's epigenetic makeup, conceivably leading to health concerns such as diabetes. I wish to pursue the plausible connections between epigenetics, air pollution, and diabetes in future research.

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Stem cells and Cardiovascular Disease, Stanley Fray

Stem cells are any cells that can reproduce rapidly in an undifferentiated state; these cells can increase differentiated cells or various tissue types in the body; these cells also can divide for undetermined amounts of time which can increase specialized cells or varieties of tissues in the body (Verdeil, 2007). Stem cells are capable of self-renewal or proliferation as well as differentiating into a range of variety of cell types under proper conditions (Zhang Qi, 2005). These different types of cells can be found in an assortment of locations in the body, during both embryonic development and in adulthood. Embryonic stem cells can be harvested from the blastocyst when they are four to five days old; at this point the embryo contains 150 undifferentiated cells (Mayo clinic staff, 2011). There are also adult stem cells which can be found in a choice of places all over the body, in tissue of the brain, blood vessels, skin, teeth, skeletal muscle, the heart, the liver, and bone marrow (Bethesda, MD, 2008). Prockop's (2004) study found the following advantages and disadvantages of embryonic stem cell (ES) versus adult stem cells:

"One answer is that ES cells can readily be shown to differentiate into essentially all cell phenotypes, whereas most isolates of adult stem cells from sources such as bone marrow stroma, fat, muscle, and nervous tissue have a more limited potential for differentiation. Also, most but not all isolate of adult stem cells have a more limited life span in culture the ES cells have." (para. 1).

However, these stated advantages and disadvantages can only become valid if one would assume that the scientist has worked with stem cells before, and that he was smart enough to invent the proper living conditions for adult stem cells (Prockop, 2004). Through stem cell development there are different levels of potency for example, multipotent is the ability of one stem cell to develop into more than one cell type in the body, pluripotent cells have the capability for one stem cell to create availability towards most but not all cell types in the body, totipotent stem cells will give increase towards

all cell types that make up the body, and unipotent are stem cells that will only develop into one cell or tissue type in the body (Verdeil 2004). Furthermore there are iPSC (induced pluripotent stem cells) these adult stem cells have been altered to embryonic cell state by showing the cells genes of important properties of embryonic stem cells (Bethesda, MD, 2008). iPSCs are very important for with them the demand for embryos will go down, thus there being less controversy on the use of fetuses in labs; for in some states the use of aborted or miscarried fetuses illegal. In addition there are heart/cardiac derived stem cells; these cells are derived from the . Bone marrow cells are extracted from bones can be harvested and further differentiated into heart cells (Philippe, 2008). Correspondingly there are mesenchymal stem cells (MSCs and bone marrow cells), these multipotent progenitor stem cells have been shown to differentiate in vitro or in vivo; MSCs have loosely organized undifferentiated mesodermal cells that give rise to different structures including fat, bone, cartilage, and skeletal muscle (Ott, Davis & Taylor, 2005). Transdifferentiation is the process in which a stem cell of one certain tissue differentiates into cells of another tissue ("Glossary," 2011). Stem cells can play a major role in finding ways treat cardiovascular disease and myocardial infarction.

Myocardial infarction (or heart attack) is when the blood flow to a heart is blocked for a long period of time long enough for the heart muscle to become damaged or die because of the lack of oxygen rich blood reaching the heart. Diseases of heart (myocardial infarction, CAD, cardiovascular disease) are number one in the top ten leading causes of death in the United States as of 2007 ("Leading causes of," 2007). The diseased coronary arteries (CAD) of the patient have been blocked due to plaque buildup in the arteries, the lack of blood circulation in the heart results in cell muscle death ("Myocardial infarction heart," 2011). When the muscle dies the heart shuts down and stops pumping oxygen rich blood resulting in mortality. The contributing factors towards cardiovascular disease are smoking, high cholesterol, poor lack of exercise, use of drugs such as cocaine or amphetamines, prior records of coronary artery disease (CAD), and previous myocardial infarction or strokes ("Myocardial infarction heart," 2011). Family genetics, as well as lifestyle are important in predisposing patients toward myocardial infarction. To take care of myocardial infarction there are two things you could do, either partake in the numerous treatments for myocardial infarction or change your lifestyle choices. One treatment is to open up the clogged coronary artery by using either medication or a procedure called an angioplasty ("Myocardial infarction heart," 2011). The anti-platelet medications like aspirin and Plavix stops the buildup of platelets which are tiny little plugs which play a major part when it comes to clogged arteries; those medications are helpful when it comes towards preventing future heart attacks ("Myocardial infarction heart," 2011). Following this further there are also angiotensin converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs), these two medications diminish resistance of how hard the heart has to pump against; the two also help when it comes to preventing enlargement of heart after a heart attack ("Myocardial infarction heart," 2011). There is also procedure that a patient with CAD can undergo which is an angioplasty ("Myocardial infarction heart," 2011). An angioplasty consist of a balloon tipped catheter inserted into a blood vessel in the patients leg or arm which goes to a blood vessel connecting to the heart, after reaching the blood vessel connecting to the heart the balloon tipped catheter passes through the clog artery inflates then deflates causing the artery to increase in size, then a stent is placed in restoring to blood flow to the artery ("Myocardial infarction heart," 2011). Both the medication approach and procedural approach towards cardiovascular disease are equally successful in their own ways, but neither of these approaches involves the use of stem cells.

There have been various experiments towards treating and repairing heart due to cardiovascular disease. In a cliniical trial done by Ott et al. their experiment compared different cell types in cellular cardiomyoplasty, a procedure in where a healthy muscle (in this case a skeletal muscle) surrounds muscle of a failing heart, hoping that as a result it will cause the hearts damaged muscle to heal (Ott, Davis & Taylor, 2005). A type of adult MSCs were used skeletal myoblast (SKMB) and bone marrow mononuclear cells (BM-MNC) were used to see which one would be more effective in healing damage done to the left ventricle of a rats heart (Ott, Davis & Taylor, 2005). The experiment was in vivo using a live rat in the trial; the cells were injected into the infracted part of the heart and the outcome was that the SKMBs were superior to the BM-MNCs, for the left ventricle function in the rat's heart had increased (Ott, Davis & Taylor, 2005). Resulting that although one cell type was superior to the other the combination of both cells types being used shows a synergetic in the damaged heart which can then be used for future reference (Ott, Davis & Taylor, 2005). The vari-

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ous problems that are to come in the future that Ott et al. (2005) stated was that, "the timing and technique of cell delivery and what type of cell should be optimized for various types of cardiac injury" (pg. 358), (Ott, Davis & Taylor, 2005). Another obstacle yet to be prevailed is the survival rate of transplanted cells, to take into consideration is that 70%-90% of transplanted cells die in the first few days after being injected into the infracted scar; there is also immune rejection where the body rejects or the immune system attacks the injected cells (Ott, Davis & Taylor, 2005). Although there was success in the clinical trial, there are still many factors keep scientist from achieving what they want from stem cells.

Firstly there are many stem cell types that can be used in clinical trials both in vivo and in vitro, from adult stem cells to embryonic stem cell to MSCs, Skeletal muscle cells and so on; can all be used in various ways towards treating cardiovascular disease. Secondly myocardial infarction has many of its own procedures and medications that work already but there is also room for new treatments for heart attacks with the use of stem cells. Lastly although there have been success with some experiments there are still many hurdles that scientist have to overcome towards getting the full potential and use of stem cells. All in all stem cells research is slowing making its way towards perfection.

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Helmet Designs Development for Concussion Prevention, by Justin Fischer

Do you know what a concussion is? Do we really know how to effectively diagnose a concussion on sight? The current system used to test concussions is not very accurate, it's mostly based on the physician's judgment; (Schatz, 2005) therefore we do not really know that much about how to diagnose them. There are many definitions for a concussion, but the most reliable definition of a concussion is head trauma caused by a great hit or force against a part of the head (WebMD, LLC; 2012).

If you can find out a way to prevent a concussion you would never have to deal with them. Ivy league football teams have attempted to study this. They found out that the best way to prevent a concussion is to minimize your chances at receiving them. Certain coaches at certain Ivy League schools agreed to minimize their full-contact practices in a week (New York Times, 2011). They had two full contact practices a week rather then the NCAA limit which is 5, and during the preseason they would only be able to hold one full-contact session, in a two-session practice. Overall the full-contact practices year round would be reduced 42%. The league also wanted to emphasize teaching proper tackling during practices. Statistics also show that a lot of concussing hits occur during practice, with each team experiencing about 200 practice collisions that would have caused a concussion, year round. So the league concluded that not only will this movement, cause less concussions, but it will also keep the players fresh during the week and before their games (New York Times, 2011).

The diagnosis of a concussion relies on many symptoms. There are four different main signs of a concussions, physical symptoms, cognitive symptoms, emotional symptoms, and sleep symptoms. The physical symptoms are (but are not limited to) headaches, nausea, vomiting, balance problems, dizziness, visual problems, fatigue, sensitivity to light, sensitivity to noise, numbness/tingling, and being dazed or stunned. Cognitive symptoms are feeling mentally foggy, feel-

ing slow, concentration difficulties, memorization difficulties, forgetting or being confused about recent information or conversations, repeating of questions, or slow in answering questions. The emotional symptoms are irritability, sadness, nervousness, and being more emotional. The Sleep symptoms are drowsiness, less sleep usual, more sleep usual, or just trouble falling asleep. Another huge way physicians diagnose concussions is the spot on the head or neck where they were struck and how attentive they were when the hit happened. A person who was struck without seeing the hit coming is more susceptible to injury then a person who saw the hit coming. That is when you see the hit coming; naturally your muscles tighten up creating a protection. And if you are struck right on the cranium rather then the lower part of your face, you are much more likely to get a concussion. All of these symptoms are what physicians, or trainers use to test if a person has a concussion and its severity (Concussion Diagnosis, CDC, 2011).

These injuries can cause many short-term, and long-term disorders. These disorders consist of early development of epilepsy, or dementia for the long term. For the short term, there are accumulated headaches, nausea, blurred vision, memory loss, and difficulty doing cognitive or athletic activity (Concussion diagnosis, CDC, 2011). A lot of these short-term disorders are used as indicators for seeing if the designated person has to take the ImPACT test, which is taken as a baseline test before the seasons start. Then taken again if the person is suspected to have a concussion, and if the person scores significantly lower on the second test then the person is diagnosed with a concussion. These indicators are not used to diagnose a concussion, because players normally experience a few, but not all of these symptoms mildly due to physical activities (Rice, 2008).

Concussion amounts, and concussion severity also varies by the location at which the head that was struck. Studies also show that multiple blows to the head can be more severe then one clean hard strike. And as stated before, most of these blows occur during practices rather than games. But the positions that suffered the most amounts of hits per season, during practice and during the games were the defensive lineman, next being the linebackers, and then offensive lineman right behind them. Followed by running backs, then Defensive backs, and last being wide receivers and quarterbacks. But the lineman or linebackers did not attain the most severe, or hardest hits; it was actually the quarterbacks and running backs that suffered the hardest hits. With the linebackers and wide receivers receiving the hardest after, and then defensive backs, and lineman being last. Also quarterbacks and wide receivers received the most hits in the back of the head, which could explain why the very few hits on the quarterbacks to the head are the most severe and also why wide receivers, which receive the smallest amount of hits on the field, have one of the highest amounts of severe hits. Lineman, and linebackers received more of their hits to the front of their heads, which could explain why they have less amounts of hard blows (Crisco, 2011).

This also proves that an accumulated amount of hits can be more severe then just a few concussing hits, in the long term. But in the short term the very few hard hits can be extremely harmful as the short term blows can cause the symptoms shown in the above paragraphs. And the long-term symptoms are obviously more harmful in the long run. So that is why there are more severe long-term injuries induced onto retired lineman and linebackers, rather then retired running backs or quarterbacks.

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Justin Fischer

Sophomores Projects

Edward Gilman

Enhanced Hydrophobic Properties in Cotton-based Fabrics, Edward Gilman

There are many characteristics that go into a "high performance" garment. The Ideal performance fabric is breathable, waterproof, and, depending on weather, it lets air flow freely or keeps air to the outside. This can be achieved in a number of different methods. Densely woven waterproof breathable fabrics consist of cotton or synthetic microfilament yarns with a compacted weave structure. Laminated waterproof breathable fabrics are made by the application of membranes into textile product, which can be either Micro porous, or hydrophilic. Coated waterproof breathable fabrics consist of polymeric material applied to the surface of fabric. (Lheritier,2005)

All of the listed textile types make for eleven key characteristics: optimum heat and moisture regulation, good air and water permeability, rapid moisture absorption and conveyance capacity, absence of dampness, rapid drying to prevent catching cold, low water absorption of the layer of the clothing just positioned to the skin, dimensionally stable even when wet, durable, easy care, lightweight, soft and pleasant touch. (Chaudhari,2005)

The most important of those factors is wettability-the ability for a fabric to retain water- although there is a different measured level of comfort for each side of the garment; moisture must be let out, and kept outside as well. A layer of fabric must let moisture out (perspiration), but keep moisture out (precipitation). Some fabric constructions that can achieve this are Laminated Waterproof Breathable Fabrics, made of a membrane applied into the textile, usually made from a polymeric material that offer resistance to water penetration, but allow water vapor to pass through at the same time. Also used is a Hydrophilic membrane, a thin film of modified polyester or polyurethane. (Chaudhari, 2005)

The skin and garment create a multiphase atmospheric microclimate, which should result in a proper contact state and a temperature and humidity balance for human body comfort and protection. Mild abnormality of the garment/skin regulatory interaction itself could lead to such uncomfortable consequences such as tickling, rushing, and blistering. These irritations would become catastrophic in athletic competition or during a military mission when reduced performance or mobility becomes adversely consequential or even fatal. This alone creates a need for the study for the understanding of interactions between garment and skin under various conditions. (Pan, 2005)

What allows water vapor to move freely through their membranes, but keep out liquid water, is their moisture transport mechanism. Textiles transport liquids much like capillaries. Spaces in between fibers effectively form capillaries, allowing moisture to move upwards through the fabric. But capillary action ceases when all parts of the fabric are equally wet. This is why the textile must be impenetrable by water (Xue, 2008).

While superhydrophobic surfaces are excellent at repelling water, it is very hard to manufacture them so that water vapor is allowed to move upwards through them. It is the property that these surfaces lack that regulate body temperature, improve muscle performance, and delay exhaustion. Its applications are not limited to fabrics: Hydrophobic treatments can be used in an array of different ways, but at this point is an underdeveloped technology. As of now the only way to artificially achieve superhydrophobicity is through an acid based treatment, usually consisting of some form of Silica (Xue, 2008).

In a 2008 study by the College of Resource and Environment, Shaanxi University of Science and Technology, different fibers were coated with Titanium Oxide solution to generate a dual –size surface roughness. The following hydrophobization process used steric acid and a combination of other acids made hydrophobic fibers superhydrophobic. (Xue, 2008)

Superhydrophobicity is often measured by water contact angle- the angle at which water is repelled from a surface upon impact. The higher the contact angle, the more water repellant the surface is.

While there are treatments that can alter the cotton textiles levels of hydrophobicity, not effective process has been yet developed to modify cotton on the molecular level, so that they acquire superhydrophobic properties.

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The Effects of Media on Adolescent Body Image and Perception of Beauty (Abstract), by Jordanna Jervis

The media has an enormous impact on its viewers in terms of body image, fashion/style, physical appearance, and many other aspects. Adolescent/ Teenage boys and girls are more prone to be influenced by what they see in the media (i.e. TV, internet, and magazines); and want to mirror what they see. This is probably due to the fact they have yet to develop self- confidence, and certainty of who they are or who they wish to be. In the near future, several surveys will be conducted to evaluate body image, self-perception, and influences of the media on both males and females attending middle or high school. Correlations between how girls and boys feel about the way they look, based on what they see in the media will be measured. The way boys and girls feel that the opposite sex should look (on average), or how they want their ideal guy/girl to look will also be determined. Using online surveys and targeted interviews, image perception and attractiveness of different body types and facial features will be evaluated. On the survey, participants will be asked their age, gender, BMI, a self-evaluation of their body type/shape, level of self-esteem, how often they exercise, media consumption, and family background to study what factors may influence their opinions. Participants with also look at various pictures of different body types and facial features and rate the attractiveness from a scale of one to ten (one being the least attractive and ten being the most attractive).

This will help us determine potential causes for eating disorders due to low self-esteem and negative body image perception in adolescents.

Jordanna Jervis

Generation of Deja Vu Experiences Using Virtual Reality Scenes for Neurological Studies, by Matt Kenny

Freshmen Projects

Many people experience the phenomenon of deja vu: it comes at a frequency of once a year. Despite this, scientists have not been able to fully understand the way in which deja vu happens. Deja vu is directly translated into french as "already seen." It feels as if you have already been in a situation numerous times, where you are really in for the first time (serendip 2009). The experience of deja vu is defined as an erroneous feeling of having already done, seen, heard, thought, or visited once before.

Many hypotheses have been formulated to explain how deja vu occurs, but all seem to have some evidence that disproves them. Most of these hypotheses have been limited to only certain types of deja vu. For example, one hypothesis that was trusted by many is the "optic pathway delay" hypothesis. The optic pathway delay hypothesis states that there is a delay in between the eyes actually seeing an event occur and the brain processing it into a recent memory. Because of this delay, rather than the event go into the "short term memory file," the event will "skip" the short term memory and therefore go straight into the long term memory of a person (O'Connor 2006).This hypothesis would explain the reason as to why some people feel as if they have the ability to predict what will happen next and feel as if they know what will then happen. Because the event becomes a long term memory, the person experiencing deja vu may think that once something happened, they could have been able to predict this. This theory was challenged when a blind man was found to have experienced deja vu frequently, in fact more than the average person of his age, through the age of thirty (O'Connor 2006). This shows that it is not necessary for someone to have a visual delay with the brain in order for someone to experience deja vu. It is not known why, but this man was not born blind, but progressively lost his sight until he was completely blind by the age of ten. Despite the inability to see, this man still lives a normal life as compared to humans with the ability to see, and is very normal in terms of his health. He is totally similar to other humans other than the fact that he is blind, yet still experiences deja vu.

Many feel upset after going through the experience of deja vu. For that reason, it would be important to stop it at certain times. Understanding the neurological process behind deja vu would allow us to monitor and record data on it. If deja vu can be stopped, then the feeling of confusion and sadness as a reaction can be taken away. Also, a precursor of an oncoming seizure is a feeling of deja vu (Adachi 1999). There may be a possibility that deja vu is a cause of a seizure or generates it. Therefore, by knowing how deja vu arises and stops, there may be a way to stop or diminish temporal lobe epilepsy's consequences.

Matt Kenny

Freshmen Projects

The most believed and trusted theory as to how deja vu works is the optical pathway delay theory, which states that there is a malfunction between the eyes actually seeing what is going on and the brain processing this into a memory. Another theory to the deja vu phenomenon is the "cell phone" theory. This theory states that when a person is focusing on one specific thing, they are still able to see the surrounding objects through their peripheral vision. The person seems consious of the object being there without fully focusing on it. Then, when the person fully focuses on the object, they feel as if they remember it from somewhere else (Obringer 1998-2012). This hypothesis is interesting because it poses the question: could it be possible for someone to provoke deja vu? If so, then scientists would be able to provoke it and would be able to generate models to study deja vu efficiently; Currently, deja vu cannot be studied due to the fact that it only arises about once a year and the time in which this phenomenon will take place cannot really be predicted. Therefore, with this hypothesis, we could collect data about it, and further understand how this common phenomenon really does work. However, this theory is challenged constantly because of the fact that to this date no scientist has been able to provoke deja vu.

Deja vu is also very common in patients with schizophrenia. Of all people, the ones with schizophrenia experience deja vu far more than anyone else. Therefore, there is a large possibility to use patients with schizophrenia in order to receive more in-depth information on deja vu and its functioning (Adachi 1999). So, therefore, why has no scientist done this yet? The only answer to this question would have to be moral ethics. The patients with schizophrenia would receive medical problems if they were to experience deja vu. Therefore, if these people are constantly forced to experience deja vu, they could legitimately be out at risk for some very serious medical complications (Adachi 1999). Therefore, scientists unfortunately are not able to work deeper into the field of deja vu by testing on anyone with schizophrenia.

There may be a possibility that deja vu is not just a common phenomenon that everyone can experience without any medical problems being created. The feeling of deja vu could be an extremely small seizure (serendip 2009). This could mean that normal humans and humans with temporal lobe epilepsy would really have a lot of similarities. This, again, would help patients with temporal lobe epilepsy. Scientists would be able to relate what happens to patients with temporal lobe epilepsy to what happens to normal humans. Also, many patients with temporal lobe epilepsy take medication in order to stop their seizures. In order to stop deja vu, people could possibly take much smaller doses of these medications to stop their seizures. Deja vu is obviously connected so many ways with temporal lobe epilepsy.

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The Accuracy of the Correlation of SPECT Imaging and Ethomatic Software in Determining the Lateralized Zone of Epileptogenesis, by Jonathan Young

Epilepsy is a condition that affects more than three percent of the world's population. It is a disorder that affects the cerebrum or the part of the brain that contains motor functions, language, logic, etc. Epilepsy as a result has a wider range of variability in its presentation, and can affect any brain function that someone would have control over. For this reason epilepsy can present as: Spitting, vomiting, speaking, screaming, babbling, hallucinations, paranoia, automatisms [actions performed automatically, often with the person performing the action not realizing they had done anything], tonic-clonic posturing [this presentation is the commonly known shaking and convulsions], and in some complex seizures just blankly staring.

However the best way to diagnose a seizure of any sort is for a neurologist to apply electrodes to a patient's scalp, in a special pattern which picks up electrical activity from all parts of the brain, and perform a neurological exam called an Electroencephalograph or EEG. The EEG monitors and analyzes a patient's brain function and wave patterns. During the inter-ictal period a patient's wave patterns should be smooth and rhythmic, but during the ictal period the patient's EEG would show Electrical Seizure Activity in the form of spikes on the wave patterns indicating neurons [brain cells] firing electrical impulses randomly. In the field of epilepsy these random neuronal discharges are called Periodic Epileptiform Discharges or PLEDs. (Hartings 2003)

In over half of all recorded cases of epilepsy the patient has an intractable form of epilepsy in which case a Resective surgery is the indicated therapy. A resective surgery is basically the removal of damaged tissue. For a neurosurgeon to operate on someone's brain they would have to have an exact location because just a millimeter off could kill the patient or leave them mentally or physically disabled for life. To get this degree of precision before this new alternative way was found to be effective would require a Pre-Surgical examination, an MRI and PET scan both with contrast material, a Video/EEG which records the EEG results. This process could take years and since the first resective surgery on an epileptic was performed there has been no alternative. (Bertti 2010)

Very recently a new alternative to this method has been established. It is faster and more effective than the other method. This new method requires Ethomatic software correlated with an imaging technique called SPECT or Single-photon emission computed tomography. In this imaging technique a radionuclide, or radioisotope that releases gamma waves, is injected into the bloodstream of the patient and an image is taken during a seizure showing the amount of blood flow to different parts of the brain. The area to which the largest amount of blood is delivered to is the probable zone affected and can most likely be removed. However to receive the complete picture they use the ethomatic software. The Ethomatic software correlating the imaging and the recorded behaviors organizes the data into a flowchart. (Bertti 2010)

The neurologist looks at the flowchart and can lock down the location to one of the hemispheres and on occasion even the lobe. Final tests show the exact location of the Epileptogenic Zone and they inform the neurosurgeon of its location.

Once the neurosurgeon knows where to operate the patient can have the surgery to remove the damaged brain tissue. They are carefully watched for two years on an outpatient basis and after that will be declared seizure free. This would all take a little more than a year not including the two years after the surgery.

With further research this new method can be used for the localization of the epileptogenic zone. In a healthcare setting this can be revolutionary greatly increasing the amounts of patients being able to have the surgery and be seizure free, possibly indefinitely. However this technology is only just starting to be studied in humans.

Jonathan Young

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Acknowledgments

Mentors and Scientists:
Thank you to all of the scientists who have taken our high school students under their wings and given them unprecedented possibilities to access genuine scientific research, to act as professionals and to express their talents and creativity in their projects.

- Dr. Matthias Quick, Columbia University.
- Dr. Steven Suib, University of Connecticut.
- Dr. Teresa Wood, New Jersey Medical School And Cancer Center.
- Lauren Rota, New Jersey Medical School And Cancer Center.
- Dr. Deborah Lazzarino, New Jersey Medical School and Cancer Center.
- Dr. Herbert Lachman, Albert Einstein College of Medicine.
- Dr. Todd Gardner and Chris Paparo, Atlantis Marine World Aquarium, Riverhead.
- Dr. Kathleen Keller, Columbia University College of Physicians and Surgeons
- Dr. Kathleen Curtis, Oklahoma State University
- Professor Jeffrey Holly, Bristol University, United Kingdom.

Sponsors:
I want to thank the **Hendrick Hudson Community Educational Foundation (HHCEF).** We have received on-going funding from the HHCEF to support this program over the years. The HHCEF support has been instrumental in providing equipment, covering registration fees for competitions, travel funds and logistic support to the program.

Parents:
This program would not be successful without the support of the students families, who sacrifice so much of their time to ensure that the students get the best possible experience when they work on site, or in labs and during the school year.

Administrators, Teachers and Staff:

- Dr, Daniel McCann, Superintendent.
- Dr. Alice Gottlieb, Assistant Superintendent for Curriculum.
- Enrique Catalan, Assistant Superintendent for Business.
- James Mackin, Hendrick Hudson High School Principal.
- Daniel Callahan, HHHS Assistant Principal.
- Anecia Bell-Jefferson, HHHS Assistant Principal.
- Mathew Swerdloff, director of technology.
- Beth Gruber, HHCEF
- Catherine Johnson, HHCEF
- Marie Green, HHCEF
- Everton Henriques, Science Teacher for his help with printing large size posters.
- Laurie Small and Jill Wheeler for their sustaining help and support.
- Clare Carey for catering services.
- The Custodial staff.

Science Research Colleagues:

- Michael Blueglass, Science Research Teacher, Yorktown High School for providing valuable advice and help.
- Angelo Piccirillo, Science Research Teacher, Ossining high school.
- Donna Light-Donovan, Science Research Teacher, Croton-on-Hudson.
- Leonard Behr and Dr. Dan Wulff, Science Research in the High School Program, University at Albany.

Members of the School Institutional Review Board:

Thank you for taking the time to review students projects throughout the year.

- James Mackin, principal.
- Eileen Cancro, Science teacher.
- Dr. Jennifer Lupiani, School psychologist.
- Kathy Frost, School nurse.

Notes