

Hendrick Hudson
High School

Science

MAY
2013

Research



“YOU ARE LIMITED ONLY BY YOUR IMAGINATION”

► AUDREY HECHT AND ALEXIS MANGUAL PARTICIPATED AND WERE AWARDED AT WESEF THIS SPRING.

○ MAY 2013

**Hendrick Hudson
High School**

► DR. CHRISTINE ROGERS RECEIVED A 2013 INTEL STS TEACHER BADGE.

► ALEXIS MANGUAL RECEIVED A 2013 INTEL STS RESEARCH REPORT BADGE FOR AN EXCELLENT PAPER.

Science Research

"YOU ARE LIMITED ONLY BY YOUR IMAGINATION"

DR. MARCO CASTALDI, ASSOCIATE PROFESSOR, CUNY

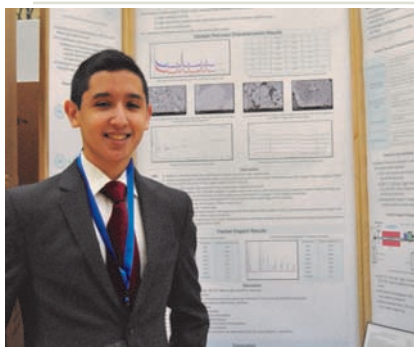
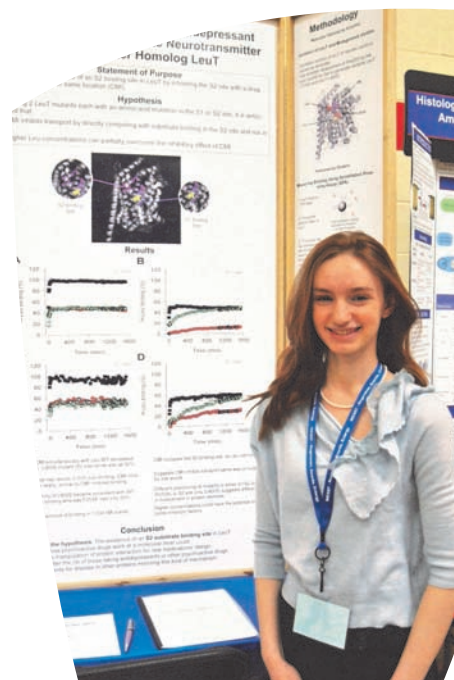
Hen Hud Science Research students compete in WESEF and JSHS.

Over a period of three years, students in the science research program engage in designing, implementing and presenting scientific research experiments, which are conducted with the help of scientists, often at well renowned academic institutions. They learn important skills, such as reading and researching bibliographic references, planning research experiments with the correct methodology and presenting their research findings in presentations, written scientific publications and posters. They also learn how to interact with scientists in the professional environment. Students earn up to 12 credits at University at Albany (SUNY). These research and professional skills are critical for success in college and this program is well regarded and recognized by universities.

This year, Audrey Hecht, a junior, won First place in the Biology category at the Westchester Rockland Junior Science and Humanities Symposium (JSJS), Second Place in the Tri-County Competition and 4th place at the

Westchester Science and Engineering Symposium (WESEF). Audrey will continue working with her mentor, Dr. Matthias Quick at Columbia University, over the summer and compete again as a senior. Alexis Mangual, a senior, won 5th place at JSJS in the chemistry category and received the Momentive Performance Materials Award, for top projects that demonstrate innovation, sustainability, productivity, and/or use of a catalyst as an agent for change for his project on fuel cells catalysts.

Please read excerpts of Audrey and Alexis' INTEL papers below. Other papers presented in this edition are the midterm papers and introductory paragraphs of research proposal written by students in the program. Their projects are all at different levels of development, as some students already are working with scientists, while other students are still looking for mentors.



A novel copper doped cobalt carbonate catalyst reduced from copper doped cobalt oxide for Fischer-Tropsch synthesis (INTEL STS paper excerpt) by Alexis Mangual (Senior)

Introduction: The modern world needs to find a new and renewable source of fuel in response to the dwindling supply of oil if society is to thrive for many years to come. Fuel cells and nuclear energy are long-term solutions to the global energy demand; however, the global infrastructure is not ready to phase out hydrocarbon-based fuels as the main energy source. The world may never be able to fully eliminate

its need for hydrocarbon-based fuels. The Fischer-Tropsch (F-T) process presents itself as a viable and near-term solution to the diminishing global oil reserves. The F-T process converts hydrogen and carbon monoxide, usually obtained from coal, methane, or biomass, into a useable hydrocarbon. F-T plants can be used to synthesize both saturated and unsaturated hydrocarbons which have a wide variety of applications including fuels. As a byproduct of the reaction, water is formed. The chemical reactions involved in the conversion of CO and H₂ to hydrocarbons in Fischer-Tropsch rely on the presence of a catalyst. The catalyst in this reaction allows for the dissociation of the carbon-oxygen bonds in order for the carbon to react with hydrogen to form hydrocarbons. The presence of a catalyst is required in order to make Fischer-Tropsch synthesis an efficient and viable process.

There are a multitude of catalysts that have been suggested for use in the Fischer-Tropsch reaction. The catalyst determines what the reaction will yield in terms of a usable product, how much of that product is produced in relation to byproducts other than water formed (selectivity), how much product is made overall (yield), and how quickly the product is formed (rate of reaction). Despite all the catalysts discussed for the Fischer-Tropsch reaction, there remains a need for further optimized catalysts. Catalysts need to display greater yield, higher selectivity, faster rates of reaction, and more useful final products. Many Fischer-Tropsch catalysts tend to result in the formation of hydrocarbons that are not as desirable as liquid fuels.

Currently, the most widely used catalysts for the Fischer-Tropsch reaction are the transition metals cobalt and iron. Other metals used as catalysts in the reaction include ruthenium, nickel, and manganese. Cobalt metal catalysts tend to display high activity in the Fischer-Tropsch reaction when the feedstock has high hydrogen content. In cases where the feedstock has low hydrogen content, as in the case of coal, Iron based catalyst are preferred as they promote the water gas shift reaction which converts carbon monoxide and water into

carbon dioxide and hydrogen. Aside from pure metal catalysts, a variety of other catalysts have been proposed for the Fischer-Tropsch reaction.

Catalysts for the Fischer-Tropsch reaction must also demonstrate a certain level of resilience to the harsh conditions inside the reactor. Temperatures inside an F-T reactor generally vary between 150 and 300 degrees Celsius. Conditions within an F-T reactor also involve very high pressure as greater pressure correlates to higher conversion rates and the production of long-chained alkanes from the reaction. Fischer-Tropsch catalysts are also very sensitive to sulfur-containing compounds and can degrade rapidly in the presence of high concentrations of sulfur. Cobalt based catalysts, while generally being more active than other catalysts, can demonstrate a greater sensitivity to sulfur than other catalysts.

A new catalyst for the Fischer-Tropsch reaction must be capable of resisting poisoning by sulfur-containing compounds, has to demonstrate high activity in the reaction, must be able to withstand the harsh conditions inside the reactor, and should result in the formation of more useful hydrocarbons versus less useful ones such as methane. Iron based catalysts are less active than cobalt based catalyst and are generally only more useful than cobalt if the feedstock is of low quality, such as in the case of coal or biomass. Cobalt based catalysts tend to be less expensive than other catalyst options increasing their viability in comparison to other catalysts.

Cobalt metal based Fischer-Tropsch catalysis presents a series of advantages and disadvantages in comparison to other catalysts. Cobalt metal catalysts, as previously stated, generally display higher activity than other catalysts and are more adequate for low-temperature Fischer-Tropsch with a high quality feedstock. Cobalt metal catalysts, however, are more

susceptible to deactivation. There are a multitude of causes for the deactivation of cobalt catalysts in F-T the main ones being poisoning, re-oxidation of active sites, formation of surface carbon species, carburization, surface reconstruction, sintering, solid state reactions between support and catalyst, and attrition (Tsakoumis, 2010). The variety of ways in which cobalt catalysts can be deactivated highlights the need for improvement in F-T catalysis.

Iron metal based catalysts tend to be less active but more resilient than cobalt catalysts. They are still susceptible to deactivation but not nearly as susceptible as cobalt-based catalysts. Deactivation of iron catalysts in F-T can result from the formation of inactive carbide phases and surface carbonaceous species like graphite (Nakhaei, 2010). There are still more ways in which Iron catalysts can be deactivated. Iron catalysts may be more resistant to deactivation than cobalt catalysts, but the lower general activity indicates the necessity for catalysts improvement in Fischer-Tropsch catalysis.

Catalyst based on other metals lack the advantages that cobalt and iron based catalysts present in F-T. Metals other than cobalt and iron are generally used as catalyst promoters in the Fischer-Tropsch process. Promoters can increase the activity of Fischer-Tropsch catalysts and can greatly affect what the main catalyst will tend to synthesize. For instance, manganese has been shown to decrease methane selectivity and increase C₅₊ hydrocarbon selectivity in cobalt catalysts while increasing catalytic activity. Copper has also been described as a promoter Iron as a Fischer-Tropsch catalyst. While not considered an effective promoter for cobalt in Fischer-Tropsch, copper may have a different impact on catalysis when the catalyst is not cobalt metal.

One potential replacement for pure metal catalysts in Fischer-Tropsch Synthesis is the metal

MENTOR Dr. Steven Suib, Ph.D.



Board of Trustees
Distinguished Professor, Department of Chemistry, University of Connecticut.
We are very grateful for the help and dedication of Dr. Suib, as well as his graduate students, Curt Guild and Dave Kriz, for their guidance and help with Alexis Mangual's project.

carbide. Iron carbides and molybdenum carbide were proposed as a catalyst for Fischer-Tropsch in multiple journals (Ding, 2011). Molybdenum carbide as an F-T catalyst demonstrated moderate activity and high selectivity towards C₂₊ olefins (Vo, 2011). The effectiveness of molybdenum carbide as an F-T catalyst suggests that cobalt carbide may also be an effective Fischer-Tropsch catalyst. Copper could potentially be a promoter to a cobalt carbide catalyst despite its ineffectiveness as a promoter to cobalt metal.

Another group of catalysts reported as viable Fischer-Tropsch catalysts are metal oxides. Iron oxide nanoparticles were reported as having high selectivity towards C₅₊ hydrocarbons. Similarly, cobalt/manganese oxides were described as being very practical due to their production of an industrially viable product spectrum (Everson, 1998).

The reduction of molybdenum oxide to molybdenum carbide prior to F-T catalysis was also described in recent literature (Vo, 2011). Both metal oxides and metal carbides show

potential as catalysts in Fischer-Tropsch synthesis. Using a metal oxide, such as cobalt oxide, as a precursor to a metal carbide catalyst could have a positive effect on the catalytic performance of the metal carbide in F-T. As total reduction of the metal oxide will likely not occur, a synergistic relationship could emerge between the metal carbide and metal oxide resulting in increased catalytic activity.

As previously described, copper could potentially be a promoter to a cobalt-based catalyst despite its inadequacy as a promoter to pure cobalt metal. Copper doped cobalt oxide nanoparticles reduced to cobalt carbide could potentially demonstrate high activity in F-T while maintaining greater selectivity towards more desirable products. This proposed cobalt-based catalyst has the added benefit of being very inexpensive to produce and, therefore, economically feasible.

Purpose: The purpose of this research project was to develop a novel and inexpensive Fischer-Tropsch catalyst that would exhibit high catalytic activity and demonstrate an excellent selectivity towards a variety of industrially feasible Fischer-Tropsch products.

Hypothesis: Synthesized copper doped cobalt oxide nanoparticles reduced *in situ* to cobalt carbide would demonstrate high catalytic activity in the Fischer-Tropsch reaction. The described catalysts would exhibit a high selectivity towards more desirable F-T products, such as paraffins, and would not show a tendency towards methanation. The performance of the synthesized

catalyst would allow the justification of the catalyst as industrially viable.

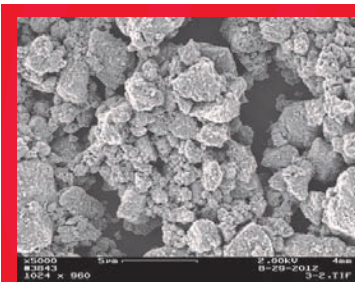
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Conclusion: Synthesized Cu doped Co_3O_4 nanoparticles at a 1:20 mol ratio of copper metal to cobalt metal were reduced to Cu doped cobalt carbide inside a tube furnace for application as a Fischer-Tropsch catalyst. The cobalt oxide nanoparticles were characterized by a variety of methods in order to confirm their identity and study their structure. Fischer-Tropsch catalysis using the doped cobalt carbide was done at 350°C at 200 psi while a gas mixture of 60% carbon monoxide and 30% hydrogen gas, helium balance, was passed through the reactor. In-line gas chromatograph (GC) analysis of the gaseous products of the reaction was done using a 2 column SRI GC. A small quantity of liquid condensed from the reaction products was collected from the microporous filter following the reaction and stored for future analysis by a mass spectrometer. The copper doped cobalt carbide catalyst exhibited extremely high CO conversion and minimal water byproduct formation early in the reaction. Selectivities for light paraffins were high after 20 minutes of catalysis. After 2 hours, general activity decreased dramatically as the catalyst shifted from the reduction of CO to the formation of CO_2 . The early product yield shows the promise of this cata-

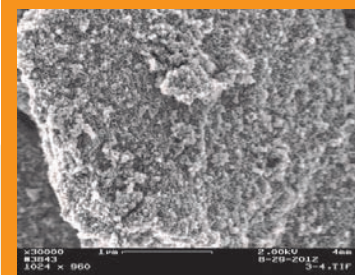
lyst as an industrially viable and inexpensive catalyst for Fischer-Tropsch synthesis. The tendency of this catalyst to oxidize carbon monoxide rather than form hydrocarbons also leads to a new possibility. This catalyst could potentially be used in a hybrid Fischer-Tropsch/greenhouse energy plant to maximize its usefulness. Synthetic leaves that can produce usable energy have already been produced and are still being optimized for efficiency. The combination of a Fischer-Tropsch plant and a synthetic leaf greenhouse as facilitated by the doped cobalt carbide catalyst could bridge the gap between modern day fuels and the energy sources of the future. Further optimization of the synthesized catalyst still must be done to maximize the potential of this catalyst in Fischer-Tropsch.

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Lower magnification image of a region of copper doped cobalt oxide nanoparticles

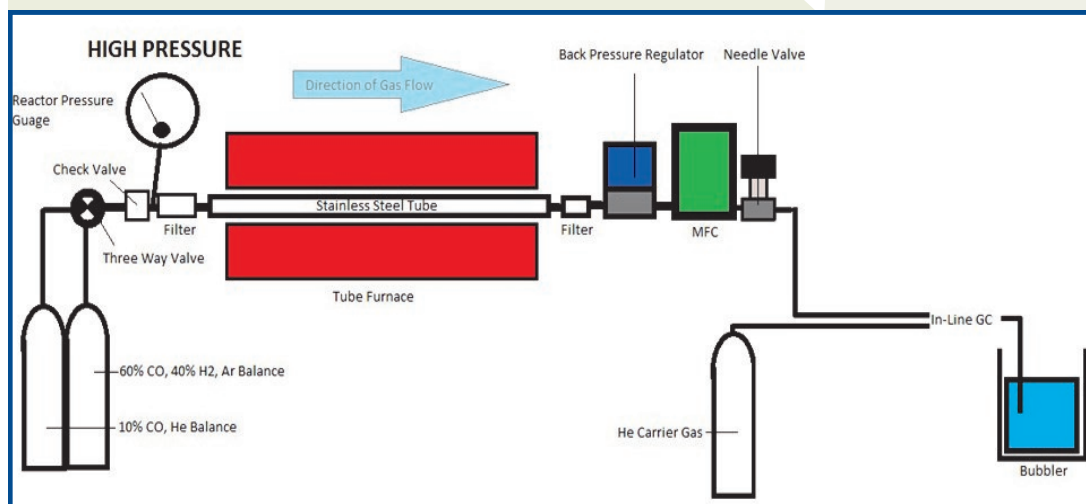


High magnification image focusing on the surface of a single Cu doped cobalt oxide particle

Reza Housaindokht, Sayyed Faramarz Tayyari, Jamshid Zarkesh, and Mohammad Reza Alaei. *Journal of Molecular Catalysis A: Chemical* 330.1-2 (2010): 112-20.

Tsakoumis, Nikolaos E., Magnus Rønning, Øyvind Borg, Erling Rytter, and Anders Holmen. *Catalysis Today* 154.3-4 (2010): 162-82.

Vo, Dai-Viet N., and Adesoji A. Adesina. *Applied Catalysis A: General* 399.1-2 (2011): 221-32.





Interaction of a tricyclic antidepressant with specific sites in the neurotransmitter transporter homolog LeuT, by Audrey Hecht (Junior)

Introduction:

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 smells, sights, sounds, textures and taste, as well as internal stimuli such as muscle tension, carbon dioxide levels, and blood pressure. Received by dendrites, the branched projections on one end of the neuronal cell, these stimuli 'travel' along specialized neuron extensions, called axons, as electrochemical signals, i.e., action potentials, throughout the central nervous system. Near the end of the axon, the neuron splits into several branches, called the axon terminals (Fig. 1). At the extreme end of each axon terminal is the presynaptic terminal. Together with the postsynaptic terminal located at the end of a dendrite of another neuron, these structures build the so-called synapse (Fig. 2). 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. 2 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, where it is recycled back into the communicating cell, thereby preventing the continued interaction of the neurotransmitter with the postsynaptic receptors. Different neurotransmitters, such as the monoamines (dopamine, serotonin, or norepinephrine) bind to specific receptors (Campbell & 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 sodium ion (Na^+) gradient across the presynaptic membrane as source of energy to transport their respective substrate into the cell (Campbell & Reece, 2005).

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 of neurotransmitters outside of the cell by mediating their re-uptake across the cell membrane of the presynaptic neuron (Fig. 2). The SLC6 transporters mediate the cotransport of neurotransmitters in a Na^+ and chloride ion (Cl^-) dependent mechanism (Chen, et al., 2004). In addition to their functional similarity, SLC6 proteins are anticipated to possess a similar structure as they share a high degree of similarity in their amino acid sequence. It was expected that these proteins consist of hydrophobic α -helices that span the membrane in zigzag fashion and are connected with hydrophilic loops. However, the properties of a protein also differ based on the composition of its amino acid residues, 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. On the other hand, 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 the high resolution crystal structure of the *Aquifex aeolicus* NSS homolog LeuT (Yamashita et al., 2005), the first, and to date, only

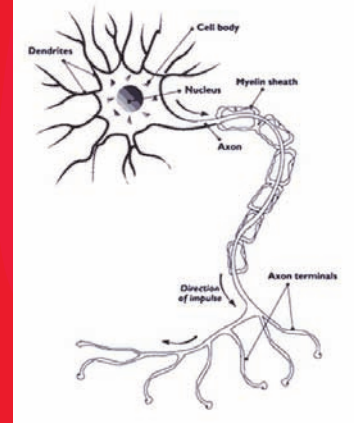


Figure 1: Schematic representation of a neuron

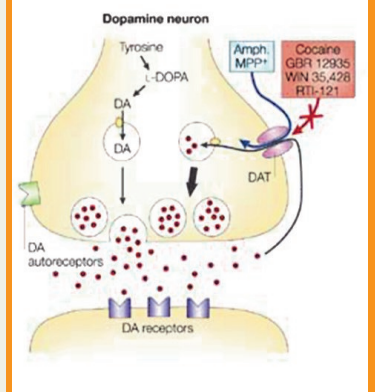


Figure 2: The Dopamine synapse. Dopamine (DA) is accumulated in vesicles in the presynaptic neurons and released in the synaptic cleft where it can bind to DA receptors located in the post-synaptic neurons. Clearance of DA from the synapse is achieved by re-uptake of DA in the presynaptic neuron by the Dopamine transporter (DAT). Psychoactive drugs, e.g. cocaine or amphetamines, inhibit the reuptake leading to increased levels of DA in the synapse.

NSS member for which crystallographic structure data are available. LeuT, a leucine transporter (Fig. 3), has become the primary template for studying SLC6 transporters due to its 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 facilitate genetic engineering 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 (Singh 2007). Using the high-resolution LeuT crystal structure as template for computational simulations in conjunction with biochemical and radiotracer flux and binding studies, comprehensive studies could be performed that to date provided unprecedented information about the mode of action of these medically important proteins (Shi et al., 2008).

Alternative modeling of the LeuT transport mechanism with a second binding site

Studies carried out by the Quick lab proposed an innovative transport mechanism involving the binding of the 2 Na⁺ (in the so-called Na1/Na2 sites) required for the transport of one molecule of leucine across the membrane (Shi, et. al, 2008). Previous studies done by the Quick lab

showed that the binding of Na⁺ is required for the conformational changes that occur within the protein allowing it to shift from the “outward-facing” conformation position (where binding sites are exposed to the synapse), to the “inward-facing” conformation (where binding sites are exposed to the cytoplasm and the substrates are released) creating the transport (Quick et. al, 2006). More recent studies using steered molecular dynamics (SMD) simulations together with a variety of substrate binding and transport studies (Shi et. al, 2008) suggest that in order for Leu to be transported, the substrate must bind to LeuT in the primary (S1) binding site (observed in crystallization, see Fig. 3) and second substrate (S2) binding site located about 11 Angstrom (Å) over the S1 site (Fig. 4). The S2 site overlaps with an extracellular vestibule that was shown to bind human drugs (see paragraph below). The S2 site is comprised of 2 parts: a hydrophobic pocket composed of Leu29, Tyr107, Ile111, Trp114, Ala319, Phe320, Phe324, and Leu400 which accommodates the Leu hydrophobic side chain, and a polar region composed of the Asp404 and Arg30 (Fig. 4). Allosteric interactions between the network of the two Na⁺ and two Leu sites, particularly the binding of Leu in the S2 site, are responsible for triggering the release of Na1 and S1 into the cytoplasm (Shi et. al, 2008; Quick et al., 2009).

LeuT Binding TCAs

Until very recently, studies done with drugs used in the treatment of psychiatric disorders concluded that these psychoactive compounds compete with substrate for binding in the S1 site (Chen et al., 2002). However, recent crystallographic evidence reveals that some of these drugs, such as TCA's (tricyclic antidepressants) bind to an extracellular vestibule (Singh et al., 2007; Zhou et al., 2007). Since this extracellular vestibule and the S2 site in NSS overlap (see Fig. 4), it has been postulated that

the inhibitory action of TCA's is based on the disruption of the transport mechanism by blocking access of substrate to the S2 site (Shi et. al, 2008). The binding of a TCA (such as clomipramine, CMI), while it occupies the second binding site, produces the same effect as mutated or dysfunctional secondary binding site (Fig. 4, Fig. 5) with respect to the transport of Leu, therefore consistent with the behavior of transport inhibitors (Shi et. al., 2008).

Statement of Purpose

Previous studies have shown that the interaction of CMI with LeuT impairs substrate transport by this NSS member. Due to the fact that CMI was found in a location that physically overlaps with the S2 substrate binding site it was postulated that binding of CMI binding prevents substrate binding to the S2 site, thereby ablating substrate transport. Using direct radiolabeled Leu binding studies it is hypothesized that CMI inhibits transport by directly competing with substrate binding to the S2 site but not with the S1 site and that higher Leu concentrations can (partially) overcome the inhibitory effect of CMI. To dissect these molecular events, binding of Leu was measured in the presence or absence of CMI to Leu-WT and two different mutants, each with a mutation of an amino acid that interacts with Leu in the S1 or S2 site (LeuT-F253A and L400S, respectively, Fig. 5).

(...)

Discussion and Conclusion

From these data collected, it is plausible to assume CMI competes with Leu binding only in the S2 site. The data shows that Leu binds 50% less effectively to WT in the presence of CMI because when S2 is occupied with the inhibitor, Leu can only bind to the protein in the S1 site. Experiments with mutants F253A and L400S in the absence of CMI where the S1 or S2 binding sites were disabled showed 50% less binding than WT. When Leu and CMI were simultaneously added to WT, a 50% decrease in bind-

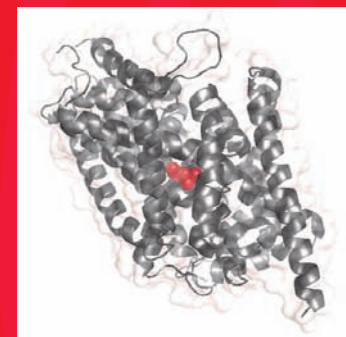


Figure 3: Three dimensional structure of the NSS Homologue LeuT. The 3D structure of LeuT of *A. Aeolicus* is shown with helices 1-12 in cartoon representation and transparent surface display; the centrally bound substrate, Leucine, is shown in ball representation highlighted in red.

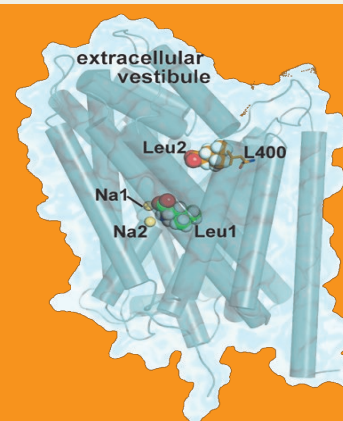


Figure 4. Three dimensional structure of LeuT illustrating Leu binding in the primary and secondary binding, along with the interacting amino acid residue L400

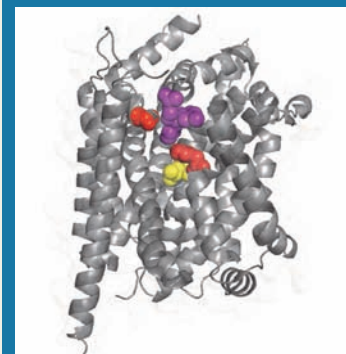


Figure 5. Three dimensional structure of LeuT bound with Leu (yellow) in the S1 binding site, and CMI (purple) binding in the extracellular vestibule overlapping with the S2 binding site. Amino acids F253 (lower right 'holding' Leu) and L400 (upper left near CMI) are interacting amino acids represented in red.

MENTOR Dr. Matthias Quick, Ph.D.



Assistant Professor of Clinical Neurobiology, Department of Psychiatry and Center for Molecular Recognition, Columbia University and College of Physicians and Surgeons.

Dr. Quick has been a dedicated supporter of the Science Research Program and has mentored Audrey Hecht in his laboratory, spending countless hours working with her on her project. Dr. Quick will continue working with Audrey through her senior year.

ing was observed making it a valid assumption to suggest CMI impairs the binding of Leu the same way disabling the S2 does in non-mutated LeuT protein.

Based on the results from adding CMI simultaneously with Leu, it may also be concluded that although CMI does impair the binding of Leu through direct competition in the S2 site, when tested at concentrations of 100 nM and 1,000 nM Leu can still partially bind to LeuT, although there is a significant 50% decrease in binding. Further research could be conducted to test this same variable at even higher concentrations of Leu and/or CMI to observe if Leu has the affinity to displace CMI in the second binding site, which would result in a less dramatic decrease in Leu binding in the presence of CMI.

By understanding the specifics of the functional binding patterns of Leu, more can be revealed about the mechanism that would allow for the manipulation of protein interaction. Investigation and further discovery about the binding mechanism of NSS provides a glimpse of how psychoactive drugs such as TCA's and other drugs are influencing the human body at the molecular level. With a better understanding of the mechanism by which drugs acting on the NSS transport mechanism work; there is the potential for the knowledge to be expanded upon that would improve medical treatment for people with disorders such as depression or addiction, as well as lead to

treatments for diseases that mirror this kind of molecular mechanism.

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Fine Particulate Matter 2.5 and Diabetes, by Dylan Gambardella (Junior)

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, which 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 secre-

tion 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 Diabe-

MENTOR Dr. Lung-Chi Chen, Ph.D.



Professor, Department of Environmental Medicine, New York University Langone Medical Center. We are grateful to Dr. Chen and Joshua Vaughan, PhD Candidate, New York University School of Medicine, for their guidance.

tes 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, PM_{2.5}, 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 PM_{2.5} 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 PM_{2.5} 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 exposure 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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Nanoparticles: Multifunctional Platforms for Controlled Targeting of Tumors, by Jazz Munitz (Sophomore)



In recent years, the field of nanomedicine has attracted much interest by the biomedical community. (Jarzyna et al., 2009) Nanomedicine, or the "development and application of nanoparticles for biomedical purposes", integrates a broad range of fields, and typically applies particles ranging from 2-100 nm in size. Nanomaterials such as iron oxides, quantum dots, as well as gold nanocrystals are being recognized and utilized for their abilities in "diagnostics as well as therapeutics". In recent years, nanomedicine has undergone unprecedented growth in the scientific community, resulting

in many new advances in the fields of nanoparticle-facilitated molecular imaging, (Jarzyna et al., 2009) targeted drug delivery, potentially offering new treatment paradigms for many diseases facing our society.

Many discoveries in the field of biomedical engineering have facilitated the current success of nanomedicine. One discovery, the enhanced permeability and retention effect, or EPR effect, is a useful phenomenon when developing, which can be exploited developing nanoparticle tumor targeting. The EPR effect, first identified by Matsumura and

Maeda in 1986, describes a process in which nanoparticles accumulate at sites where large gaps exist between endothelial cells in tumor blood vessels. When tumors become a certain size, hypoxia induces angiogenesis, the growth of new blood vessels to vascularize the tumor and allow oxygenation and nutrition of the rapidly dividing cells. The tumor cells stimulate the growth of new blood vessels through vascular endothelial growth factor, or VEGF. VEGF is a protein produced by tumor cells, as well as normal cells to stimulate the process of both vasculogenesis as well as angiogenesis. Both processes stimulate the growth of new blood vessels. In

normal body cells, and body function, under non-pathological conditions, VEGF is used to grow new blood vessels when old ones have been broken down or damaged through regular exercise or injury. However, tumor cells utilize this process to gain more nutrients than the normal cell, in order to stimulate rapid growth, while other cells receive fewer nutrients. Through the over-use of VEGF, vascular diseases and disorders can develop, further adding to the damage tumor cells can cause on the body. Although drugs, like bevacizumab can contribute to the stopping of tumors growing blood vessels, (Fang, Nakamura, & Maeda, 2010) the EPR effect proves very useful in finding and treating tumors through the use of nanoparticles.

Matsumura and Maeda observed and recorded, "Most solid tumors therefore exhibit enhanced vascular permeability, which will ensure a sufficient supply of nutrients and oxygen to tumor tissues for rapid growth. The EPR effect considers this unique anatomical-pathophysiological nature of tumor blood vessels that facilitates transport of macromolecules into tumor tissues...this EPR effect-driven drug delivery does not occur in normal tissues." (Fang, Nakamura, & Maeda, 2010). In developing a drug that can target, and in future advancements in the field, treat the tumor tissue, it is crucial that cells targeted are only tumor cells, as in both fields of study, healthy cells are not useful to image, or kill. While the EPR effect is a non-specific phenomenon that causes nanoparticles to accumulate only in

tumors, more specific targeting may be obtained by adding ligands on the surface of the nanoparticle, which will recognize tumor cells. Alternatively, the tumor cells endocytosis activity can be used to internalize the nanoparticles fitted with apoptotic agents.

A successful nanoparticle was developed by the Nanomedicine team at the Mount Sinai Translational and Molecular Imaging Institute, the Clinical Institute in Denmark, the Angiogenesis Laboratory at the VU University Medical Center, A Cy5.5-PEG-DSPE lipid based particle was developed for enhanced imaging. A usable particle was developed utilizing the EPR effect. Additionally, previous research with nanoemulsions took into account nanoparticle size for the targeting of various tumors.. (Jarzyna et al., 2009)

In the beginning research of nanomedicine and its uses in tumor imaging and treatment, one great boundary had to do with the originally thought to be lack of control over particles once they were administered into the body. With surgery, and other externally controlled treatments for removing tumors, doctors can see and control the processes and functions happening in the body during the treatment. However, with treatments on the nanoscale, the processes taking place once the drug has been administered cannot be controlled by doctors. Therefore, during development of said drug, the particle must be designed in a specific and precise

way, assuring that the particle can find the tumor and perform its function without human assistance and control. In the development and synthesis of the previously discussed particle, researchers and developers instituted various timed out processes in order for the particle to function properly. (Cormode, Jarzyna, Mulder, & Fayad, 2010)

Developed by researchers at the Translational and Molecular Imaging Institute, SyMo-Chem in the Netherlands, Cardiovascular Research Center and Mount Sinai School of Medicine, The Laboratory of Nanomedicine and Biomaterials, Department of Anesthesiology, Brigham & Women's Hospital, Harvard Medical School, Department of Chemical Engineering at the Massachusetts Institute of Technology, in Cambridge, the Department of Vascular Medicine, in Amsterdam, a complex multifunctional and surface-switchable nanoemulsion platform is a first of its kind. The challenges previously discussed with nanoparticle development and utilization have been overcome due to many inventions and new implementations demonstrated in this particle. "To deal with the aforementioned limitations of ligand functionalized nanoparticles we have developed a methoxy-polyethylene glycol (PEG)-lipid that can be cleaved by matrix metalloproteinase-2 (MMP2)." [An enzyme released by tumor cells during the metastasis process] "Incorporation of this cleavable PEG-lipid into a multifunctional nanoemulsion platform renders

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We are very grateful for the dedication of the entire Nanomedicine group at the Translational and Molecular Imaging Institute to our program.

nanoparticles with a surface-switchable coating...shielded by the cleavable PEG-lipids...chosen MMP2 cleavable sites as the enzyme is highly expressed at...solid tumors and atherosclerotic plaques." PEG-lipid coating will then be removed after an accumulation or particles around site of tumor causes the coatings to be removed when exposed to MMP2. Thus, the next step in the release of the substance suspended within the soybean oil will follow suit. (Gianella et al., 2011).

Another obstacle in the synthesis of such a nanoparticle lies in the production of such. Due to the size of said particle, many are needed for a single administration of such a drug into a patient. In order to mass-manufacture the particle, developers of the surface-switching nanoemulsion developed a self-assembling construction, allowing for efficient production of particle. To do this, they utilized the amphiphilic properties of phospholipids, which form the corona of the particle. A phospholipid is a molecule that has amphiphilic properties, meaning that it is both hydrophobic and hydrophilic. The

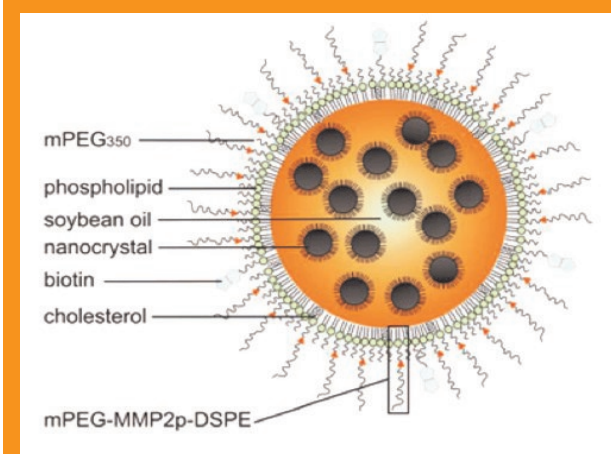


Figure 1: A Cy5.5-PEG-DSPE lipid based nanoparticle consisting of a soybean oil submerged bank crystal core, surrounded by an amphiphilic self assembling lipoprotein containment barrier, subsequently coated in a polyethylene glycol solution protecting the nanoparticle from the MPS (Macrophagocyte system), while increasing its longevity with in the body. (Gianella, 2011)

structure of the phospholipid contains a hydrophobic tail, and a hydrophilic head. The head is hydrophilic, since it is polar, which makes it attracted to the polar nature of water. The hydrophobic tail points itself toward the inside of the particle, because it contains oil, which is nonpolar. The reason the tail is hydrophobic and nonpolar is because of the carbon chain that forms the tail, and the occasional double bond resulting in the "kick" 3d shape. The hydrophilic head points itself outwards towards the water environment. The polarity is caused by the phosphate groups that give the head a charge. The polarities of the phospholipids lock themselves together forming a fatty barrier containing the soybean oil, and proteins suspended within. The size of the over-all particle can be altered by adding or releasing the volume of oil inside, and correspondingly adding or getting rid of the number of phospholipids present that forms the barrier, hereby modifying the surface to volume

ratio (Jarzyna et al., 2009)

A self-assembling, surface switchable and multifunctional nanoemulsion particle opens up many doors for the future of this research and opportunity in the field of utilizing nanoparticles for the use of tumor imaging, as well as therapy. This particle, the most recent in nanomedicine endeavors of its kind, can be replaced with different drugs or substances suspended in the soybean oil, to be delivered to the tumor tissues. Due to the flexibility of the particle, both structurally and from an opportunity standpoint, further advancements in therapy and imaging can be developed. Due to the dual functioning nature of this particle, as it can be utilized for both imaging and treatment, from the time of administration, through the treatment, and post-therapy, the particles movement and work can be observed through CT and MRI imaging. (Jarzyna et al., 2009)

Where this particle stands out from others in its class, is that it includes a "a lipophilic therapeutic...renders this platform theranostic." This means that in the case of the utilization of stem cells or other patient-specific, tailored treatment, such a personalized drug or therapy could be instituted in synthesis, and administered through the particle. Another breakthrough with this new platform has to do with its possible future and ability to treat diseases other than cancer. MMP2, the enzyme by which the particle cleaves is present in regions of other bodily disorders and diseases, including, but not limited to, "atherosclerosis, inflammation after stroke or myocardial infarction, as well as rheumatoid arthritis." If the enzyme and cleavable agent were to be switched to a pair suited for other diseases, the nanoemulsion could be altered in synthesis, or in size, (as it can currently be stable in sizes 30, 60, and 95

nm), in order to fulfill the specific hydro-environments of other diseases in other regions of the body. (Jarzyna et al., 2009)

The field of nanomedicine is ever evolving, and ever expanding. Although nanotechnology has been present recently, researchers are only now beginning to utilize the full potential of this exciting and promising field.

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The Promise of Gasification as an Alternative Energy Resource and the Potential for the Use of Gasification By-products, by Aidan Datené (Sophomore)

demands for fuels and electricity have coupled with this fear, and a heightened awareness of the influence of various pollutant species (including greenhouse gases) on the global climate system has created the incentive to develop alternative energy sources (Sallam, 2012). The consumption and depletion of Earth's natural resources (minerals, fossil fuels, and ecosystems) has contributed to global warming and loss of biodiversity. The 21st century coming of age science of industrial ecology is defined as the "re-thinking and re-designing [of] industrial activities with full knowledge of their environmental impacts (Themelis, 2007)," and is primed to find sustainable ways of handling the billions of tons of waste solids created by humans (Themelis, 2007).

Gasification is becoming a promi-

nent alternative to landfilling and basic methods of incineration (Butterman, Castaldi, 2008). Gasification breaks down the composition of biomass (biomass is carbon based with organic molecules), plastics, mixed waste materials, and municipal solid waste, and creates a desirable "syngas gas" or synthetic gas along with byproducts including ash and char (Leonard, 2012). Gasification is a thermochemical process that consists of two main stages, not including the refinement of products (Klein, Themelis, 2003). Pyrolysis, the first stage, occurs at temperatures below 600°C (1112 °F). Pyrolysis is the initial gasifying of the feedstock, allowing volatile components (parts of organic compounds) to be released (Klein, Themelis, 2003). This devolatilization process produces volatile gases, char, and ash

materials (Pantelides, 2006). In the second stage, the carbon in the ash and char are introduced to and treated with a co-reactant, oxygen, steam (water vapor), or carbon dioxide, resulting in the producer gas "syngas" (Klein, Themelis, 2003). The exothermic reaction in the gasification char and ash, between the carbon and the co-reactant, provides thermal energy that drives the pyrolysis phase. The reactions that create syngas are either endothermic or exothermic, and their rates depend on pressure, temperature, the co-reactant, and co-reactant concentration. The product gas can be used as fuels in steam boilers, reciprocating engines, and combined cycle turbines (Klein, Themelis, 2003).

Gasification products can be controlled; removing metal ions, tars, and oils during the thermochemical process and during the product's

In the United States, about 225 million tons of municipal solid waste (commonly referred to as garbage) are generated annually. One third of this waste is either recycled or composted. Landfilling or incineration manages about 150 million tons of municipal wastes. Landfilling faces increasing opposition because of its drastic effect on the environment (Sallam, 2012). Increasing

refinement, results in a reduction of carbon dioxide emissions (Themelis, 2007). Recovering energy from municipal solid waste in Waste to Energy Plants (such as gasification facilities) can reduce landfilling as well as air and water emissions, further lessening dependence on fossil fuels for power generation (Klein, Themelis, 2003). The produced syngas is primarily composed of hydrogen and carbon monoxide, desirable molecules in creating fuel. During oxidation of biomass the Boudouard reaction ($C + CO_2 \rightarrow 2CO$), the Water Gas Shift reaction ($CO + H_2O \rightarrow H_2 + CO_2$), and the reverse Water Gas reaction ($C + H_2O \rightarrow H_2 + CO$) take place, resulting in the gain of creating preferable molecules for energy production when refined (GRENWHICH). Refinement includes fuels, notably gasoline, and electricity among others (Butterman, Castaldi, 2008).

The gasification process is a self-sustainable process. Studies have concluded that gasification has beneficial capital gains and is an ecologically ample process. According to a recent study, the gasification process has been seen to cost less per kWh of electricity generated than that of combustion plants (Klein, Themelis, 2003). This attests to gasification's higher efficiency of converting thermal to electrical energy once feedstock components are converted into synthetic gas (Klein, Themelis, 2003). These are encouraging numbers that can cause a prevalent use of the process in place of other, biomass to energy plants.

Gasification of biomass is a growing because of its positive attributes. Biomass maintains a carbon neutral status, meaning it maintains a zero carbon footprint (Butterman, Castaldi, 2009). This makes biomass a prime candidate for gasification because there would be no carbon emissions in any form, when biomass is oxidized during pyrolysis (beginning phase of gasification). Predictions made from current studies suggest that if 20% of liquid fuels were produced from a carbon neutral source, such as biomass, there would be a resounding 15% reduction in CO_2

emissions (Butterman, Castaldi, 2009). Total global CO_2 emissions amount to nearly 29.5 billion metric tons annually. One third of these emissions come from burning gases in the transportation sector equivalent to 1.4 billion metric tons, which so can be averted through fuel replacement alone (Butterman, Castaldi, 2009). A resounding statistic, "for a typical automobile producing 6 tons of CO_2 /per year, this would be equivalent to removing 308 million vehicles from the road annually (Butterman, Castaldi, 2009)" provides great optimism for the future of gasification with biomass as the world looks to control its impact on the environment.

Biomass fuels are globally available (Butterman, Castaldi 2007). Biomass does not have the intermittency associated with wind and photovoltaics (wind and solar converting methods), and they constitute nearly one quarter of the fuel available from municipal waste. Biomass fuels currently provide 3% of the U.S. energy production. Nearly one third of the renewable energy sources in the United States can be attributed to woods, grasses, and agricultural forestry wastes, and residues (Butterman, Castaldi 2007). On the basis of a recent study, biomass has been found to be capable for supplying 5% of the nation's power by 2030. Biomass derived fuels have the potential to provide 20% of the U.S. transportation (Butterman, Castaldi 2007).

Currently there are limited uses for the byproducts of gasification, ash or char (Morf, 2012). These byproducts of the gasification process are generally discarded in a landfill (CEWEP). Recently char and ash have been used in construction applications or as an alternate cover for landfills (CEWEP), and because of energy shortages and environmental pollution (Zhou, 2011) has even created an inquiry for their capabilities as a cathode, in a microbial fuel cell (Livolt, 2013). However, the majority of char and ash are still discarded. Studies by Morf (2012), Gori (2009), Sallam

(2008), and Klinhoffer, Castaldi, (Livolt, 2013), and Nizhou (2012), focus on sundry uses for these byproducts and their beneficial properties.

Char is a residue that is produced during the pyrolysis phase in gasification (Klinghoffer, Castaldi, Nizhou, 2012). Char is composed of material from the original feedstock that was not successfully converted to syngas for fuel production. Char has been tested for catalytic properties and performance in an effort to replace commercial catalysts by efficiency serving as a cheaper alternative (Klinghoffer, Castaldi, Nizhou, 2012). This was tested under the hypothesis that char is dependent on gasification conditions. When char samples of biomass gasification are treated with different co-reactants, they release various amounts of carbon (which has been used in catalytic applications), and can chemically convert tars to useful products by breaking down the sample's composition (Klinghoffer, Castaldi, Nizhou, 2012). Results found were favorable; all parts of the hypothesis were confirmed, and optimistic results have char replacing commercial catalysts under certain conditions (Klinghoffer, Castaldi, Nizhou, 2012). Analyzing chars that were created under different temperatures with different co-reactants, (reactants such as water and carbon dioxide were selectively added to the pyrolysis phase), demonstrated that the surface area of char could be modified by changing gasification conditions. Results continued to show that char was able to break down hydrocarbons methane, propane, and to an extent, nitrogen (Klinghoffer, Castaldi, Nizhou, 2012). The char proved high efficiency when breaking down aliphatic bonds present in methane and propane. This performance was measured against two commercial catalysts and the char had superior results between 500 and 900 degrees Celsius (sample H_2O -750-60). When not under these temperature constraints, both H_2O and CO_2

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samples under 500 degrees did not provide adequate results, and above 900 degrees Celsius H_2O samples experienced sintering; the breaking down of the sample's microporous network while those that were produced under CO_2 did not provide satisfactory results (Klinghoffer, Castaldi, Nizhou, 2012).

Recent advances in recovering precious metals and rare earth elements from bottom ash have been cultivated from municipal solid waste (Morf, 2012). Efforts to recover these metals and elements have recently increased. This is not a relatively new concept. Law and Gordon (1979) found that there were considerable amounts of silver concentrations in bottom ash (38 ± 8 mg/kg). In 1996, Simon wrote a cover article for a Swiss paper stressing the economic potential for valuable metals that are present in bottom ash, "which showed 40-400 times higher levels of Ag [silver] compared to earth crust levels (Morf, 2012)" Although promising at the time, concentration levels were considered too low to make a recovery of the elements and the metals that made the product economically

feasible (Morf, 2012).

In recent years, research has continued to find economically sound methods of recovering metals and rare earth metal elements because of the correlation with an increase demand for these metals (Morf, 2012). Among investigated techniques, there is a magnetic separation method and an eddy current separation method used to generate iron metal fractions and non-ferrous fractions (metals that are light weight, have a higher conductivity than ferrous metals, and do not corrode) (Morf, 2012). The conclusions of the study proved that it is feasible to recover metals but in small concentrations (direct recovery). However, there were unexpectedly large annual amounts of heterogeneous solid waste in the substance flow, which hindered the ability to extract precious metals and rare earth elements (Morf, 2012). This is an area that needs further research into finding ways to facilitate effective methods of extracting precious subsidies while maintaining an economic equi-

librium. Potentially high market values for these metals and elements provide optimism. A large reduction on environmental impact by means of future waste and resource management systems may be achieved for economic revenue orchestrated by the high trading value of such metals as gold and silver, provided that efforts to develop recovery techniques are sustained (Morf, 2012).

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The Effects of Methylation on the 5HTR1A Receptor Gene Promoter Region versus the Concept of Increased Receptor Density, by Robert McIlrath (sophomore)

The electrochemical transmitting synapses of the brain, and subsequently their receptors, are extraordinarily important to the topic of psychopathology. Such synapses function by emitting electrochemical signals (in the forms of serotonin, dopamine and others) via the long axon of the presynaptic cell, where they pass through the gap known as the synaptic cleft, and into receptors at the dendrites, or receiving ends, of the postsynaptic cells (Carrad, 2011). Here the neurotransmitters bind to the receptors

of the dendrites, and if enough excitatory electrochemical signals are received to balance out inhibitory signals, an action potential passes through and carried onward through to the next neuron (Carrad, 2011). After this occurs there are often quantities of the neurotransmitters left amongst the synaptic cleft. To solve this issue the presynaptic cell detects, using a receptor, the amount of residual neurotransmitters, and activates the reuptake, of these neurotransmitters for later use (Carrad, 2011).

Serotonin, or 5HT, is an important neurotransmitter found in the central nervous system (CNS). Serotonin is currently thought to act on many different bodily functions, however, with respect to psychological actions, serotonin seems to contribute to feelings of well being, and security and controls mood, sleep, and appe-

tite (Carrad, 2011). The treatment of faulty transmitters with selective serotonin reuptake inhibitors (SSRI's), or other psychological medication is being more intently researched in past years as links between 5HT receptors and psychological diseases grow stronger, bringing 5HT near the level of importance of dopamine in the creation of antipsychotics. Diseases such as Schizophrenia, bipolar disorder, obsessive-compulsive disorder (OCD), major depressive disorder, and others are now being linked to 5HT receptors (Carrad, 2011).

Schizophrenia, classified using the DSM-IV manual, afflicts 15 persons per every 100,000, despite difficulties in its diagnosis. It categorizes itself with a spectrum of differing symptoms, at a variety of severities; symptoms include delusions, disorganization, and hallucinations as well as social/

emotional issues. The disease shows an affinity to males, and those who were born in/ raised in cities and urban environments. No single gene variation has been found responsible for the development of schizophrenia, though hereditary links exist. In the cases of monozygotic twins it is possible for only one of the pair to contract schizophrenia, leading researchers to believe the mechanisms responsible for the disease may be those of epigenetics (Tandon, 2008).

One such epigenetic mechanism is known as methylation, the action that occurs when CH₃ groups, or methyl groups, covalently attach to cytosine in CPG islands, or locations of high occurrences of cytosine and guanine pairings (Reynolds, 2006). When in a gene promoter, methylated cytosine can affect the accessibility of the RNA polymerase and henceforth hinder gene transcription, causing this change to be

passed down to successive cells. When the RNA polymerase attempts to read the genetic information of the receptors, due to the methylation and silencing of certain parts of the code, it is unable to do so, and the new receptors it creates with this information will all share the same malfunction (Reynolds, 2006).

A popular belief is that the 5HT_{1A} receptor promoter region is being methylated. The 5HT_{1A} receptor controls education motivation, emotional association with memory and receives input from raphe nuclei, a cluster of nuclei in the brainstem that regulates the firing rates of 5HT neurons, which release serotonin to the rest of the brain (Meltzer, 1999). Seeing as the 5HT_{1A} receptor has such a great effect on other serotonergic functions it is understandable why it would be so important, and its methylation and silencing is being linked with psychological disease. If this were true it would explain why monozygotic twins, twins who share the same zygote and therefore genetic information, could independently develop disorders such as schizophrenia. One twin would experience increased methylation, and henceforward silencing, of 5HT_{1A} receptors, whereas the other would not, causing each to appear outwardly to have differing genetic information, but internally not be as such. If the link could be further proven it would prove a massive breakthrough in the creation and development of schizophrenic antipsychotics as well as general antipsychotics, and

it would also open a great deal of information to the fields of psychopathology.

One opposing theory is that the 20-79 % increased density of 5HT receptors, as noted in post mortem studies of schizophrenic patients, is responsible for such symptoms. The highest density levels are noted in the limbic cortex region, secondary to this is the brainstem and raphe nuclei, then the frontal cortex/ neocortical regions, and finally the density variations are very marginally detected in the cerebellum. The thought premise is that the increased density of receptors in a certain area of the brain will cause differed reactions than if receptors were more equally spread out. Though post-mortem studies agree with such a hypothesis, in vivo studies have not been so effective. Greater evidence lies in the more to the methylation-based principals above, comparatively. If such experiments were tested again a link could be further strengthened and lend credence to one side of the debate, or uncover entirely new elements to the puzzle (Meltzer, 1999).

The serotonin receptors, primarily the 5HTR1A receptor, have been commonly seen in relation to psychiatric disorders (Schizophrenia, Bipolar Disorder, Major Depressive disorder, etc.) and this connection continues to be seen as experiments are repeated (Lesch, 1998, Carrad, 2011). This correlation could

have drastic implications on advancements for anti psychotic medication. According to past studies this is due to methylation of the promoter region of the 5HTR1A receptor (Reynolds, 2006). When the 5HTR1A serotonin receptor is methylated certain parts of its genetic code are suppressed. Such epigenetic reasoning for the cause of psychiatric diseases could explain how two monozygotic twins are able to each have different genealogical status'. Opposing such theories are ones about receptor densities, which fail to uphold during in vivo studies (Tauscher 2002). The further exploration of this field, and the causation of psychological symptoms on such a level is highly important and will prove vital to the development of antipsychotics and the improvement of the lives of those with such disorders.

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MENTOR Dr. Miklos Toth MD, PhD



Professor of Pharmacology, Weill Cornell Medical Center.
We are delighted to announce that Dr. Toth will welcome Robert McIlarth in his lab this summer.



The Bone Marrow Stem Cells and Cardiovascular Disease, by Stanley Fray (Junior)

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 (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 stage 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)

The proper culture of adult stem cells in defined environments may allow scientist them for therapies. Although adult tem cells have a (Prockop, 2004). Through stem cell development there are different levels of potency: 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 back to embryonic cell state (Bethesda, MD, 2008). iPSCs are very important for by using them the demand for embryos/fetuses (used in embryonic stem cells) will go down. With there being less controversy on the use of embryos/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 bone marrow cell. They are then extracted from bones and harvested to further differentiate into heart cells (Philippe, 2008). Correspondingly, mesenchymal stem cells (MSCs and bone marrow cells) are multipotent progenitor stem cells, which 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). Because of these properties, stem cells can play a major role in finding ways treat cardiovascular disease and myocardial infarction.

In myocardial infarction (or heart attack), the blood flow to a heart is blocked for a 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. 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 stop the buildup of platelets which play a major part when it comes to clotting; those medications are helpful when it comes towards preventing future heart attacks ("Myocardial infarction heart," 2011). 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). Another treatment for coronary artery disease is a angioplasty. 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 clogged artery, inflates then deflates causing the artery to increase in size. A stent is placed in restoring blood flow to the artery at the location ("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 an injured heart. In a 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 it will cause the hearts damaged muscle to heal (Ott, Davis & Taylor, 2005). Adult MSCs were skeletal myoblast (SKMB) and bone marrow mononuclear cells (BM-MNC) were used to determine the most effective to a damaged left ventricle in rat heart (Ott, Davis & Taylor, 2005). The experiment was done in vivo using live rats in the trial; the cells were injected into the infarcted part of the heart. The SKMBs were superior to the BM-MNCs, as the left ventricle function in the rat's heart had increased (Ott, Davis & Taylor, 2005). Although one cell type was superior to the other the combination of both cells types being used have shown a synergetic in the damaged heart (Ott, Davis & Taylor, 2005). Timing and cell types used can be a critical factor in optimizing these procedures (Ott, Davis & Taylor, 2005). The survival rate of transplanted cells have to be taken into consideration, since 70%- 90% of transplanted cells die in the first few

days after being injected into the infarcted scar; there maybe immune rejection, the immune system attacks the injected cells (Ott, Davis & Taylor, 2005). Although there was success in the clinical trial, there are still many factors need to be interjected.

Many stem cell types can be used in clinical trials both in vivo and in vitro, from adult stem cells to embryonic stem cell to MSCs and Skeletal muscle cells. Myocardial infarction is already effectively treated by clinical medications and procedures, but use of stem cells could result in a faster recovery by repairing the damaged tissue.

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Concussion research, by Justin Fischer (Junior)

Baugh, Nowinski, McKee, Stern & Cantu, 2011).

If you can find out a way to prevent a concussion you would never have to deal with them. Large research for football, especially Ivy League football teams, has been attempted. The Ivy League 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, they had two full contact practices a week rather than 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 could have caused a concussion, year round. So the league concluded that not only will this rule, lessen concussion rates, but it will also keep the players fresh during the week and before their games (Belson, 2011).

The diagnosis of concussions is also vital for advancement in football concussion research. The diagnosis of a concussion relies on many symptoms, but a big symptom that correlates with concussions is sleep. The Sleep symptom that people see is sleep disturbance. But those symptoms are very general, and can just be coincidence. A probable better way to find a concussion would probably be through measuring sleep rhythms in the Supra Chiasmatic Nucleus (SCN). The Supra Chiasmatic Nucleus is basically the control center for sleep rhythms, or in other words the internal

human clock. It maintains all sleep rhythms to make sure that the person gets enough sleep at night, and maintains the cycle of sleep. The Supra Chiasmatic Nucleus helps maintain the sleep rhythms by helping change how the brain shifts between sleep waves, and stages. It also helps maintain cycles of sleep by being able to keep a person sleep wake cycle at its appropriate full-length schedule. So basically when a person goes to sleep, wakes up, and then is about to go back to sleep, that is one full cycle. Disturbance in the SCN would then mean that a person's full-length cycle is not as long, or short as it should be. An example of a possible sleep wake disturbance would be a person sleeps for 8 hours, and is awake for 16 hours on most days. But then the person, after receiving brain trauma, sleeps for 4 hours, and is awake for 20 hours on most days. This is a major difference in that persons sleep-wake patterns, which could prove a person concussed (Breedlove, Watson & Rosenzweig, 2010).

When you first fall asleep you are put into stage 1 and 2 Short Wave Sleep (SWS). These sleep stages display Alpha waves; these are fast, short waves. Usually in these sleep stages you don't even know you're asleep. Next are the stage 3 and 4 SWS. These sleep stages display Delta waves; these are slow, long waves. These waves are used for the brain to rest because the brain is less active. The last sleep stage is REM sleep. This is probably the most important sleep stage, because this stage is where the brain is maintaining the bodies heat and metabolism, making sure that they don't teeter off. If both of these did teeter off then you could receive diseases due to a poor immune system (Breedlove, Watson &

Rosenzweig, 2010).

There is much more to sleep waves however. Sleep waves are actually brain potentials, which occur when you're asleep, that occur by a certain amount per second. Changes in sleep waves can be a symptom of injury, and can be measured with EEG, which measures brain functions. EEG measures brain potentials, and then scores them based on how fast, or how slow the potentials happen. If the stage 1 and 2 sleep waves are slower than usual, and stage 3 and 4 sleep waves are faster than usual, then there is definitely something wrong. Longer REM sleep can also be one of these change causes. These all are causes because changes in these stages show that there has been damage to the SCN, which is a part of the brain. So damage to the SCN means that there has been damage to the brain (Breedlove, Watson & Rosenzweig, 2010).

Concussions ultimately can lead to diseases that are catastrophic to their victims. Concussions 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).

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 is mostly based on the physician's judgment; (Schatz, Pardini, Lovell, Collins & Podell, 2005) 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 (Cunha, 2012).

Concussions happen most of the time in sports, and predominantly contact sports. The sports that concussions happen most in are football, lacrosse, hockey, soccer, basketball, cheerleading, and sometimes baseball or softball. The way concussions happen is by reoccurring hits to the head, or one giant blow to the head. But figuring out if a person has a concussion is usually a judgment call. If you are unconscious it is obvious that the person has a concussion, but most concussions happen while you are still conscious. So the concussions are hard to diagnose. But people do know that the more hits to the head, the more likely a person is to receive an injury to the head. So if a person has had more reoccurring hits to the head, than that person might be monitored. But nevertheless concussions happen in athletes daily, and concussions are one of the largest concerns for injury research today (Daneshvar,

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Deja Vu, by Matthew Kenny (Sophomore)

One of the most common and intriguing phenomena known as déjà vu. This spontaneous feeling of having done, seen, heard, or felt something before has an unknown cause, despite its common occurrences. A major contributing factor to this is the fact that déjà vu episodes will occur spontaneously, meaning that researchers do not have the ability to collect data on the brain activity that goes on during a genuine déjà vu episode. Therefore, in order to understand the actions of the brain during an episode, the brain needs to be manipulated so that a déjà vu episode can be simulated.

There are two basic ways that can be used in order to simulate déjà vu. The first is electrical brain stimulation through the use of pathological methods. This method is quite consistent in its producing of déjà vu in the participant. Using these electrical stimulations, the activity that goes on during déjà vu is simulated (Adachi 2010). While the data is quite generic and will not show any originality through different participants, electrical stimulation is quite consistent in its ability to work. However, there are many limits to the type of people that can be experimented on with this method. Only those with certain diseases

of the brain, more specifically Temporal Lobe Epilepsy and Schizophrenia, are usually used as participants due to the fact that the stimulation is more consistent in creating a déjà vu episode when participants have these brain abnormalities (Radvansky 1998). The reality for these participants is that they will face harsh consequences for taking part in these experiments. This is one of the main reasons as to why it is so necessary for another way in which déjà vu can be provoked to be found. For example, those with Temporal Lobe Epilepsy are very likely to experience a seizure after the déjà vu episode which was caused by the electrical stimulation. This is the main reason as to why many scientists are searching for alternatives to electrical stimulation that still allows them to simulate déjà vu (Brown 2008).

A second way in order to create a déjà vu episode in any participant is through the process of creating false memory in the brain. This process is quite complicated and the steps for creating this false memory need to be followed very strictly in order to be successful in creating a déjà vu episode. This process involves showing one scene to a participant, and then showing them another scene that is only slightly altered. Because the second scene seems relatively similar to the original scene, the participant will feel as if they have seen this scene before, and will therefore experience déjà vu (Brown 2008). This method leads to no known consequences for any participants, because in this case, the déjà vu episode that the patient experiences is real. While electrical stimulation shows the

activity that goes on in the brain during déjà vu, the participant does not truly experience déjà vu. In the method involving evoking false belief, the participant is simply being set up to experience déjà vu. While the scene the participant is seeing is fake, the activity in their brain is not (Moulin 2010). Still the brain activity will be recorded and can be used, but the data will always be relatively the same, without some abnormalities that could easily be seen in true cases of déjà vu.

The only problem with the method involving false belief is that it is nowhere near as consistent in creating a déjà vu episode as electrical stimulation is. Therefore, many scientists studying of déjà vu will still decide to use pathology and electrical stimulation in order to create this déjà vu activity (O'Connor 2012). In order to force scientists to turn to the use of stage simulation rather than electrical stimulation, false belief needs to be perfected so that it has a higher consistency rate.

The opportunities that are potential when there is an ability to gather data on déjà vu are immense. For example, déjà vu may have a relation to seizures (Serendip 2009). This theory is supported by the fact that most people with diseases of the brain that are related to seizures will usually experience déjà vu before a seizure occurs. Déjà vu is seen, by some, as being a "mini-seizure" rather than being its own phenomenon. Therefore, being able to stop déjà vu from happening could be very useful in learning how to stop seizures from happening in those with certain diseases, such as epilepsy and

schizophrenia.

Many feel upset after going through the experience of déjà vu. For that reason, it would be important to stop it at certain times, as some may even go as far as suffering from depression in rare cases, in which déjà vu occurs very frequently. Understanding the neurological process behind déjà vu would allow us to monitor and record data on it. If déjà 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 déjà vu (Adachi 1999). There may be a possibility that déjà vu is a cause of a seizure or generates it. Therefore, by knowing how déjà vu arises and stops, there may be a way to stop or diminish temporal lobe epilepsy consequences. Also, another theory may be that the feeling of déjà vu is simply the beginning of a seizure. Therefore, this theory would then go on to state that déjà vu is simply a smaller version of a seizure. This would coincide with the fact that not everyone experiences déjà vu, meaning that not all people experience seizures.

The most believed and trusted theory as to how déjà 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 déjà vu phenomenon is the "cellphone" 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 conscious 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). 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*.

It is necessary for scientists to find another way to provoke *deja vu*

because of all the potential findings that could come from that data. *Deja vu* has so many potential connections to many things, including Schizophrenia, Temporal Lobe Epilepsy, and much more. Without another way to provoke *deja vu*, scientists will struggle with many other fields.

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Anxiety levels can be classified using psychological, physiological, or biological responses or lack thereof. When a person is experiencing anxiety, psychological responses include feelings of worry, nervousness, or unease, typically about an impending event or something with an uncertain outcome (Roth, R., 1994). Key examples of physiological response to anxiety include a lack of focus, an inability to unconsciously remain still, rapid breathing, trembling hands, cold hands and feet, a rapid heartbeat, a dry mouth and overall restlessness (Orme-Johnson, D.W., 2001).

During periods of anxiety, the mus-

Meditation and Anxiety in the School Setting, by Jordan Hudock (Sophomore).

cle demands higher levels of glucose and oxygen running through the bloodstream. For this to be possible the heart has to pump faster and blood pressure must increase. The entire process starts when sensory cells alert the hypothalamus of a potential danger. Cells in the hypothalamus transmit a signal to the pituitary gland, which releases the adrenocorticotrophic hormone (ACTH) to the adrenal glands (D.W., 2001). ACTH signals to both adrenal glands to produce more cortisol. ACTH molecules bind to receptors on the surface of adrenal cells, which initiates a complex process that turns cholesterol into cortisol. When produced, this hormone is pumped into the body and attaches to its receptors. At this point, cortisol can interact with other cells to increase the sensitivity of the heart and blood vessels. This leads to the effects of epinephrine and norepinephrine. These hormones stimulate receptors in heart cells, which increase the rate of the heartbeat and the force with which the heart muscles contract (Shapiro, 2008). Cortisol also ensures that the

blood vessels are receptive to epinephrine and norepinephrine. These hormones attach to particular proteins, which cause the narrowing of blood vessel walls. This brings about an increase in blood pressure and allows blood to be sent to essential organs and muscles.

Meditation is commonly associated with reducing the symptoms of anxiety. It is a form of contemplation, reflection, or heightened awareness (Travis, F., 2001). Alterations in brain function during meditation have been recorded using single photon emission computed tomography, electrophysiology, PET and functional magnetic resonance imaging. Although results differ slightly between forms of meditation, overall results indicate intensified signals in brain regions related to attention control and affect regulation, with an increase in dopamine release.

The biological effects of meditation contribute to why it has been shown to reduce symptoms of anxiety. One study entitled, "Effects of Transcendental Meditation practice on brain function-

ing and anxiety reactivity in college students" was executed to determine the effects transcendental meditation practice had on brain function and anxiety levels in college students (Oman, D., 2008). This trial investigated the effects of Transcendental Meditation (TM) on broadband frontal coherence, preparatory brain responses, and power ratios, all of which were measured using the Brain Integration Scale. Fifty college students participated in a pretest, then assigned to either learn TM immediately or after a ten-week posttest. Analyses revealed significant increases in Scale scores for students who began learning TM immediately, but decreases in students whose learning was delayed. There were significant reductions in sleepiness among immediate-start students with no change in delayed-start students. The trial showed no changes in habituation rates in immediate-start students, but significant increases in Delayed-start students. These data support the value of TM practice for college students.

Anxiety is extremely prevalent in school systems and meditation is a potential tool that could be used to

alleviate symptoms. Schools could take a few minutes each day to integrate meditation into classes in productive ways. This would likely lower the anxiety levels of students and improve their abilities to concentrate and think clearly throughout the day.



In many cases, disorders have many contributing factors. For instance, in Alzheimer's disease, there may be several causes, such as epigenetics, amyloid beta proteins, and neurofibrillary tangles. Though many factors may contribute to the pathogenesis of this disease, we do not know for certain what the actual causes are.

Epigenetics is defined as "heritable changes in gene expression that occur without a change in DNA sequence" said by Dr. Alan Wolffe (Nakao 2001). Epigenetics uses DNA methylation to regulate gene function (Nakao 2001), allowing the genes to make certain proteins that are needed. Epigenetics down regulate (turns off) or up regulate (turns on) the genes (Nakao 2001), and certain environmental factors (such as Pb and oxidative stress) can use epigenetic mechanisms to turn on and off these genes. The body then makes an excess of certain proteins, and stops making other proteins, potentially causing disaster.

Alzheimer's disease (AD) is a progressive neurodegenerative disease that comes in two forms: late onset Alzheimer's disease, LOAD, and early onset Alzheimer's disease, EOAD. The most common hypothesis is that Alzheimer's disease is caused by excessive amy-

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A Possible Effect of Exposure to Lead on the Onset of Alzheimer's Disease, Clio Lieberman (Freshman)

loid beta plaques (AB) in the brain. Amyloid plaques are made up of a tangle of aggregated fibers (amyloid fibers), that are insoluble and difficult to break down or remove, and are also highly toxic (Huang et al., 2011) because they cause the cell to break down. Adding on to this hypothesis, lead (Pb) seems to use an epigenetic mechanism to alter genes and cause an overproduction of AB, which then causes AD. This would explain why Pb is so toxic, and how it might indirectly be involved with the pathogenesis of AD.

This hypothesis is very intriguing because Pb is one of the most dangerous environmental toxin humans and animals are exposed to. Recently, there has been a reduction in Pb because people have become more aware of its effects and laws have been passed. However, that may explain why people in their old age are more susceptible to AD, because they were exposed to it when they were developing and when no one was aware of the effects of Pb.

There may be a clear link between Pb exposure (and how it effects DNA) and the pathogenesis of AD (Huang et al., 2011). In some studies, it was shown that Pb might lead to poor cognitive performance (Dosunmu et al., 2012) and disabilities (Huang et al., 2011). This is interesting because poor cognitive performance may be correlated to AD later on in life (Dosunmu et al., 2012). Pb also "impairs intellectual functions and disturbances in a number of molecular, biochemical, and morphological events" (Basha et al., 2005). If Pb

leads to poor cognitive performance, and poor cognitive performance is correlated to AD, it may be safe to say that Pb indirectly leads to the pathogenesis of AD, or that cognitive impairment is the first sign of AD before it is diagnosed, since AD can only be diagnosed for certain after death.

High levels of Pb are also found in neurofibrillary tangles in the brain, which seems to be related to AD, which was seen in 52% of cases studied (Behl et al., 2009), and AD neurofibrillary tangles were found in the hippocampus of an adult who was exposed to Pb (Huang et al., 2011).

Taken together, Pb and its effects on epigenetics can come together to cause AD through AB proteins. Interestingly, developmental exposure to Pb interferes with life-long DNA methylation, behavioral and neurodegenerative outcomes, which, in turn, affects the brain's ability to respond in later years. (Dosunmu et al., 2012) (Basha et al., 2005). For example, if Pb is causing the APP gene to up-regulate, then more and more amyloid beta proteins will be made, and it will be harder to break them down and remove them, since they are insoluble. Developmental Pb exposure determined the expression and regulation of genes that effect amyloidogenesis (the process of making amyloid beta proteins) later in life, "80% of genes altered late in life due to infantile Pb exposure were down-regulated, while 20% were up-regulated" (Huang et al., 2011). This is very important because Pb seems to be using an epigenetic mechanism, controlling the genes involving AB. Developmen-

tal exposure to Pb resulted in an increase in APP (amyloid precursor protein) in mRNA (Basha et al., 2005), resulting in an increase in AB proteins. When developmentally exposed to Pb, there was an increase in APP mRNA and APP and AB levels (Basha et al., 2005), and in the study done by Behl et al, it was confirmed that developmental exposure to Pb increased AB accumulation in cells. The cerebral cortex contained increasing levels of ABPP mRNA and AB proteins that were abnormally elevated when developmentally exposed to Pb (Huang et al., 2011). Pb increased amyloid plaques by 64.3% compared to controls (Huang et al., 2011), demonstrating that Pb plays a key role in the excessive making of AB proteins. "Plaque formation that follows is brought about by the transformation of AB from a soluble form to neurotoxic aggregated, fibrillary structure" (Huang et al., 2011), and when AB molecules were exposed to Pb, there was an increase in AB fibril formation (Huang et al., 2011). This not only means that there is an increase in the AB proteins, but they are becoming insoluble and accumulating in the cells, leading to AD pathogenesis.

Another key element in the pathogenesis of AD is neprilysin. According to Dosunmu et al, neprilysin, or NEP, is "a protease degradation of AB, affected by Pb which decreases its mRNA expression and protein", but a more detailed report is given by Huang et al, saying that neprilysin is a "type II membrane-associated protein which is predominantly localized at the presynaptic terminal and is involved in degrading the monomeric and oligomeric forms of AB peptide. . . NEP is a plasma membrane glycoprotein of the neu-

tral Zn metalloendopeptidase and is also known as physiologically important AB-degrading enzyme in mammalian central nervous system". NEP is a protease that breaks down amyloid beta proteins, and regulates their levels. Interestingly, NEP is also affected by Pb exposure because NEP mRNA is down-regulated by Pb, with a decrease of NEP expression by 50% (Huang et al., 2011). The down regulation of a protease that breaks down AB makes it harder to break down AB, leading to an AB accumulation (Huang et al., 2011).

These studies make a very convincing argument supporting the

hypothesis that Pb may be involved in the pathogenesis of AD. Pb effectively causes cognitive decline, which is the first step in AD pathogenesis. Pb also causes excessive AB protein production and neurofibrillary tangle buildup, which is responsible for AD (or at least, may be the main culprit). A down-regulated the NEP gene makes it harder to remove the AB protein buildup in the brain. Pb may be modifying key genes that are responsible for the over expression of ABPP, which causes the increase of AB protein production. The down regulation of the NEP gene especially, since it

is involved in the degradation of AB proteins, leading to an accumulation of neurofibrillary tangles in cells. Further research in the field of epigenetics and genes especially involved in AD could potentially lead to a more complete understanding of the pathology of AD.

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How to improve the Identification of the Location of the Epileptogenic Zone Before Surgical Procedures in Epilepsy Patients, by Jonathan Young (Sophomore).

severity of the case and the type of epilepsy experienced it may prove to be a life threatening condition. A seizure can result in death via multiple routes, be it cardiopulmonary arrest, or a vegetative state, therefore it must be dealt with as soon as it is discovered.

Epilepsy can be treated using anticonvulsants and muscle relaxants. However, nearly half of all reported cases of epilepsy are intractable, or untreatable by anticonvulsants.(Berti, 2010). In cases such as this there is only one other viable option, this is to undergo retraction surgery to correct the issue by completely removing the section of the brain that is damaged or malfunctioning. This is no large issue to the brain because of the properties of neuroplasticity it can fill in the gap. Neuroplasticity is the brain adapting to some kind of damage inflicted to it.

To perform neurosurgery it is required to have as much detail as possible. this information is acquired from a complex presurgical evaluation. There is detailed neuroimaging involved, along with video/EEG monitoring for several weeks, and an in depth

history and physical exam (Berti, 2010).

Doctors and researchers are constantly trying to find an alternative to this method. The methods now in practice are very effective at picking up damage, however some of the testing that is done may be fairly invasive.

Is there a better method of detection being researched? The answer would be a resounding yes. Almost all researchers are going in different directions when it comes to epilepsy. All of the researchers are looking into the different types of epilepsy.

At its simplest the idea of finding the cause of the epilepsy rests on one basic goal, to find the epileptogenic zone, or the area of the brain in which the seizures originate. Once found it is possible to know exactly where the seizure's point of origin is. That is all the information you would need to perform the surgery, the exact coordinates of the epileptogenic zone. Find the epileptogenic zone or region and can cure the person of their epilepsy.

There many ways to find the epileptogenic zone; There are MRI, CAT scans, CT scans, SPECT,

SISCOM, etc. Unfortunately, the only way to get an accurate reading is to stimulate, or precipitate a seizure. After the seizure has begun, and has been recorded as beginning, a radiotracer or contrast dye must be injected within a few seconds. Then the patient is put into the imaging device and scanned, and all of that has to happen within the duration of the seizure.

One of the newer ideas on the subject of locating the epileptogenic zone is that, by finding hyperperfusion in areas of the brain that seizures have been localized to you can find the epileptogenic zone. To find out if there is hyperperfusion the researchers or doctors take a baseline image of the brain with a SPECT imager, then they repeat the procedure during a seizure. To get a more accurate result they will sometimes perform a SISCOM, or subtraction ictal SPECT co-registered to magnetic resonance imaging. Using this method they can track the blood flow with the SPECT, and the image is layered over an MRI with detailed images of the structures of the brain. They will then have specific reference images and a base of information.

There are obviously other methods being studied, but some of them are

Epilepsy is a disorder of the brain which affects approximately fifty million people worldwide (Mandal, 2012). Epilepsy is among the most common neurological disorders. However the question remains, what is epilepsy? Epilepsy is defined as a disorder of the nervous system, characterized either by mild, episodic loss of attention or sleepiness, or by severe convulsions with loss of consciousness (dictionary.com, 2012).

Whichever one characterizes the sufferer of the disorder it is obviously a hinderance. Also epilepsy is variable in how it presents itself. There are many different ways that epilepsy can present itself, such as: drooling, swallowing, vomiting, coughing, flatulence, urination, uncoordinated tonic clonic posturing, aggression, hallucinations, and the list goes on (Berti, 2010). However based on the

more invasive such as surgically implanted electrodes that would stimulate a region of the brain with electricity and read the response signal then feed the signal through a computer program to discern the point of origin. (MacDougall, 2009)

The point being that there are many different methods of detection being researched. Science is

bringing people to a place where epilepsy is no longer a problem. Detection methods are essential to curing epilepsy.

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- Dr. Alice Gottlieb, Assistant Superintendent for Curriculum.
- Enrique Catalan, Assistant Superintendent for Business.
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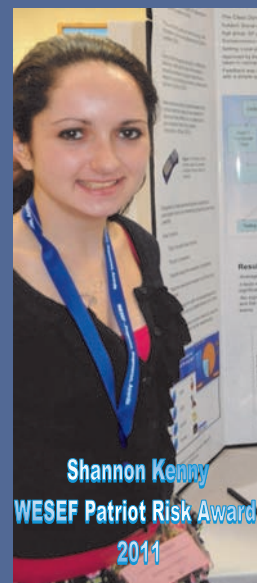
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Audrey Hecht,
WR-JSHS 1st Place Poster
WESEF 4th Place,
Tri-Country Science Fair, 2nd Place,
2013



Cassandra Rogers
NYSSEF 2nd Place
WESEF 4th Place
2012



Shannon Kenny
WESEF Patriot Risk Award
2011



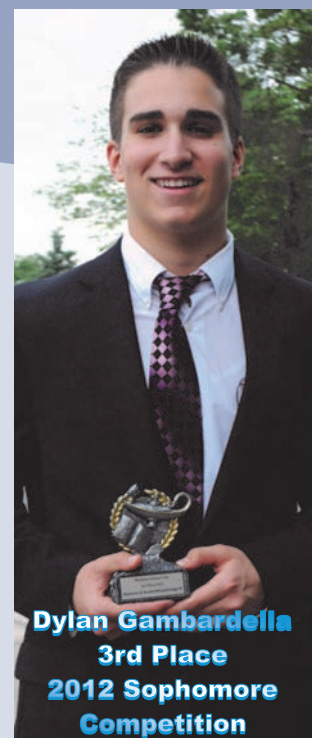
Alex Alves
WR-JSHS 2nd Place Poster
WESEF Visual Impact Award
2012



Shivane Shah
WESEF Fischer Award
2012



Alexis Mangual
WR-JSHS 5th Place Speaker
WESEF Momentum Award
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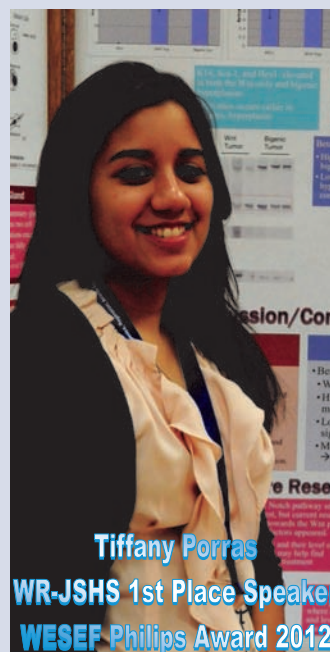
Dylan Gambardella
3rd Place
2012 Sophomore
Competition



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WR-JSHS 4th Place Speaker
WESEF 3rd Place
NYSSEF 3rd Place



Naimah Hakim
WR-JSHS 1st Place Speaker
WESEF 3rd Place
2011



Tiffany Porras
WR-JSHS 1st Place Speaker
WESEF Phillips Award 2012



David Eisenberg
Full Paid Internship
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