the Berkeley Carroll School

Proceedings of the Berkeley Carroll

1ST INDEPENDENT RESEARCH CONFERENCE

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Welcome from the Editor

Welcome to the inaugural issue of the *Proceedings of the Berkeley Carroll Science Research Conference,* a scientific journal representing several years of work by Berkeley Carroll high school students in the Science Research & Design Program.

The first issue of any publication is very special as it provides an opportunity to establish a high standard of depth and breadth of scientific understanding; to that end, these students have done a wonderful job. Further, this journal represents the culmination of a great deal of effort; students have worked independently, in small groups, or with external mentors to produce original research or literature reviews in areas of their own choosing.

In these pages, you'll read about possible causes of epilepsy in low-income neighborhoods and about the potential for cutting edge photovoltaic cells in New York City architecture. You'll explore alternative, or "atypical," treatment for schizophrenia and the long-term effect of concussions on former athletes. You'll also examine the new field of epigenetics, the effect of the environment on genetic structure, and how that field can apply to potential treatment for cancer.

In many ways, a scientific journal is the ideal product for a science course. When students take a test, they demonstrate understanding of material presented by a teacher for that teacher, but when they produce a peer-reviewed journal article, they become the teacher and the expert, and their audience is the entire school community, if not more.

You, the reader, are the audience for this journal.

Explore these pages fairly and critically. Examine the articles for consistency and for application to other areas of science and the world at large. Take the time to completely digest and understand the material, and feel free to agree or disagree with the results.

Most importantly, enjoy these articles. They are the first in what we hope will be a long and respected series.

Sincerely,

Scott W. Rubin
Science Department Chair

Cole Kitchen

Cole is a senior at Berkeley Carroll and is passionate about researching sports injuries. After a year of studying climate change, he chose to change topics to focus on his true interest in sports and injuries. He has focused his research and reading on sport-related concussions, both at the youth and professional levels, while comparing short and long term effects. His paper discusses the flaws in the culture surrounding concussions, and the danger that playing with a concussion has on athletes at every level.

Francesca Longo

Francesca is a senior who has focused on the new field of epigenetics and their relation to women's cancers, in particular, breast cancer. She has spent the last two years reading about the implications of epigenetics and has honed in on what they can tell us about the scientific, medical fate of the cancerous disease. Going forward, she would like to pursue genetic research in her higher education.

Gabe Dash

Gabe is currently a senior at Berkeley Carroll. Next year, he will be attending Washington University in St. Louis, where he will be studying architecture. In his paper, he discusses the history of photovoltaics (solar cells) and the current state of research. Gabe then analyzes a hypothetical penthouse apartment in Manhattan that would incorporate solar panels into its design. He wishes to continue this research (albeit not officially) in college, where he wants to focus on sustainable development and eco-building.

Rebecca Glanzer

Rebecca is a senior at Berkeley Carroll and has been studying epilepsy in low socio-economic status neighborhoods and its relationship to other diseases over the past year at the Harlem Health and Well Being Study with Dale Hesdorffer, Ph.D. The study seeks the prevalence of epilepsy in Harlem and Washington Heights as well as determining other factors that may cause or correlate to new epilepsy after the first seizure. Her interest in epilepsy stemmed from her research in memory consolidation last year, as both are either the function or malfunction of the brain. She hopes to continue to study neurology in the future.

Samantha Bellamy

Samantha is a senior at Berkeley Carroll who has been interested in psychology all of high school and who is pleased to have gotten the opportunity to delve into her interest for the past two years. She has been researching schizophrenia and reading about the differences between atypical and typical antipsychotics as treatment for schizophrenia, and which is more effective. Sam will be majoring in psychology in the fall.

Table of Contents

The Life-Long Battle Against Concussions

by Cole Kitchen

Today sports-related concussions are being recognized as a major public health concern. With the recent volume of NFL (National Football League) athletes being forced to end their careers early because of head injuries, it's vital to understand the long and short-term affects of concussions in high school athletes. The Centers for Disease Control and Prevention estimates that approximately 300,000 sports-related concussions happen annually in the States, with an incidence rate of 3-6%. The flaw in those numbers however is that many concussions are going unreported and therefore the incidence rates are most likely far greater. The danger in unreported concussions lies in the possibility of SIS (Second-Impact Syndrome) which occurs after a person suffers a second concussion before healing from the first, essentially causing injuries to the brain that are either fatal or life changing. Case studies and tests have shown that far too few athletes truly understand the meaning of a concussion or the dangers of stepping back on the field too soon after experiencing one.

Unfortunately, a universally recognized definition of a concussion does not yet exist. In 2001, the first International Symposium on Concussion in Sport was organized by the International Olympic Committee Medical Commission. They defined a concussion as "a complex pathological process affecting the brain, induced by traumatic biomechanical forces." They also agreed that concussions involve temporary neurological function impairment that only heals with time, and that concussions rarely cause any structural damage to the brain. Historically physicians have categorized concussions through grading systems *(see figure 1)* that were far less accurate than we realized at the time. Additionally, CT scans and MRI's, once prominent testing methods, also have their flaws, in that concussions are ghost-like and such scans will only pick up structural damage. Therefore, past methods have been deter-

figure 1

Comparison of Historic Concussion Grading Scales

(Not currently recommended for use by medical professionals)

Guidelines	Grade 1	Grade 2	Grade 3
Cantu	Post-traumatic amnesia <30 minutes, no loss of consciousness	Loss of consciousness <5 minutes or amnesia lasting 30 min - 24 hours	Loss of consciousness <5 minutes or amnesia >24 hours
Colorado Medical Society	Confusion, no loss of consciousness	Confusion, post-traumatic amnesia, no loss of of consciousness	Any loss of of consciousness
American Academy of Neurology	Confusion, symptoms last <15 minutes, no loss of consciousness	Symptoms last >15 min	Loss of consciousness

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mined as inconclusive in observing concussive symptoms. The techniques of studying concussions the right and wrong ways will be discussed again further into the paper.

In 2003 the case study, *Unreported Concussion in High School Football Players,* written by Michael McCrea et al., opened eyes to glaring flaws in the culture of concussions in high school. The study was organized so that a total of 1,532 varsity football players in Milwaukee would anonymously be surveyed on questions concerning the frequency of concussions that they have experienced and how often incidents would go unreported. As previously stated, when unreported accounts are considered, the incidence rate, which is supposed to be at 3-6%, is commonly believed to be much greater. When the results came back from the high school students, the numbers showed that 30.4% and 29.9% *(see figure 2 and table 1)* reported a previous history of concussions in the preseason and postseason, respectively. Interestingly, 15.3% of players said that they suffered concussions throughout their season, and only 47.3% of those players ever reported the injury. It's generally believed that players that do not actually report concussion symptoms due to fear of being benched. However, the study shows another interesting statistic. The overwhelming majority of players said that it was because they didn't think it was serious enough at the time, while others said they did not know it was a concussion at the time. Both of these reasons simply stem from not being educated on concussions. In the study, a concussion was defined as "a blow to the head followed by a variety of symptoms that may include any of the following: headache, dizziness, loss of balance, blurred vision, 'seeing stars', 'feeling in a fog' or slowed down, memory problems, poor concentration, nausea, or throwing up. Getting "knocked out" or being unconscious does NOT always occur with a concussion *(Unreported Concussion in High School Football Played).*" Ultimately, the study showed that "…players seem to be largely unaware of common signs and symptoms indicating concussions and the potential

figure 2

Percentage of subjects with prior history of concussion

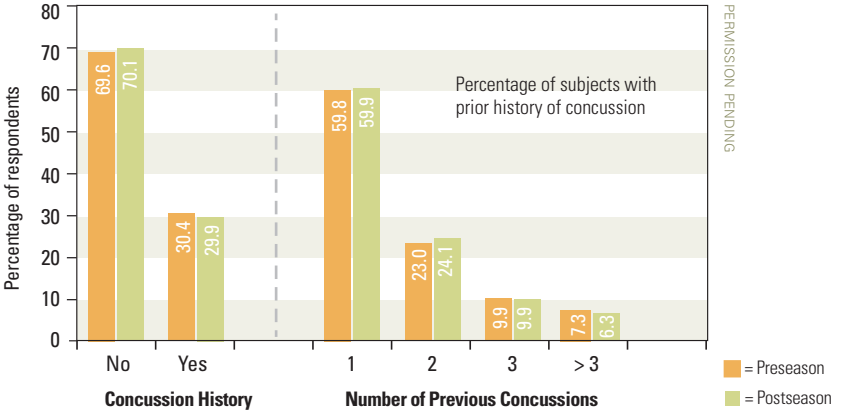

table 1

Concussion Reporting Data

Concussion Reported to	Percent of Subjects
Certified Athletic Trainer	76.7
Coach	38.8
Parent	35.9
Teammate	27.2
Other (eg, family physician, student)	11.7

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* Categories are not mutually exclusive; subjects were asked to check all that apply.

seriousness of continued participation…*(Unreported Concussion in High School Football Played).*"

Mild traumatic brain injuries (MTBI) are some of the most common injuries in children and teenagers. An MTBI is a "physiologic disruption of brain function caused by injury, with at least one of the following features: (1) a documented period of loss of consciousness of any length; (2) an antegrade or retrograde amnesic period; (3) any change of mental status at the time of injury; or (4) focal neurologic deficits that may be transient *(Second Impact Syndrome: A Rare, Catastrophic, Preventable Complication on Concussion in Young Athletes).*" While an MTBI is not a concussion alone, in most cases, when an MTBI is caused by a direct or indirect rotational force to the head and is followed by concussion-like symptoms, a concussion may be diagnosed. In the absence of visible symptoms, a young athlete might still be suffering from head trauma, sometimes weeks after sustaining the injury. As earlier stated, "Skull X-rays, CT scans, and MRI scans have failed to detect the abnormalities that might guide a physician to protect how soon a player might safely resume play *(Returning To Play After Concussion).*" Additionally, a method to predict reoccurring concussions has not yet been determined.

The danger in not documenting a concussion and/or not resting and letting your brain heal until it returns to normal functioning can be deadly. Normally an MTBI concussion will take one to two weeks to heal. However in some cases, symptoms may not be apparent for days or even weeks. "During the minutes to several days following a concussion, brain cells that are not irreversibly destroyed remain intact but are in vulnerable state produced by metabolic dysfunction" *(Second Impact Syndrome: A Rare, Catastrophic, Preventable Complication on Concussion in Young Athletes).* That said, youth are extremely vulnerable to the consequences of even small changes in cerebral blood flow and to increases in intracranial pressure and cellular hypoxia. If an athlete continues to play while still concussed, essentially they are playing with an injury that is exposed and can exponentially worsen. The most frequently discussed injury that one may become susceptible to is Second Impact Syndrome (SIS), which is defined as a "rapid cerebral edema and herniation after a second head injury" *(Second Impact Syndrome: A Rare, Catastrophic, Preventable Complication on Concussion in Young Athletes).* Moments after the second concussion the

athlete will collapse to the ground and experience "semicomatose, rapid pupil dilation, lack of eye movement, and respiratory failure *(Second Impact Syndrome: A Rare, Catastrophic, Preventable Complication on Concussion in Young Athletes).*" The condition, which occurs 2-5 minutes after injury, has a rapid death rate of 50%, and of the 50% that survive, 100% of the victims will be disabled for the remainder of their lives. Though there have only been 35 football related SIS cases recorded between 1980 and 1993, Dr. Sanjay Gupta points out "In high school players it's estimated that about one in 10 players have probably had a concussion. Forty percent of them went back to play the same game, and sixty percent of players who got knocked out cold by their concussion also went back to play the same day" (A Q&A with Dr. Sanjay Gupta). That said, thousands of students are leaving themselves susceptible to SIS every year.

Controversially, the only SIS cases ever recorded have occurred in maturing brains in people below the age of 18, which indicates that either professionals may not be at risk or that further understanding of SIS is necessary. In an article titled, *Same Day Play Nixed After Teen Concussions,* Dr. Michael W. Collins, points out "the younger you are, the longer it takes to recover from the injury… the only cases of second-impact syndrome have happened in adolescents and young adults, the point being that the developing brain is more vulnerable… At the college and professional level, I absolutely think there are instances where, if an athlete who's briefly symptomatic comes out, you exert them on the sidelines, do the mental status testing, go through a whole symptom profile, and they resolve very quickly, I would allow some of those athletes to play." *(Same Day Play Nixed After Teen Concussions)* That is not to say athletes can play before a first concussion is completely symptom free, but SIS has not yet been reported at the professional level.

Conversely in a recent study observing retired NFL players, results indicated that recurrent concussions might set players up for a greater chance of late life memory impairment, Mild Cognitive Impairment (MCI), and Alzheimer's disease (AD). The study conducted by Kevin M. Guskiewicz et al., is entitled *Association Between Recurrent Concussion And Late-Life Cognitive Impairment in Retired Professional Football Players.* In conducting the test, first, all members of the National League Retired Player's Association (n=3883) were sent a general health questionnaire that asked about their musculoskeletal, cardiovascular, and neurological conditions that they experienced during their time playing and since they retired. Months later, a second questionnaire was sent to a subset group of players (n=1754) who fit the bill for questions regarding memory loss and issues related to MCI. That same questionnaire was sent to the spouse/closest family member to that player to follow up on the player's responses.

From the original 3683 questionnaires sent out, 2552 were completed.

Prominent results are as follows:

- 1513 (60.8%) of retired athletes reported having sustained at least one concussion during their professional careers.
- 597 (24%) reported sustaining three or more concussions.

- 817 (54%) reported experiencing loss in consciousness.
- 787 (52%) experienced memory loss during at least one of their concussions.
- 262 (17.6%) believe the injury has had a permanent effect on their thinking and memory as they have aged.
- The results also showed a "higher prevalence of AD (Alzheimer's Disease) in the study population to the general American male population." *(see figure 3)*
- Retired NFL players scored similarly on the Mental Component Scale when compared to the general American male. However, when observing retirees with a history of a concussion, and especially several concussions, the scores of the former athletes are far more

figure 3

Alzheimer's Disease Prevalence Ratios for NFL Retirees and US male population

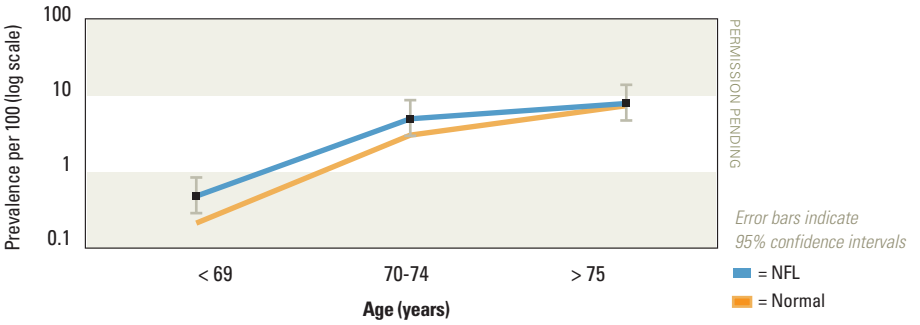


figure 4

MCS scores for the NFL retirees and population norms by age

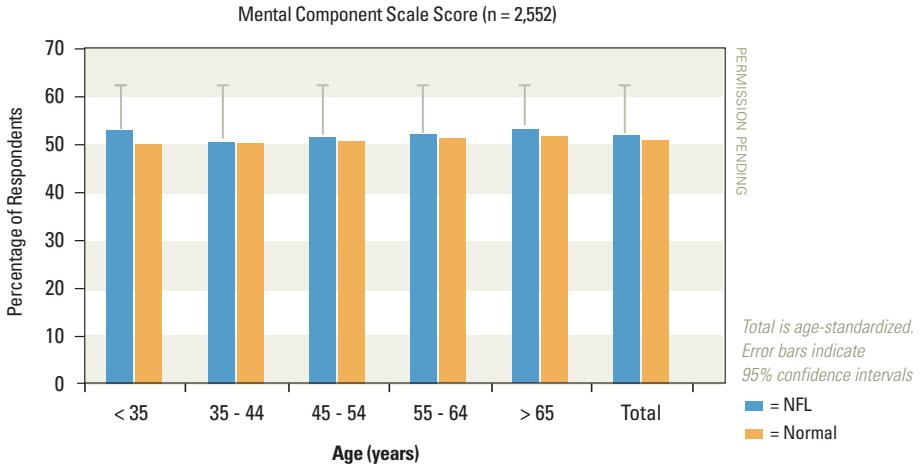
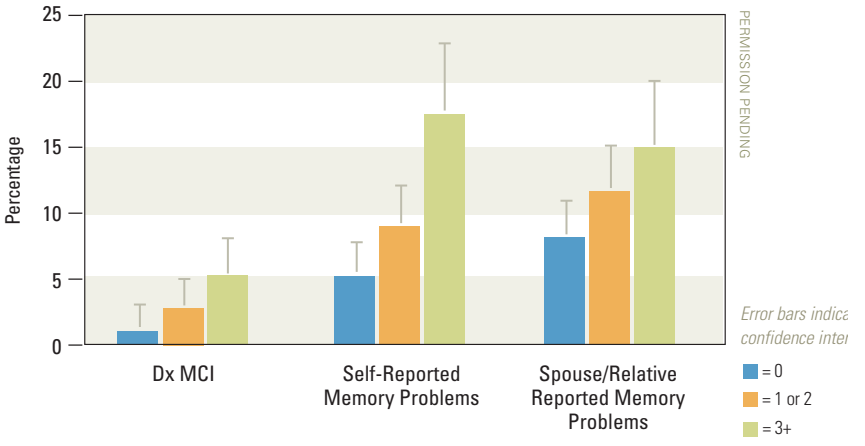


figure 5

Percentage of retired players aged 50 years or older with a diagnosis of MCI and memory problems.

(Self-reported and reported by a spouse or close relative)



disconcerting than the average American male. *(see figure 4)*

- Retired Players with a history of three or more concussions were at the highest risk of being diagnosed by a physician as having MCI and of having significant memory problems (these were based on their own accounts or those observed by their spouses or those closest to them that completed the questionnaire. *(see figure 5)*

Ultimately the data show that the mental health of former NFL players was declining at a much earlier age than the average American male. Most importantly, athletes with several concussions experienced even worse decline, which is a reason for concern considering "it is projected that the prevalence [of AD] will nearly quadruple in the next 50 years, by which time 1 in 45 Americans will be afflicted with the disease." *(Association Between Recurrent Concussion And Late-Life Cognitive Impairment in Retired Professional Football Players)* This calls into question how important playing through pain should be to players at all levels.

Some professional athletes still haven't been paying attention and as a result they're still not taking the necessary precautions to ensure their safety. In the case study titled, *A Prospective Study of Concussions Among National Hockey League Players During Regular Season Games: The NHL-NHLPA Concussion Program,* written by Brian W. Benson et al., scientists attempted to "determine rates of concussion and trends related to concussion in the NHL, to descriptively explore initial post concussion signs, symptoms, physical examination findings and time loss." *(A Prospective Study of Concussions Among National Hockey League Players During Regular Season Games: The NHL-NHLPA Concussion Program)* The scientists collected data from all NHL teams between 1997 and 2004 *(see table 2).* In the collected data, 559 in-game concussions were diagnosed, with an average of

table 2

Frequency and rate of concussion in National Hockey League (NHL) regular season games per 100 players
(1997-2004)

NHF Regular Season	Frequency of Concussion	Number Of Players	Rate of Concussion per 100 Players
1997-98	56	1218	4.6
1998-99	88	1249	7.0
1999-00	66	1347	4.9
2000-01	109	1419	7.7
2001-02	96	1470	6.5
2002-03	72	1457	4.9
2003-04	72	1459	4.9
Overall	**559**	**9619**	**5.8**

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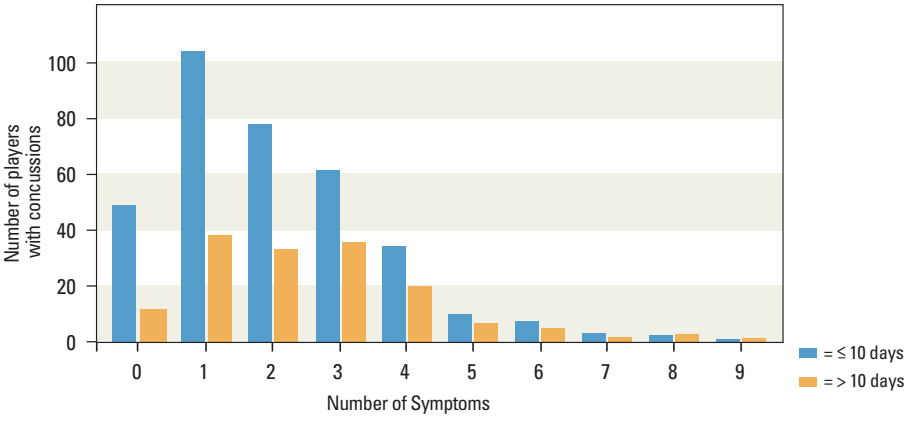
80 per season. The range of time (days) missed because of concussions ranged from 0-342 days with an interquartile range of [IQR] 2-13, which fits with the recommended 1-2 weeks rest. However, a Kruskal-Wallis test showed a significant difference in median time loss; in a "linear regression analysis that was adjusted for ages and position showed that, on average, time loss increased 2.25 (95% confidence interval) for every subsequent concussion sustained." *(A Prospective Study of Concussions Among National Hockey League Players During Regular Season Games: The NHL-NHLPA Concussion Program)* In another linear regression, after data was adjusted again for age and position, the average time loss was 1.89 days greater for every additional symptom that was recorded. Of those symptoms, through an invariable logistic regression analysis, data showed that headaches, loss of consciousness, low energy or fatigue, and light sensation were significant predictors in athletes who missed greater than ten days *(see figure 6)*. Interestingly, in 8% of all concussions, players were allowed to return to the game, with 57% of those players not missing any additional time after that game. Ultimately, the results showed that time loss due to concussions significantly increased after every subsequent injury and in the end enough hasn't been done to educate players on the dangers of playing through injury.

Looking ahead, a lot is being changed in how we understand concussions. Several doctors such as Alan B Ashare and Michael W. Collins have endorsed newer methods and bashed the old. Dr. Ashare writes, "The best new technique for evaluating concussion is cognitive and psychometric testing that begins with a preseason screening that can be used as a baseline when compared with congruent tests performed after concussion occurs." *(Returning to Play After Concussion)* Dr. Collins agrees and adds, "The consensus guidelines urge moving away from concussion severity scales… we know grading scales are not effective. You will never hear a grade of concussion come out of my mouth. It doesn't predict anything in terms of prognostic outcomes." *(Same-Day Play Nixed After Teen Concussions)* Also concurring Dr. Ashere's statement earlier in this paper regarding the ineffectiveness of

figure 6

Distribution of symptoms in players who experienced time loss of more than 10 days and less than 10 days.

Consistently documented initial postconcussion symptoms were headache, nausea, neck pain, fatigue or low energy, irritability, nervousness or anxiety, blurred vision, photophobia or sensitivity to light, dizziness and vomiting.



neuroimaging, Dr. Collins points out that until structural injury is suspected, it's pointless. Dr. Collins goes on further to add, "Concussion is not a structural brain injury, it's a metabolic (chemical processes) crisis… metabolic crisis involves injury-induced increased neuronal energy demand at the same time cerebral vasoconstriction (the constriction of blood vessels, which increases blood pressure) decreased energy delivery." *(Same-Day Play Nixed After Teen Concussions)*

It's inevitable that concussions will occur and that they are part of sports. However, scientists' understanding of traumatic head injuries has vastly changed for the better. The problem however is deeply rooted in the fact that the public is insufficiently educated on what a concussion specifically is and the danger it can possess. The short-term effects, especially in youth, can be deadly, as observed in cases of SIS. While the number of cases of SIS have been limited, the long term effects are very realistic for not only professional athletes, but everybody who experiences one or more concussions. As observed in the study on retired NFL players, the greater the number of concussions, the sooner one may not be able to perform simple tasks as efficiently as the typical human being. With changing techniques in determining concussions and their levels of severity, doctors have now begun to understand each patient on a more personal level, rather than going through symptoms and determining ones severity based on a graph. While leagues, both at the high school and professional levels, are implementing more rules to improve safety and monitor heads injuries, tests are continuing to show that the risk of playing hurt or obtaining several concussions only increases the risk of a life changing condition that young athletes in our 'hard hitting' culture hardly ever think about.

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Epigenetic Abnormalities and Breast Cancer

by Francesca Longo

Science is extensive. No individual biological phenomenon is capable of explaining the world around us. Rarely is there ever a single implication from scientific discoveries. Studying epigenetics and its relation to breast cancer has proven just how far-reaching scientific revelations can be. Epigenetics have been traced in some ways as responsible for or correlated to cancerous activity on a cellular level, but not just in one apparent manner. Epigenetics in cancer can be associated to the medical recognition of the disease, the deformities in the cell cycle, and in the end--hopefully the treatment.

Similar to a genome, we all have an *epi*genome. The field of epigenetics studies the changes to a chromosome that result without alterations to the DNA sequence itself. The word *epigenetics* means "above" genetics, which is fitting because there are no changes to our pre-determined genome involved. These "above" alterations can arise from surrounding molecules, such as proteins that interact with the DNA strand, as well as from the folding structure of the DNA strand itself.[2] This gets at the concept of methyl groups; a chemical structure being added on top of the strand of DNA.[4] Both the organization and chemical changes alter the way the genes in our DNA are expressed or inhibited, and therefore how proteins are produced and how our cells ultimately function.

The common understanding is that epigenetics have always been essential to our body's functioning. As is pointed out in 'Epigenetics and Human Disease: Translating Basic Biology into Clinical Applications', it is easy to forget that the overall time frame of knowledge of genetics at the molecular level began with the first accurate model of DNA by Watson and Crick only fifty years ago. Epigenetics have always been encoded in our systems, and the knowledge of their existence just explains gaps in complex human processes.

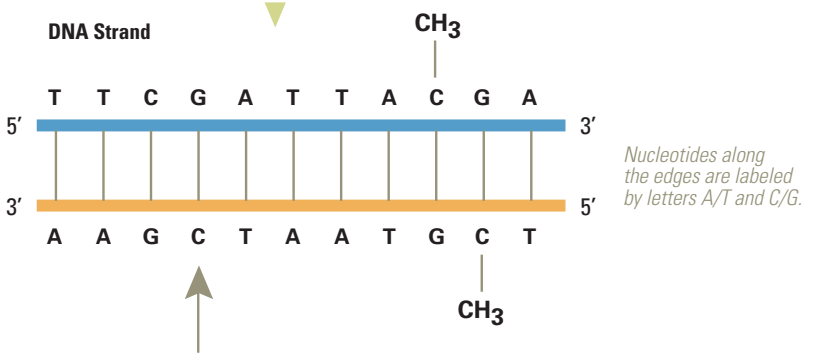
Epigenetic patterns are responsible for a variety of cellular functions in different stages of our lives, ranging from tissue specification to the production of diverse proteins in each cell.[2] This is *normal* and not 'deformed.' Our cells use methylation and histone (specific highly alkaline proteins in nuclei) modification to cater to what our body needs to maintain and function properly. This, for example, can normally entail the silencing of a protein that receives a stress hormone, or potentially the enabling of a gene that codes for a timed action. The problem with studying these patterns is that it is incredibly difficult to determine a 'normal' pattern of DNA methylation or chromatin (uncondensed DNA) structure in our bodies because of how circumstantial it is based on nature, nurture, and even just the time of day.[6] Changes in epigenetic patterns are normal and expected in our cells, but only to perform our bodies' functions. Regardless, scientists have now embarked on the search for cracking 'The Human Epigenome Project,' to determine the norm.[8] This knowledge will be valuable in studying the implications of epigenetics, but also to simply differentiate the normal from the abnormal.

The science of epigenetics can be broken down into two large subgroups: DNA methylations, and histone modifications. DNA methylation is the attachment of a methyl group to the cytosine or guanine bases of the nucleotide sequence, which changes the expression of these nucleotides[4] *(see figure 1)*. This system is regulated primarily by enzymes called DNA methyltransferases, otherwise known as DNMTs. 'Epigenetic and Human Disease' also accredits "correct organization of chromatin...and histone modifications, silencing repetitive elements, genomic imprinting*, and X chromosome inactivation" to DNA methylation. Epigenetics is about not only the chemical additions of methyl groups but also the DNA's organization and the changes required to get it to an altered state. Histone modifications are the alterations in how the DNA strands are packaged within the cell, enabling some sections to be literally hidden or exposed as sections *(see figure 2)*. This can resemble a sort of scrunching of chromatin, or changes in where the strand curls or stretches. These subgroups are how the epigenetics really are altered on a molecular level.

A huge part of these changes to be analyzed in future science entails the role of epigenetics in cancer (an obviously urgent field) and the implications for the world of medicine. Cancer is linked to DNA methylation in one way because of specific deformed conditions such as hypermethylation, hypomethylation, mutations at the methylated cytosines, and imprinting defects.[11] These altered states have been traced in diseased tissues. Hypermethylation silences genetic material such as tumor suppressors by condensing the chromatin. Hypomethylation "activates oncogenes [cancer cells], results in chromosomal instability [mutations upon replication], activates transposons [genes that can 'jump' placement]."[15] Mutations on this level result in incorrect or abnormal gene expressions. And finally,

figure 1

How a methyl group literally sits on top of this nucleotide at the cytosine location.[8]



Nucleotides along the edges are labeled by letters A/T and C/G.

figure 2

Histone modification has the ability to change how a DNA strand folds upon itself, hiding and exposing different segments of the genome.[5]

Genomic imprinting means that while normally an offspring's genome is a combination of the two parental genomes, DNA methylation patterns can be passed down directly from one parent to the offspring.

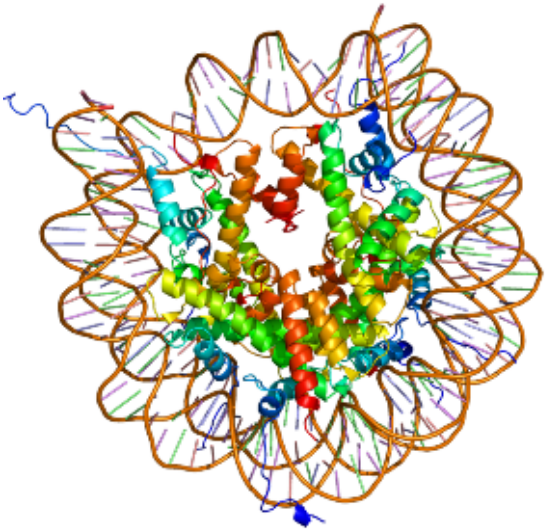
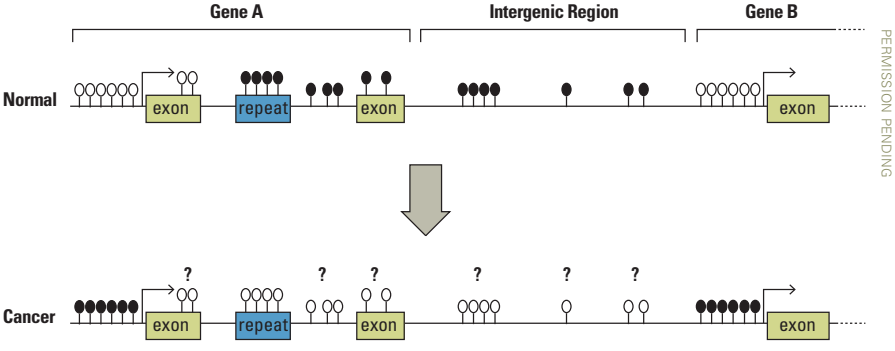


figure 3

Exhibiting the differences between normal methylation patterns on our DNA and those of cancer.

It is most important to pay attention to the opposite shading of the extra-molecular figures represented as black and white dots. These show what changes in abnormal methylation belts.[3]



imprinting defects "result in loss of parental identity." This basically means that upon imprinting, the cell is changed to an unrecognizable state compared to its original identity.

Obviously the term 'cancer' is used in a vague sense, but in fact each specific cancer and many other diseases can be connected to these methylation abnormalities in unique ways. The Bellvitge Institute for Biomedical Research in Barcelona elaborates: "both appropriate DNA methylation and histone modifications play a crucial role in the maintenance of normal cell function and cellular identity. In cancerous cells, these 'epigenetic belts' [segment of pattern of DNA methylation] become massively perturbed, leading to significant changes in expression profiles which confer advantage to the development of a malignant phenotype" *(see figure 3)*. The article 'Epigenetics and Human Disease' charts many of these different relationships and the genes they involve, whether it is the genes controlling the cell cycle or changing proteins which regulate this. It suggests that while there are many specific methylation conditions that affect the evolution of cancer, three main factors play a primary role in this. These include the overall changes in DNA methylation patterns for cancer-related DNA information, the activation of oncogenes and chromosomal instability, and lastly, the silencing of tumor suppressing DNA.

Epigenetics are essential to studying cancer prevention in addition to the causes of cancer. The article "Diagnostic and Therapeutic Applications of Epigenetics" discusses the implications of using specific methylation patterns to detect the risks of tumors, as well as the presence of active cancer cells. Theoretically, different epigenetic patterns can be used to identify cancer by scans and tissue tests if scientists and doctors could properly identify what they know to be cancerous and what is normal.

All of these applications show how prevalent epigenetic studies have become in the past decade. Since their discovery, issues that were stumping scientists beforehand, have begun to make more sense when traced to epigenetics. Unfortunately, this is not a simple correlation to observe and make sense of because it requires great molecular observation. The University College London's Institute for Women's Health draws this connection in regards to women's cancers:

> ***Over the last two decades, survival rates from women's cancers (breast, ovarian, endometrial and cervical cancer) have all but modestly improved despite huge efforts from both research and clinical communities. In parallel with this, the field of epigenetics has grown from its infancy into a promising scientific discipline. In particular, DNA methylation analysis has been adopted by oncologists in an attempt to better understand and manage cancer.*** *– Jones*

The field of epigenetics opens an envelope to all cancers in general, to women's cancers particular, and, extremely active right now, to breast cancer. Breast cancer exists as the primary cause of female cancer mortality, resulting in more than 450,000 annual deaths.[3] As the British Institute for Women's Health explained, survival rates remain steady regardless of breakthroughs in epigenetic science because observing trends in belt patterns is not concrete without a norm with which to compare them. While it makes sense to analyze abnormal tissues such as breast cancer, a great deal remains undiscovered about epigenetics in general.

In May 2011, The European Society for Medical Oncology reported on the "most comprehensive analysis yet of the epigenetic modifications present in breast cancer."[13] This follows up on the idea of a Human Epigenome Project, but in regards to breast cancer tissue. The study conducted by Dr. Sarah Dedeurwaerder from L'Université Libre de Bruxelles attempted to map out what normal frozen snapshots of breast tissue methylation patterns look like on the DNA strand, and how abnormal belts would appear.

In its introduction, the study lays the framework by explaining the four respected sub-types of breast cancer. Each is unique in its protein hormone receptors, and how their cells react to different stimuli amongst the cells. As the New York Times simplifies, "breast cancer cells may contain receptors, or binding sites, for the hormones estrogen and progesterone. Cells containing these binding sites are known as hormone receptor-positive cells. If cells lack these connectors, they are called hormone receptor-negative cells."[7] This explanation more clearly spells out all the complicated factors used to describe sub-types of breast cancer, as they often seem very complex and unapproachable:

a. Basal-like cancers (negative for estrogen receptor, progesterone receptor, and human epidermal growth factor receptor 2 [HER2])
b. HER2-positive cancers
c. Luminal A cancers (estrogen receptor-positive and lowly proliferative)
d. Luminal B cancers (estrogen receptor-positive yet highly proliferative)

These sub-types are relevant in terms of classifying cancers, and this study seeks to further the knowledge of each sub-type by associating DNA methylation patterns with it:

> ***An epigenetic difference between the tumors of these patients might explain the difference observed in terms of treatment response. Therefore, DNA methylation profiling could help to refine the current breast cancer classification and thus might help to stratify patients within a particular sub-type both in terms of prognosis and prediction to treatment response.*** *– Pavinato*

Upon identifying the key sub-types of breast cancer tissues, the article explains that in general, "cancer cells are characterized by a massive global loss of DNA methylation." It goes on in great detail to identify the problem genes in the breast cancer genome that affect different aspects of the cell cycle. By listing these pieces of the cell cycle and their corresponding gene names, the scientists explain all the different places abnormal belt patterns can alter how a cancerous cell will function in comparison. "The concerned genes are involved in important biological functions, such as cell cycle regulation (CDKN2A, CDKN2B, CCND2), DNA repair (MLH1, MGMT, BRCA1), cell adhesion and invasion (CDH1, TIMP3), and growth-inhibitory signalling (RARB, RASSF1A, SFRP1)." In increasing detail, the scientists sought correlations between the methylation state of these genes and their breast cancer sub-type. They looked to past research of specific gene sequences, and drew conclusions from their culminating findings. One example they provide of this significance specifically is an association "between methylated RASSF1A, CCND2, GSTP1, TWIST and the estrogen receptor-positivity." After providing more specific examples of methylation states and corresponding breast cancer sub-types, the authors conclude that the studies show a significant relationship between epigenetics and the classification of breast cancer habits. Different methylation patterns provide a route to identifying specific sub-types of breast cancer, which can increase the efficiency in diagnosis and treatment assignments. This will inevitably improve therapeutic care because it allows doctors to work within homogenous groups of the disease, instead of making generalized assumptions with specific variations of the disease.

Continuing on, the team evaluates the sub-types based on origin of cancerous cells and how that can be traced through methylation patterns. In conclusion they do find hints within different belt sequences. If nothing else, this suggests a significance in analyzing the close relationship between epigenetics and cancerous activity. The scientists explain that they are "heading towards a better understanding of breast cancer biology, but true genome-wide DNA methylation profiling studies [Human Epigenome Project] are needed in order to assess the contribution of the entire DNA methylome."

Dr. Dedeurwaerder and team worked to amplify the knowledge of epigenetics. By looking at one specific yet prevalent analysis, it becomes clear how far-reaching this science can be. While these scientists in Belgium conducted this particular study on extremely specific gene sequences in breast cancer cell cycles, a parallel study could be performed in regards to other women's cancers, other cancers, and other diseases that show correlation to epigenetic activity. The researchers continue on in their writing to say that their analyses, "highlighted the need to evaluate the epigenetic component in order to gain better knowledge...the epigenomic exploration of breast cancers has only just begun."[3]

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Photovoltaics and Applications in Architecture

by Gabe Dash

Abstract

As energy demands continue rising to ever-higher levels, it is important to create energy source infrastructure that will help keep power flowing both now and in the future. Solar power is a viable option to satisfy growing energy needs. Annually, the sun provides 10,000 times the Earth's energy consumption requirements. If as little as three percent of the planet's paved area were devoted to photovoltaic collectors, it would provide more than enough energy for the world's needs. However, solar panels are not widely distributed because they require a lot of space to generate significant amounts of power and are very expensive, both in terms of production and maintenance, which means both high resource and labor costs. A growing section of photovoltaics, organic photovoltaic cells, or OPV, appear to be able to solve the main problems associated with solar power. OPVs are smaller, more flexible, and cheaper to produce than the standard silicon-based collectors. A major application for OPVs is in architecture, through sustainable development and eco-building. Architects can incorporate OPVs into buildings to solve space limitations and also reduce solar energy costs. This research paper will explore the different types of solar panels, focusing specifically on OPVs and their uses. It will discuss the possibilities of incorporating these solar panels into modern architecture by using an example of a hypothetical penthouse apartment at 135 West 14th, Manhattan.

Introduction

Photovoltaics can trace their roots back to 1839, when A. E. Becquerel discovered the photovoltaic effect, or the creation of a current when an object is exposed to light.[1] Almost fifty years later, Edward Weston filed US Patent 389,124, "Apparatus For Utilizing Solar Radiant Energy," on October 17, 1887.[2] This is generally recognized as the first solar panel. The next great breakthrough for solar energy occurred in 1954, when Bell Laboratories announced the creation of the first silicon-based solar cell.[3] The way silicon or crystalline-based solar cells work has not changed since then. Sunlight hits the solar cell, exciting electrons on the semiconducting silicon, which is between an anode and a cathode. These excited electrons then create a current, which flows from the anode to the cathode and out of the cell to power a vast array of devices. The layer of silicon is known as the "active layer," or where the electrons are energized. Bell Laboratories' first cell was able to convert roughly 6% of sunlight into usable energy. Since then, the basic structure of silicon-based, or crystalline structure, cells have changed very little, but the materials composing the cell have. The technology has progressed in two directions. The first is to increase the amount of energy from the Sun converted into usable energy, or efficiency. The second is to reduce the cost of production and maintenance.

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A Bell Laboratories engineer with a solar cell, in 1954 [4]

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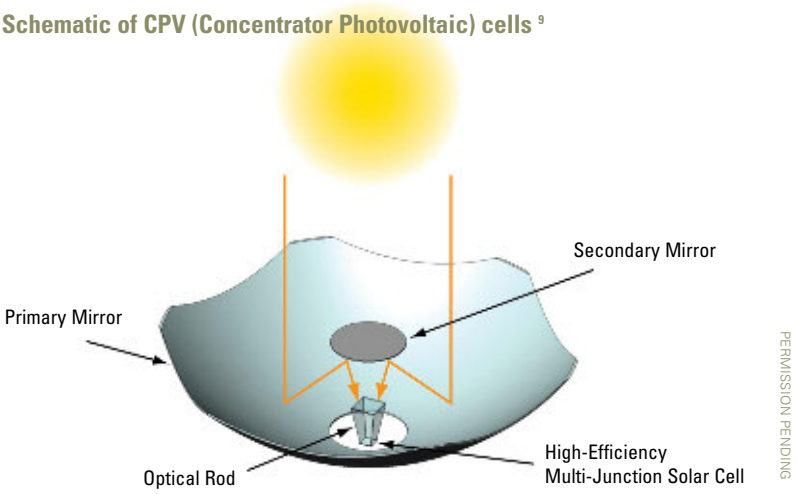
A rooftop solar panel installation in Munich [5]

There have been two noteworthy advances in conversion efficiency in the past few years. The first is a U.S. Department of Energy funded cell, created by Beoing-Spectrolab, which achieved a record-setting 40.7% conversion efficiency in laboratory conditions in 2006.[6] The cell is a 'concentrating solar cell', meaning it does not use only natural sunlight, but also concentrated photovoltaic (CPV) technology. CPV concentrates light into a smaller area with optic lenses, increasing efficiency of each individual cell.[7] Besides using CPV technology, the cell also employs a multi-junction. It is composed of layers; each one specialized for a different section of the spectrum of light emitted by the sun. In theory, an infinite-junction cell could have a conversion efficiency of 86.8%. Most multi-junction cells are currently made with around 20 layers.[8] Combining CPV and multi-junction cells lead to very efficient cells, due to the fact that concentrated light amplifies the multi-junction effect *(see figure 1)*.

In 2007, University of Delaware announced they had created a solar cell that reached a conversion rate of 42.8% in laboratory conditions, breaking the U.S. Department of Energy

figure 1

Schematic of CPV (Concentrator Photovoltaic) cells [9]



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record. While a 2% increase may not seem large, technological advancements in efficiency has been measured mainly in tenths of a percent, so such a large jump is a significant achievement. The University of Delaware's cell uses CPV, however, instead of simply firing concentrated light to the solar panel, it separates the light into high, medium, and low energy light waves, allowing for increased absorption and therefore increased efficiency. This technology is initially intended for use in the military. Soldiers carry packs upwards of 100 lbs, almost a fifth of which are batteries. Incorporating solar panels could greatly reduce the weight of field equipment. Looking towards the future, this technology could possibly be much more portable and therefore be suitable for laptops, cars, and more.[10]

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CPV solar array[11]

Problems

Crystalline structure solar cells have a few drawbacks that make them less attractive than other types of solar cells. For one, they have consistently been very expensive. One of the main focuses of solar energy research has been to find ways to produce solar panels more cheaply, lower the cost of maintaining them, and increase the lifetime of the cells. Since increasing efficiency effectively lowers the cost, most research is invested in increasing conversion rate. By some estimates, the best silicon-based solar cells cost around US $0.25-0.65/kWh, (kilowatt hour) depending on location, which company used, and other factors. Electricity produced by consumption of fossil fuels, on the other hand, costs around US $0.05/kWh. Additionally, crystalline structure cells are costly to maintain, as they are made of rigid and fragile materials.[12] Advances in efficiency, production techniques, or installation costs will help resolve the price disparity between solar cells and fossil fuels. However, it is generally agreed that making solar energy competitive with fossil fuels is not a far off goal.[7]

A second problem encountered by silicon-based solar cells is lack of raw materials, namely, silicon. The solar industry must go up against the computer industry, the largest user of silicon in the world. Solar-grade silicon is known as polycrystalline silicon (silicon composed of many small crystals), and in the early 2000s, demand far outstripped supply. Only twelve plants were operating worldwide in 2000, and polycrystalline silicon price jumped from US $9 to US $60, over a 600% increase.[7] Recently, factory upon factory have been opening up, and there are currently 66 polycrystalline silicon producers, which has caused a price drop of 93%. This may cause many companies, especially those of Chinese ownership, to abandon ship, and the polycrystalline silicon market may lose up to two-thirds of its suppliers.[13] Needless to say, polycrystalline silicon prices and supply are not stable.

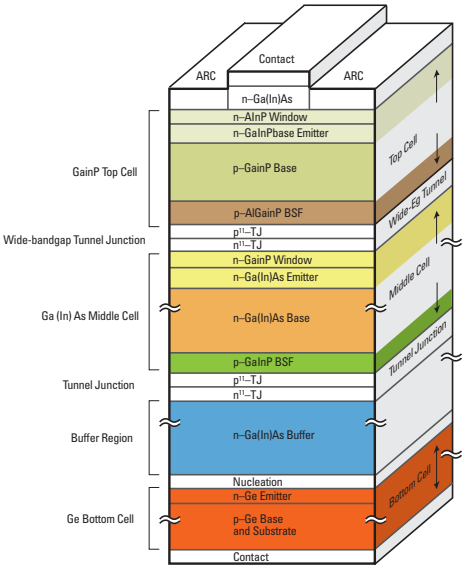
Finally, another large problem for the solar panel industry is the difference between laboratory and real-world conditions. While strict lab conditions may lead to 42.8% conversion efficiency, solar panels available on the market have only reached 17.24% (a panel manufactured by Sanyo Electric) when tested in real world conditions.[14] This disparity is often discouraging. Also, one solar cell is not effective on its own. It must be gathered together with many other cells in order to produce significant amounts of power.

OPV's (Organic Photovoltaics)

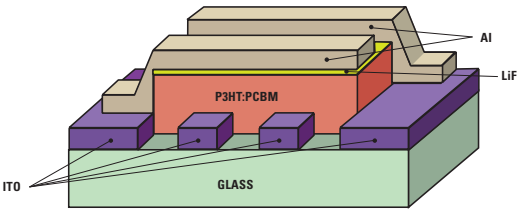
A relatively young but extremely promising area of solar energy research is in organic photovoltaic cells. These cells are different, because rather than being silicon-based, they are carbon-based, thus are called organic cells. The typical structure of an organic photovoltaic cell (OPV) is composed of three layers. The first is an anode and the last is a cathode, as with silicon-based cells, but what sets OPVs apart is the middle layer. While some solar cells use a multi-junction as the active layer, OPVs use a bulk-heterojunction. Instead of rigid layers, bulk-heterojunctions use an electron donor and acceptor mixture, so the material that provides electrons and the material that accepts electrons are mixed. There are organic cells with a single layer as the active layer, and cells with separated electron donor and acceptors, but those have not been as efficient at converting sunlight as bulk-heterojunction cells.[15]

figure 2

Comparing Crystalline Cells and OPV Cells



Crystalline Cells typically have over 20 layers[8]



OPVs Cells typically have three layers, and therefore are much thinner[15]

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OPVs can be preferable to crystalline cells because they are thinner. Instead of many layers stacked on top of each other, OPV cells only have one layer, and so can be very thin—less than 300 nanometers.[15] *(see figure 2)*

The real benefit provided by OPV cells is cost. First off, while silicon-based cells require glass to offer the cells protection, OPVs can be produced between layers of plastic, which is extremely inexpensive to produce and maintain. Second, the molecules used to create the cells are cheap and abundant, further lowering the cost. Third, organic molecules absorb a large amount of light, visible and not. What multi-junction cells have that OPVs lack are layers specialized for different light wavelengths, but the high absorption spectrum of organic molecules means that even with a smaller active layer, it can still absorb a lot of light. Finally, while crystalline-based cells are very fragile, with many layers and a glass casing, organic photovoltaics, especially those with a bulk-heterojunction cased in plastic, are much more durable. One of the biggest benefits provided by OPVs is a drop in necessary maintenance, which makes them more cost effective. These factors—production costs, resources costs, absorption spectrum, durability, and maintenance costs—are what make OPVs so attractive as a field of research.

However, OPVs do have one glaring weakness: very low conversion efficiencies. Currently, the record is around 6%.[16] OPV technology is young, only really gaining traction in

the early 2000s. Therefore, while having such a low conversion rate is not the biggest problem in the world, it is a substantial obstacle.

Luckily, there is one aspect of organic photovoltaic cells that holds seemingly unlimited potential: the flexibility of organic molecules. Because of the many ways organic molecules can be combined, organic photovoltaics have the potential to outstrip other solar energy sources.[15] And when combined with nanotechnology, another infant field, the combinations are endless. Nanotechnology is predicted to have applications in every area of life, from medical research to communications to environmental rehabilitation. Nanotechnology is so wide reaching because it concerns manipulating matter on an atomic scale. Creating such small technology, usually on the order of 1 to 100 nanometers, offers a wildly different approach to conventional processes. This field, which currently knows almost no limits, combined with OPV, is an interesting and promising combination. The flexibility of OPVs is only compounded by the flexibility of nanotechnology. Currently, there is little research being done into nanotech applications for OPVs, but what has been published is very promising.

Architecture

Bringing together architecture and solar energy is key for sustainable development. Buildings sit in the sun all day long, and so it is a smart idea to utilize passive solar collectors. Many buildings in New York already collect water when it rains, and use it to provide heat or power. Using the city's infrastructure to our advantage is not a radical idea; rather, it is simply not common practice with solar panels, despite how many buildings are prime candidates for this method of energy production. Take, for example, 135 W 14th Street, Manhattan, New York *(see figure 3)*. There are innumerable ways to equip the building with solar collectors, but this paper will focus on a hypothetical penthouse apartment.

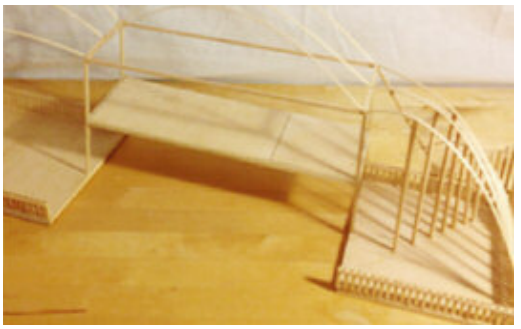
The New York grid plan, which every building north of 14th street adheres to, lies 27.5° off of the north arrow. This means that the southeastern tip of each building receives the most sunlight each day. The penthouse has vertical slats in the southeastern corner, which could hold solar collectors quite easily. The arches from the southern wall to the eastern wall are situated to catch light at almost all angles of the sun as it moves across the sky. These would require a more flexible material, however. The middle section of the penthouse would also hold solar panels. However, the rear section is tilted away from the sun, so solar panels there would have little or no effect, and therefore would be a waste of money. Finally, the rear section of the penthouse would be a bad location for solar panels because beginning in the early afternoon, surrounding buildings cast shadows on it.[17]

The best-suited material for the southeastern corner is Pramac Luce MCPH. These models have 9.1% conversion efficiency, and can also absorb much of the infrared spectrum. The cell is a multi-junction cell, rather than OPV, but it is thin enough to make it very suitable for buildings *(see figure 4)*.[17]

figure 3

135 West 14th Street, Manhattan, NY proposed penthouse

The majority of solar collectors would be placed on the southernmost portion, to maximize sunlight exposure.



135 W 14th Street, Manhattan, NY

Proposed penthouse constructed with wood (from the west)

For the rest of the building, there are a few more options for solar collectors. The first is Konarka Power Plastic. Konarka is different because it responds to sunlight, and efficiency grows during the hours with the most sun *(see figure 4)*.[17]

A second option is a Kalzip product, AluPlusSolar. This collector is a thick sheet that is rolled out on top of a roof, like a tarp. This is suitable for the penthouse because of the many curved edges. Also, AluPlusSolar is thick and difficult to damage, providing both insulation and durability *(see figure 4)*.[17]

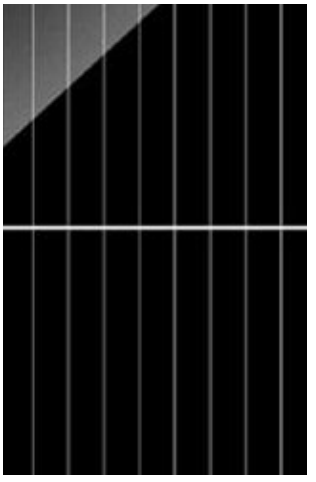
Finally, an MIT researcher has developed a 'solar textile.' While intended for flexible housing in places such as Africa, it is very effective because it grows in response to the sun, increasing the volume of the cell. It can also be as transparent or opaque as desired *(see figure 4)*.[17]

Conclusion

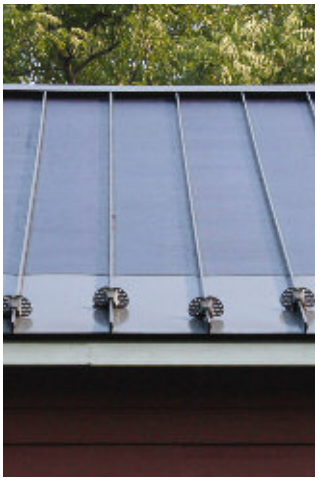
We are headed towards an energy crisis. Energy consumption is skyrocketing, and meeting the new demand with fossil fuels will wreak havoc on our environment. We can avoid poten-

figure 4

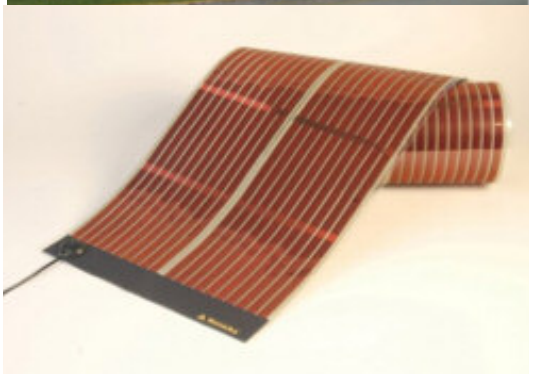
Proposed Penthouse Solar Options



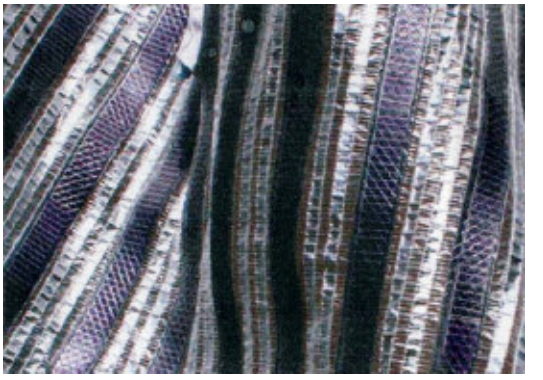
Pramac Luce MCPH[17]



Kalzip AluPlusSolar[17]



Konarka Power Plastic[17]



Solar textiles[17]

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tial disaster through the liberal and effective use of alternative energy. Of all the different energy sources available, solar power is a very powerful tool. To not utilize the sun would be a foolish mistake, as the sun can and will provide all the energy needed by humans. And by incorporating solar energy into architecture, it can be easily brought into our cities and power supply.

There are a variety of solar collector products available. They range in almost every dimension: size, composition, cost, conversion efficiency, conversion method, and more. However, the market for solar collectors is not huge. Therefore, mass distribution of solar panels is not feasible here and now. However, at the rate the solar industry is growing, supply should catch up to demand in a few years.

Alternative energy needs to become second nature. It needs to become part of our culture. It is cleaner and less destructive than mining for coal, drilling for oil, or burning natural gas. And why not solar? By incorporating solar panels into the infrastructure, energy costs will lower, the air will be less polluted, and a new industry will rise, perhaps based right here in America to bolster our sagging economy.

In the end, the benefits of solar panels are vast and numerous, whereas the costs are small and surmountable.

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Could Stress be the Missing Link Between Epilepsy and Low-Income?

The Harlem Health and Well Being Study

by Rebecca Glanzer

Introduction

This school year I have had the privilege of working under Dale Hesdorffer, Ph.D. at the Columbia University Medical Center. Under the larger umbrella of convulsive disorders and traumatic brain injury, Dale Hesdorffer has focused her studies especially on epilepsy's relationship to lower-income neighborhoods in New York City. I am so thankful for the chance to observe the intricacies of professional scientific research through the Harlem Health and Well Being Study.

Abstract

The goal of this experiment is to estimate the incidence of first unprovoked seizure and epilepsy in lower income neighborhoods and to find the missing link between higher rates of epilepsy and lower socio-economic statuses. Questionnaires are performed at the first seizure, and then again every four months for newly diagnosed cases of epilepsy. The hypothesis is that low socio-economic status causes stress, which in turn causes epilepsy. Stress is measured by the existence of comorbidities, the collective efficacy of a neighborhood, a lack of epilepsy specialist care, and a history of depression and anxiety.

Background

According to the National Institute of Neurological Disorders and Stroke, epilepsy is a disorder in which neurons in the brain signal abnormally. In a functioning brain, neurons communicate over a synapse. The following image *(see figure 1)* is of one neuron[3]. The shorter extensions are dendrites, which accept neurotransmitters from across the synapse. A synapse is just a space between one neuron's dendrite and the other's axon, the tail-like extension at the other end, which in this picture is labeled the nerve ending. Neurons line up so that as neurotransmitters are released by one neuron's axon and then taken up by the other's dendrite, a message is passed along until it reaches a body part that will do something in response to the stimulus that caused the neurotransmitter message to be sent. The chemical composition of the neurotransmitter corresponds to a certain response. Most neurotransmitters fall into two categories: inhibitory and excitatory. Inhibitory neurotransmitters stop a current action, while excitatory neurotransmitters begin one. In an epileptic brain, inhibitory and excitatory neurotransmitters in the brain can either elicit the wrong responses or improperly regulate the quantities of the neurotransmitters. This results in a much more intense signal than would occur in a non-epileptic brain. This burst is manifested in "strange sensations, emotions, and behavior or sometimes convulsions, muscle spasms, and loss of consciousness"[1]. A seizure is an incident of these electrical signal bursts. A person is then diagnosed with epilepsy if they have had at least two seizures.

figure 1

Illustration of a Neuron

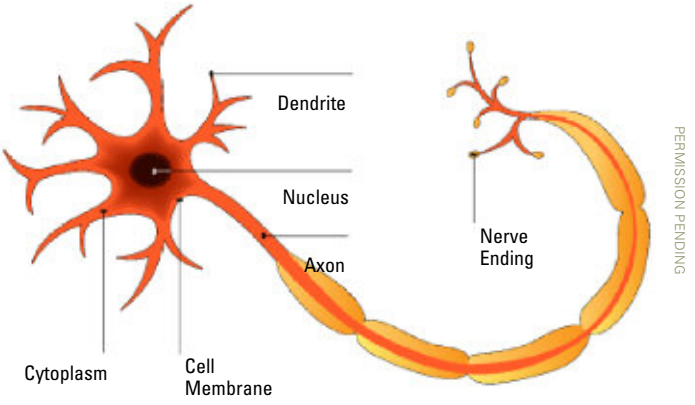

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figure 2

An Electroencephalogram shows a Person's Brain during a Seizure[11]

EEG's measure and display electrical brain activity, which increases during an epileptic episode. The dotted vertical lines represent time in seconds, and we can see the seizure occur on the right half of the image as electrical impulses are being fired more rapidly by neurons.

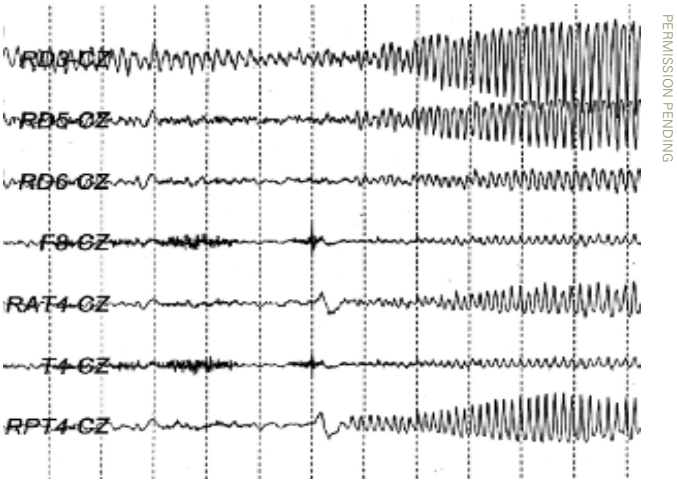

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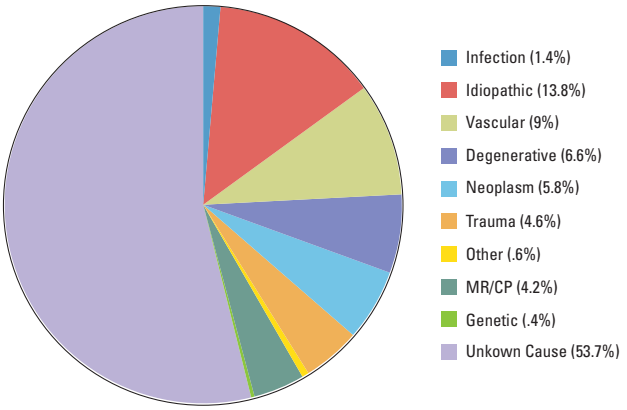
Figure 2 above [11] shows an electroencephalogram of a person's brain during a seizure. Epilepsy is a broad term in that there are many possible etiologies, or causes. Causes for seizures can range from brain damage in a motorcycle accident to a genetic sequence, but what is most interesting in research today are those cases without any obvious source and the fact that these make up 53.7% of epilepsy incidents. The pie chart [2] *(see figure 3)* breaks down the known causes. Infection encompasses mainly viruses, for example meningitis, that

figure 3

Etiologies of Epilepsy



infiltrate the brain and disrupt neuron function. Idiopathic epilepsy manifests itself in early childhood and adolescence. Often there are genetic markers, but no anatomical brain abnormalities show up in tests. Vascular epilepsy comes from malformations that develop and press on the brain, damaging neurons. Degenerative epilepsy results from diseases like Alzheimer's that break down brain matter. Neoplasm is another word for tumor, in this case a brain tumor. Tumors are groups of cells that do not regulate their growth and then cannot perform usual functions, like cell signaling. This malfunctioning signaling can lead to seizures. Traumas can lead to brain damage, which in turn can cause seizures. MR stands for mental retardation. It is thought that similar genes cause both mental disability and epilepsy (8); however little is known about why they are connected aside from that they both are due to abnormalities in brain function. Genetic etiologies are similar to idiopathic ones, except that the anatomical differences between the epileptic brain and the non-epileptic brain are clearer when epilepsy is clearly genetic. And then of course, the majority of cases have no known cause, and even the known causes can't be entirely explained.

Epilepsy is often treated as if it has one cause and one manifestation. This study investigates epilepsy of a specific variety—seizures with an unknown cause in low-income neighborhoods—to gather specialized data about incidence in this particular situation. In surveys by the Centers for Disease Control, it has been found that rates of epilepsy increase as income decreases and education decreases. In 2003, Hesdorffer also conducted a study, which found higher rates of epilepsy in lower-income neighborhoods[6]. The collected data in the current study may provide a link between low incomes and higher rates of epilepsy, in this case hypothesized to be stress.

Stress comes in the form of comorbidities: diseases that are linked together either because one causes the other or because they are both caused by a third factor. Comorbidities add to stress because they are another disease to treat and their treatments

must be paid for, which is difficult especially when one is in poverty. When looking for relevant comorbidities, we begin with diseases that affect the brain because epilepsy is a malfunction of brain. Epilepsy is therefore usually grouped with depression, anxiety, and other psychological disorders.

Depression and suicide attempts are thought to be due to neurochemical pathways important to the development of epilepsy[7]. It is not clear whether there is correlation or causality, but the two disorders seem to have a bidirectional relationship: people with epilepsy seem more likely to develop depression and people with depression seem more likely to develop epilepsy. There are probably genetic factors that play into the development of both diseases, but the current belief also holds that depression and epilepsy are due to dysfunctions of similar parts of the brain, which is what causes them to arise so often together.

A second form of stress lies in the collective efficacy of a neighborhood, which is defined as a measure of how willing neighbors are to assist each other[4]. In other words, it describes the sense of shared space and unity of an area or neighborhood. In poorer neighborhoods, there tends to be less collective efficacy. It is thought that being unable to rely as heavily on neighbors or being in a less safe environment adds to stress.

Procedure

When people come into the emergency room because of a seizure, they are screened for this experiment. Subjects are gathered from the Columbia Presbyterian Hospital and St. Luke's Hospital. If the patient's seizure fits the study's criteria, the patient is asked if he or she would be interested in participating.

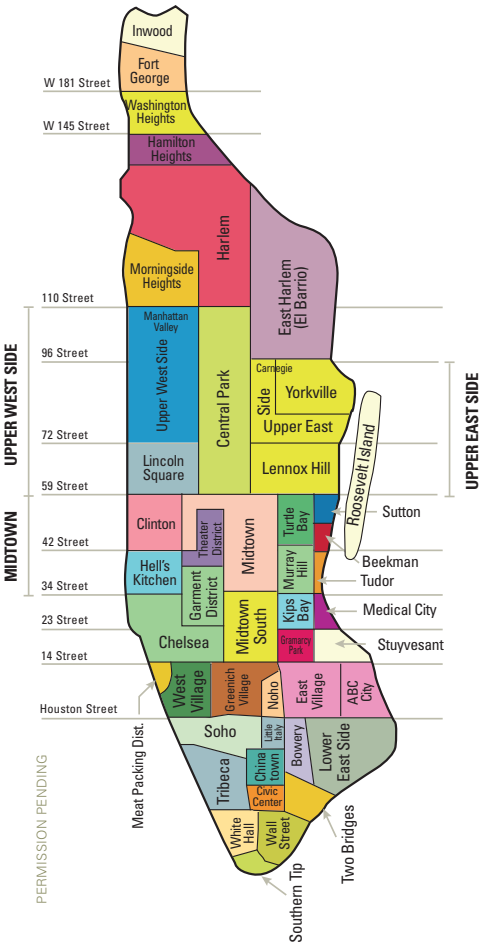
The first interview is conducted at baseline after the first seizure. Data are gathered on demographics, the subject's first seizure, a personal and family history of depression and anxiety, psychological disorders, comorbidities, and the collective efficacy of their neighborhood. Follow-up interviews are conducted in four-month intervals, during which time data are added about any more incidents of seizure. The subjects are followed for 24 months.

Collective efficacy in this experiment is measured with a questionnaire. An example question is, "If children were skipping school on a regular basis, would the community respond?" Selecting 1 means the subject highly agrees that action would be taken, while 4 means no action would be taken by the community. Answers correspond to a point system, and scores dictate levels of collective efficacy.

In this experiment, initial seizures cannot be acute symptomatic seizures—the direct result of alcohol abuse, drug abuse, trauma, high fever, or other isolated event—because seizures with these causes are one-time events. Subjects therefore will not likely develop epilepsy. Subjects must also live in Washington Heights and Harlem, shown on the map (9). The included zip codes are 10026, 10027, 10030, 10031, 10032, 10033, 10034, 10035, 10037, 10039, and 10040 *(see figure 4)*. These neighborhoods were chosen because they are poorer neighborhoods in New York City, and conveniently the laboratory is in Washington Heights next to Columbia Presbyterian Hospital, where potential subjects might seek medical treatment.

figure 4

Etiologies of Epilepsy



Error Analysis

The room for error in this experiment comes from the fact that subjects do not always remember accurately their experiences. Especially if a parent is speaking for a young child who is not yet capable of expressing how he or she felt, many details can be lost. This problem is exacerbated when the events described took place months or years before interviews.

Another issue is that most of the subjects' psychological data come from a standardized test whose results are based entirely on what the subject has described, which may be inaccurate, exaggerated or understated. The exam, the SCID (Structured Clinical Interview for Disorders), cannot even be performed on children because it is so highly subjective.

The last important point is that the population size for this study cannot possibly be as large as would be ideal. This is mainly due to the fact that the criteria are so specific and rigid. Regardless, the study is likely to provide useful data.

Conclusion

The aim of this experiment really is to learn more about this mysterious disease by seeking its relationship to lower income neighborhoods through diseases and conditions that cause stress.

While the experiment is still in progress, if there is a correlation found between epilepsy and psychological disorders, that could alter the way we treat both diseases. If a patient is diagnosed with epilepsy, greater attention might be given to their emotional health as well. By analyzing family histories we can also find patterns that might help in preventative treatment. Both depression and epilepsy have genetic factors, and identifying those would be crucial in anticipating future cases.

Most experiments are looking for a specific result that will prove a hypothesis; however, this experiment is one of the first to gather mass amounts of data about epilepsy so that future experiments can be designed with a stronger base of knowledge.

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Treatment of Schizophrenia with Atypical Antipsychotic Medication

by Samantha Bellamy

Background

Schizophrenia is a chronic psychotic illness that affects about 1% of all Americans. Cases of schizophrenia are said to have appeared as early as the 18th century, but the term "schizophrenia" (from Greek roots meaning "splitting of the mind", although schizophrenia is not split personalities—a common misconception) was coined in 1908. Symptoms of schizophrenia may start appearing in people from the age of sixteen to thirty years old. Men and women are both equally affected by the illness. The symptoms of schizophrenia are split into positive and negative symptoms. Positive symptoms are symptoms of psychosis, or losing touch with reality. Negative symptoms are associated with emotional changes. The positive symptoms include hallucinations, delusions, and agitated thoughts and movements. There are five different types of hallucinations including tactile (eg, feeling bugs crawling up one's arms), visual (sight), auditory (hearing), olfactory (scent), and gustatory (taste). Delusions are beliefs that are irrational and unshakeable. There are two types of delusions: non-bizarre and bizarre. Non-bizarre delusions are beliefs that are plausible, but still untrue and strange. Bizarre delusions are completely impossible, such as the idea that one has godly powers or can control the weather. The most common type of delusion in schizophrenics is paranoia. Schizophrenics may display symptoms of thought disorder, which means that they cannot organize their thoughts or may randomly start and stop talking. Agitated movement causes a person to either become catatonic or to display repetitive movement. The negative symptoms of schizophrenia have to do with mood. These symptoms include most of the symptoms of depression such as anhedonia (the loss of interest in activities that should be pleasurable), the inability to keep friendships and jobs, or laziness. Negative symptoms can become so intense that schizophrenics are unable to perform everyday tasks on their own. In order to be diagnosed with schizophrenia, a person must display two or more positive and negative symptoms for a full month, and any number of positive or negative symptoms for six months (National Institute of Mental Health).

The most common treatment for schizophrenia is medication; more specifically, antipsychotics. Antipsychotics affect the dopamine levels of the brain. Dopamine is a neurotransmitter that is associated with a person's sleep patterns, attention span, memory, motivation, voluntary movement, and gratification. Symptoms of psychosis are said to have to do with a flood of dopamine in the brain. Below is a visual of how a neurotransmitter travels through the nervous system; the "flood" of dopamine would be an excess of the neurotransmitter being transmitted to the synapse *(see figure 1)*.

figure 1

How Neurotransmitters Travel through the Nervous System

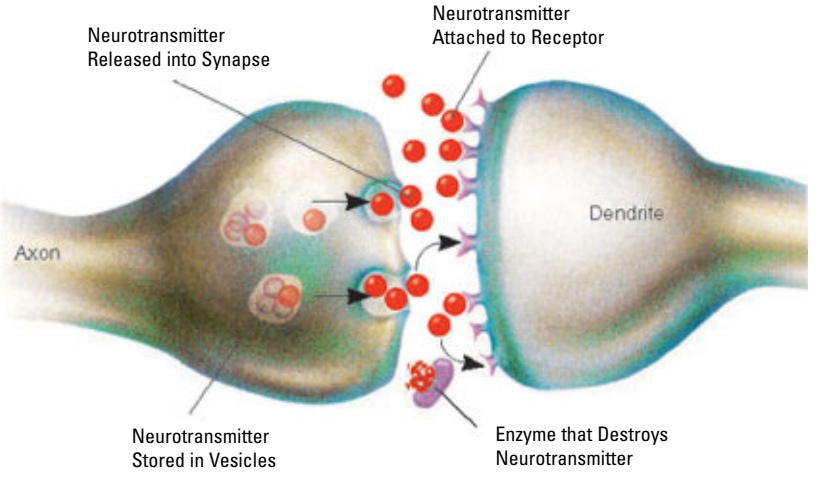

IMAGE FROM "THE SYNAPSE AND DRUG ADDICTION"

Symptoms of psychosis are said to have to do with a flood of the neurotransmitter dopamine in the brain. The relationship between dopamine and the psychotic symptoms are clear, so most drugs that are used to treat schizophrenia aim at controlling the amount of dopamine in the brain. Antipsychotic drugs block D2 receptors (sites in brain synapses where dopamine is received), leveling out the amount of dopamine that is being transported through the pathways of the nervous system (The American Journal of Psychiatry).

Recently, atypical antipsychotics have been introduced to treat symptoms of psychosis. Atypical antipsychotics are also called second generation antipsychotics, implying that they are similar to conventional antipsychotics, but newer.The first atypical antipsychotic to be introduced was clozapine in the 1990s and most other atypical antipsychotics are modeled after clozapine. Atypical antispychotics have been shown to have less extrapyramidal side effects. These side effects have to do with movement; it is common for antipsychotics to either restrict a person's movement or make them unable to stop moving. Atypical antipsychotics have also been shown to be more effective in treating the depressive mood symptoms of schizophrenia (ACTA Psychiatrica Scandinavica). This review will examine studies that explore the efficacy of these atypical antipsychotics as a treatment for schizophrenia.

Goals

Because atypical antipsychotics have been introduced fairly recently, the distinct differences between typical and atypical antipsychotics have not been completely defined yet. There is still research being done on how effective atypical antipsychotics are as treatment for psychosis disorders and as treatment for schizophrenia specifically. Three studies will be discussed in this review: a systematic review and meta-regression analysis of the use of atypical antipsychotics as treatment for schizophrenia by John Geddes, Nick Freemantle, Paul Harrison and Paul Bebbington published by the British Medical Journal, research on the actual effectiveness of atypical antipsychotics for the treatment of schizophrenia done by Jeffrey A. Lieberman et. al. published by the New England Journal of Medicine, and a study considering if the effectiveness of atypical antipsychotics is due to their "fast dissociation from the Dopamine D2 receptors" in the brain done by Shitij Kapur and Philip Seeman published by the American Journal of Psychiatry. These studies compare typical and atypical antipsychotics, and the efficacy of each type as a treatment for schizophrenia. The first two studies analyze atypical antipsychotics and show data for how many patients preferred them, how many patients found them ineffective, and certain side effects of the medication. The American Journal of Psychiatry article considers if there is any difference in how each antipsychotic affects the reuptake of dopamine in the brain. It hypothesizes that it has to do with how quickly the antipsychotics dissociate, or separate themselves from, the D2 receptors in the synapses. Atypical antipsychotics dissociate from the receptors faster than typical antipsychotics, and it has been theorized that this has to do with the efficacy of atypical antipsychotics and the reduction in side effects. This paper will compare and contrast each of these articles and analyze the results by discussing the outcome of each study and whether or not they concluded that atypical antipsychotics are an effective treatment for chronic schizophrenia.

Review of Results

The American Journal of Psychiatry study had to do with how and why atypical antipsychotics work. As was previously mentioned, all antipsychotics work with the D2 receptors of the neurotransmitter dopamine. To reiterate, antipsychotics block these receptors, which levels out the amount of dopamine that is being transported through the pathways of the nervous system. This study questions if what makes atypical antipsychotics different from conventional antipsychotics is the fact that they dissociate from the receptors faster than conventional antipsychotics.

The connection of the receptors and the drug that is acting on them are measured by "association and dissociation" rates. These terms refer to how much of the receptors a drug occupies before causing it to reach equilibrium, and how quickly it takes the drug to dissociate (or leave) the receptor. The longer a drug occupies a receptor, the longer it takes to work and the more effect it will have on the nervous system thereby causing more side effects.

figure 2

How Neurotransmitters Travel through the Nervous System

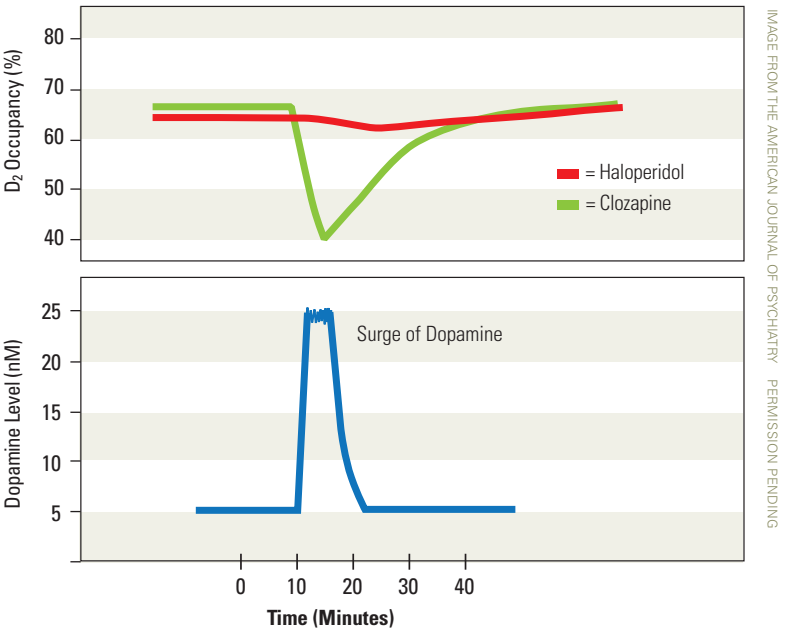

IMAGE FROM THE AMERICAN JOURNAL OF PSYCHIATRY PERMISSION PENDING

The following graph *(see figure 2)* displays how a surge of dopamine affects the amount of the D2 receptor that a conventional antipsychotic (haloperidol) and an atypical antipsychotic (clozapine) are occupying. At the moment of the surge of dopamine in the receptor, both haloperidol and clozapine occupy approximately the same amount of the receptor, but immediately after, clozapine begins to dissociate from the receptor, while haloperidol barely dissociates within forty minutes.

The discussion of the study states that "it is proposed that drugs with fast dissociation when used in doses that lead to appropriately high D2 blockade modulate the dopamine system in a manner that allows for more appropriate functioning of physiological systems and that this leads to what is currently called the atypical antipsychotic effect." In conclusion, the fast dissociation from D2 receptors is something that can define atypical antipsychotics and separate them from conventional antipsychotics. It was confirmed that in the treatment of psychosis, medication works with dopamine neurotransmitters rather than serotonin neurotransmitters (the neurotransmitter that antidepressants interact with) in the treatment of psychosis. However, the long-term effect of medications with faster dissociation was not confirmed. The hypothesis that medication with faster dissociation is more effective was not confirmed.

The study in the British Medical Journal was a "systematic review of the effectiveness and tolerability of atypical versus conventional antipsychotics in the treatment of schizophrenia". It compared six atypical antipsychotics (amisulpride, clozapine, olanzapine, quetiap-

ine, risperidone, and sertindole) with two typical antipsychotics (haloperidol and chlorpromazine). The analysis measured the percentage of patients treated with the conventional antipsychotic who "did less well than the average of the group given an atypical antipsychotic." Specifically, the standardized effect sizes (differences between two variables, i.e. people taking atypical versus typical antipsychotics) were measured. In this study, the standardized mean difference is the difference between the mean of people who preferred atypical antipsychotics, and the mean of people who preferred conventional antipsychotics, divided by a standardized deviation based off of those who strayed from the average. This distribution was turned into a percentage of patients who had more side effects from conventional antipsychotics. Each percentage represents the number of people who "had a higher symptom score" with the conventional antipsychotic compared to the average number of people on atypical antipsychotics. The results were as follows:

- Amisulpride: 64%.
- Clozapine: 75%.
- Olanzapine: 59%
- Quetiapine: No difference in overall symptom score.
- Risperidone: 66%.
- Sertindole: Irrelevant, as the drug is no longer available.

It was determined that although the atypical antipsychotics cause fewer side effects than conventional antipsychotics, they are not overall more beneficial. The discussion of the study states that "when [they] controlled for the higher than recommended dose of conventional antipsychotics used in some trials, a modest advantage in favour of atypical antipsychotics in terms of extrapyramidal side effects remains, but the differences in efficacy and overall tolerability disappear", suggesting that many of the perceived benefits of atypical antipsychotics are really due to excessive doses of the comparator drug used in the trials. In other words, a higher dose of typical antipsychotics was used than is normal used for the treatment of schizophrenia, which may have caused more side effects and therefore caused patients to prefer atypical antipsychotics. The study found that when the amount of medication was evened out (i.e., a patient was given an amount of an atypical antipsychotic that was equivalent to the regular dose of haloperidol), there was no significant difference other than the fact that the medication caused fewer extrapyramidal side effects. In conclusion, both drugs are efficient treatments for schizophrenia.

The study published in the New England Journal of Medicine discussed the efficacy of four atypical antipsychotics: olanzapine, quetiapine, risperidone, and ziprasidone, and one typical antipsychotic: perphenazine, in treatment for schizophrenia. The main focus of the study was comparing the number of patients who dropped out of treatment with each drug due to either lack of efficacy, intolerability, or drop-out due to the patient's own decision. The study did not discuss the differences in the four atypical versus the one typical antipsychotics.

Each atypical antipsychotic had a high number of patients who discontinued their treatment. For each antipsychotic, the drop out rate "for any cause" was in between 64 and 82 percent. The article discusses that drop out rate "[indicates] substantial limitations in the effectiveness of the drugs." However, it does suggest that olanzapine appeared to be the most effective of all of the drugs tested, for reasons unknown (it had the lowest drop out rate). The study also takes into account the side effects that each drug caused. None of the drugs showed significant neurologic effects, but olanzapine (even though it had the lowest drop out rate) did show side effects of weight gain and significant and concerning changes in cholesterol and hemoglobin. The study found that it is difficult to determine how this will affect future use of atypical antipsychotics, but there will be much discussion about the balance between efficacy of the drug and the number of side effects it causes. Some patients may rather have fewer side effects, for example, and therefore choose atypical antipsychotics over conventional antipsychotics.

Discussion

The American Journal of Psychiatry study does not compare the efficiency of atypical and typical antipsychotics, but does help define the line between atypical and typical antipsychotics. It proves that there is a difference in D2 occupancy between atypical and typical antipsychotics and that this plays a role in 1) how many side effects a medication causes and 2) why the medication is effective in general. The atypical antipsychotic used in this study was clozapine, which was the first atypical antipsychotic that was introduced and the one which most other atypical antipsychotics are modeled after. This study confirmed that the fast dissociation from the receptors is part of the reason why atypical antipsychotics are effective and cause fewer side effects, but did not state that atypical antipsychotics were more or less effective. Other than providing information about how antipsychotics work on a neurological level, this study does not contribute significant information in the comparison between atypical and typical antipsychotics.

While atypical antipsychotics are an advance in medication for schizophrenia, the line between typical and atypical still remains blurry. The most significant difference between the two is that atypical antipsychotics cause fewer extrapyramidal (having to do with movement) side effects--this does not mean that they are necessarily the better or more efficient medication. The meta-regression analysis in the British Medical Journal that compared typical and atypical antipsychotics calls into question the comparison of the dosage of each medication. It considers that perhaps the reason some patients prefer atypical antipsychotics is because they are being over-prescribed compared to conventional antipsychotics--causing worse side effects than if they were being prescribed a normal dosage. If both a conventional and an atypical antipsychotic were given to a patient with the same dosage, the atypical antipsychotic would be more effective because it requires a higher dosage, while patients would be less tolerant of conventional antipsychotics in higher doses because conventional atypical antipsychotics require a lower dosage, and a higher dosage would cause more side effects. So, while all of the patients taking atypical antipsychotics had higher symptom

scores with conventional antipsychotics, most of the percentages were within fifty and seventy percent, and the percentages must still be questioned due to the dosage of conventional versus atypical medication that was administered to patients.

The study in the New England Journal of Medicine showed how many patients discontinued treatment with atypical antipsychotics for various reasons. This study is important because while many patients may prefer atypical antipsychotics due to the fact that they have fewer extrapyramidal side effects, they may still be less tolerant of the medication or the medication may not be efficient for them. The percentage of patients who discontinued their treatment with antipsychotics for any reason was high with the exception of olanzapine, which was the medication that appeared most effective out of all atypical antipsychotics. Each antipsychotic had within seventy and eighty two percent of patients discontinue their treatment. This makes one reconsider the growing idea that atypical antipsychotics are "better" than conventional antipsychotics. One thing that is important to note in discussing this study (although it was not discussed within the study) is that compared to the atypical antipsychotics, perphenazine had essentially the same or close to the same rates of discontinuation as the atypical antipsychotics. There was no significant difference between typical and atypical antipsychotics, and side effects were not studied (other than the fact that all of the antipsychotic medication used in the study had no significant differences in neurological side effects). Patients and doctors essentially have to find a balance between the number of side effects that a medication causes and the efficacy of the medication.

Through these three studies, atypical antipsychotics appear to have no significant difference when compared to typical antipsychotics other than the side effects. None of the studies suggests that atypical antipsychotics are a superior medication and none of the studies recommends that they be used in place of typical antipsychotics. In fact, the meta-regression analysis states that typical antipsychotics should still be the preferred medication for schizophrenia. There is much more known about conventional antipsychotics, so it is appropriate that they are still the suggested use of medication. There is still no evidence that atypical antipsychotics are more efficient in treatment for all patients, or that there is anything about them that might make them more efficient than conventional antipsychotics.

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OR TO PERFECT THE OLD."

KING CHARLES – 1661

(from the 1661 Charter for the formation
of the Royal Academy of Science;
the proceedings of which
are the oldest journal in existence)