Undergraduate Research Abstracts

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Sincere gratitude is extended to the Yeshiva University Offices of the Provost and Admissions for their critical support and endorsement of this publication.

Dedication

In Tribute to our friends and mentors,

Eli Steinberger (USRP Co-President 2006-07)
&
Donny Ladell (USRP Co-President 2005-06)

Who founded many student research initiatives, including this journal, at Yeshiva College. May we continue to honor their memories through the exhibition of Yeshiva's ongoing undergraduate exploration of science.

Biology | Chemistry | Computer Science | Mathematics | Physics | Psychology


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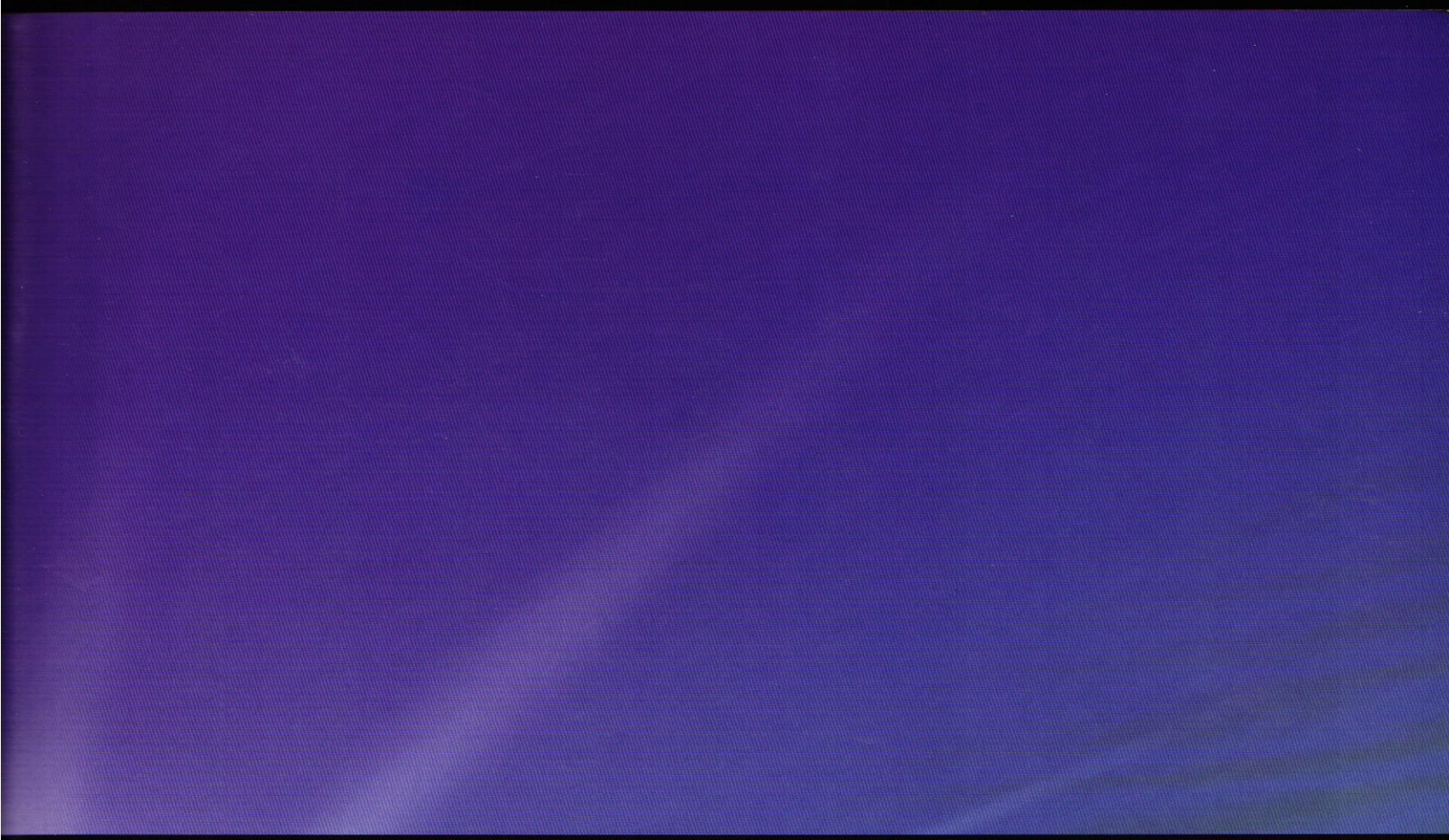

The differences between a scientific notion and one that is not can be condensed into a single word: testable.

Conducting research allows our undergraduate students to experience firsthand the process of science. It helps them realize that all scientific hypotheses and theories found in textbooks today were once just ideas that were put to the test and are now heavily supported by evidence.

Research has many formats and can be exciting or frustrating, can be simple or demanding, can be fruitful or challenging. But regardless of the specific field or project, it is a vital component of the undergraduate college experience at Yeshiva University. It challenges students to stretch their imagination, skills and knowledge and connects them with the scientific community around the world.

The scientists before us have started the journey; it is now our turn to steer the wheel of the scientific voyage towards new and exciting directions. This publication offers a glimpse into the research projects that caught the interest of our undergraduate students at Yeshiva College and Stern College for Women and reflects their commendable patience, passion and dedication.

Science is not "finished" and, I dare to say, will never be. This young generation of ambitious undergraduate students is in charge of the scientific breakthroughs of tomorrow and, as this publication shows, is already busy uncovering them one experiment at a time.

Fabiola Barrios-Landeros
Assistant Professor of Chemistry
Yeshiva University


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BIOLOGY

Neuroligin-2 Mimetics as Novel Anti-Diabetic Treatment

Rebecca van Bemmelen, Anna Munder, Shirin Kahremany, Efrat Shtriker, and Arie-Lev Gruzman
Department of Medicinal Chemistry, Bar Ilan University, Ramat Gan, Israel

The loss of β -cell functions, specifically the diminished ability to secrete insulin and respond to glucose, plays a major role in the progression of type-2 diabetes. The goal of this research is to develop an anti-diabetic treatment by synthesizing a compound that will cause pancreatic β-cells to increase their production of insulin, even if they are under stress conditions. It was recently found that similar to neurons, pancreatic β –cells contain anchor proteins: neuroligins and neurexins on their plasma membrane, which help guide 3D intracellular formation and interactions between β –cells. Among these β –cell proteins, neuroligin-2 (NL-2) and neurexin-1 (NX-1) are most important. Through computer based molecular modeling, our lab determined the binding site of the NL-2 to which NX-1 binds; this nine amino acid peptide (HSA-28) was then synthesized and conjugated to a dendrimer nanoparticle to form the compound HSA-28D (the cluster of HSA-28). We hypothesized that HSA-28D interacting with a NX-1 on the β-cell-surface would modulate insulin expression and secretion, improve β-cell resistance to cellular stress, prevent apoptosis and increase β-cell mass. To test our hypothesis, we co-cultured the HSA-28D with rat INS-1E cells, which were used as an in-vitro model for β-cell study. Our initial results showed that when cells were co-cultured with varying amounts of HSA-28D, the amount of cells greatly increased. In addition, we determined that with increased cell proliferation, the intracellular level of insulin was significantly elevated in the cells treated by HSA-28D. This was measured by the estimation of the levels of C-peptide (green signal, Figure 1), a component of the insulin precursor, which was used as a marker for insulin secretion. Finally, we also found that HSA-28D had a positive effect on the β-cells viability even in the presence of the endoplasmic reticulum and oxidative stressors. We hope that HSA-28D and other NL-2 mimetic compounds will be promising therapeutic agents for diabetes, and that they can be used for the creation of stem cell derived artificial islets for future transplantation in diabeticpatients.

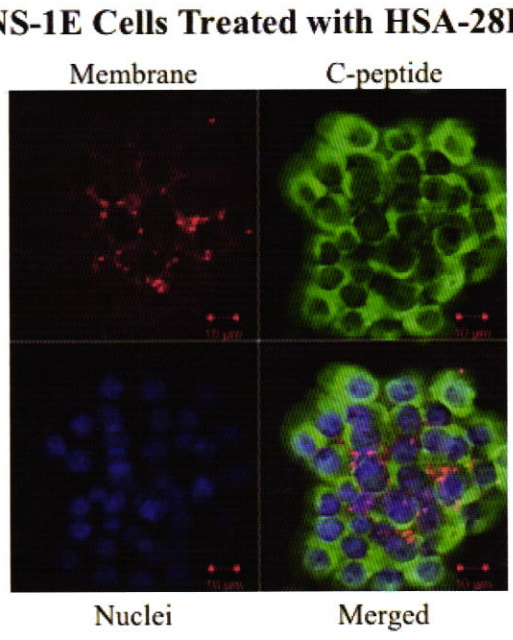


Figure 1. Effect of HSA-28D on C-peptide level in INS-1E cells, visualized using anti C-peptide antibody followed by secondary antibody-green signal.

Rebecca van Bemmelen is a senior at Stern College majoring in Biology. In her spare time, she likes to run and is a member of the YU cross-country team. She plans to pursue an MD in the coming years.

Understanding Impaired Lipid Absorption in Germ Free Mice

Dafna Meyers[1], Kristina Martinez[2], and Eugene B Chang[2]
[1]Stern College for Women, Yeshiva University, New York, NY; [2]Department of Medicine, Section of Gastroenterology, University of Chicago, Chicago, Il

It is widely accepted that the gut microbiota plays a role in the development of obesity. Recent studies reveal that germ free (GF) mice are resistant to high-fat diet-induced obesity. Reports suggest this was due to an impairment of lipid absorption in GF mice. Herein, we aimed to determine the mechanisms underlying this phenomenon. Based on preliminary results showing that GF mice have reduced lipase activity in the small intestine, we hypothesized that GF mice have impaired signaling of enteroendocrine hormones such as cholecystokinin (CCK) and secretin. To initially test this hypothesis, CCK and secretin expression in the duodenum, CCK alpha receptor (CCKaR) expression in the pancreas, and gallbladder size were examined in GF and Specific Pathogen Free (SPF) mice. Gallbladder weights were heavier in the GF mice compared to in the SPF mice (Figure 1). Although not significant, CCK and secretin gene expression in the duodenum were reduced in GF mice as compared to the SPF mice (Figure 2). Additionally, CCKaR gene expression in the pancreas was significantly lower in GF mice but pancreatic lipase (PnLip) and pancreatic amylase 2b (PnAmy2b) were not significantly altered (Figure 3). Taken together, these data suggest that due to impaired secretin and CCK signaling, less bile and pancreatic enzymes are released to properly emulsify and digest fats, respectively. This may ultimately lead to impaired lipid absorption and thus resistance to high-fat diet induced obesity in GF mice.

Figure 1

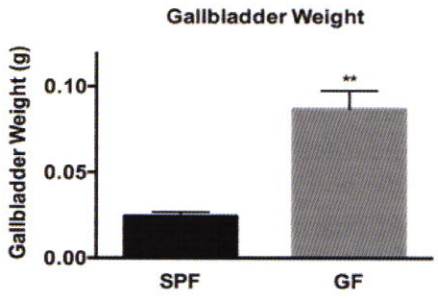


Figure 2

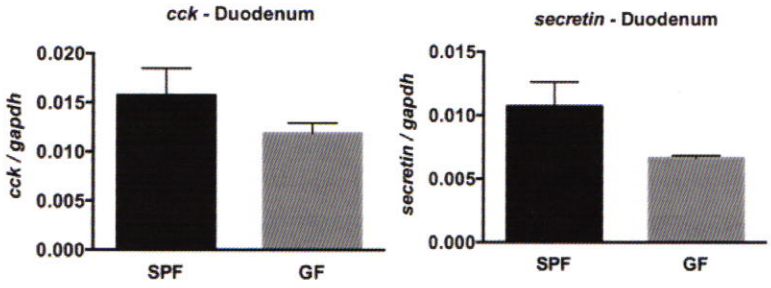
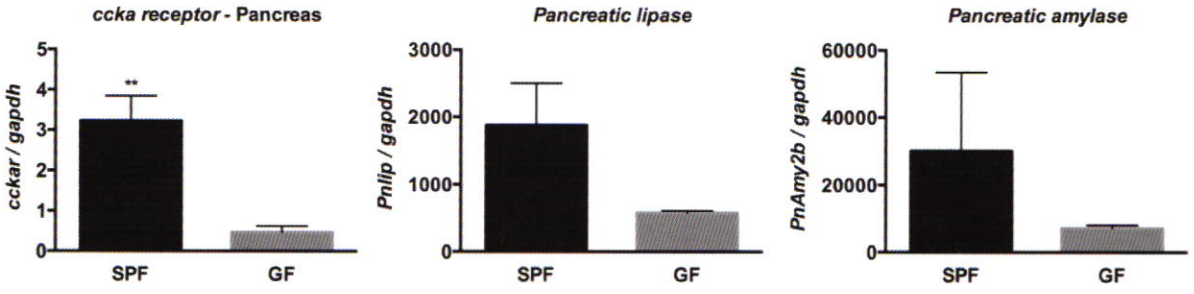


Figure 3



Dafna Meyers is a junior at Stern College for Women majoring in biology.

Neuronal Engineering: The Effect of Metallic Nanoparticles on Neuronal Growth and Differentiation

Michelle Katz, Michal Marcus, and Orit Shefi
Department of Engineering, Bar Ilan University, Ramat Gan, Israel

Nerve regeneration following tissue injury or disease is a major challenge in the neuroscience field. The search for regenerative agents that promote neuronal growth and repair is of great interest. Different molecules and materials have been shown to induce and affect neurite outgrowth and elongation. Previous experimentation has shown that iron oxide nanoparticles (NPs) promote neuronal growth and differentiation. Our study tested whether different metallic NPs (gold and silver) would promote significant neuronal growth and differentiation as well as the iron oxide NPs were shown to have promoted.

For experimental purposes, PC12 cells were utilized as the neuronal model. PC12 cells, derived from a pheochromocytoma (neurodocrine tumor) on a rat's adrenal medulla, serve as a common model for neuronal differentiation. In response to nerve growth factor (NGF), PC12 cells differentiate into neuron-like cells and grow neurites. In our project we incubated the metallic nanoparticles with PC12 cells and studied their effect on the cells' growth and differentiation process. Nanoparticles of all metals had a diameter of 20nm.

First, gold and silver nanoparticles at different concentrations were tested for their toxicities through an XTT Assay, thereby determining their cell viability in the PC12 cells. The gold NPs had an insignificant toxicity effect and proved viable for the PC12 cells to uptake; the silver NPs, however, were found to be toxic to the cells.

Next, the cells' morphology was studied along their differentiation processes. The experiment composed of capturing images and measuring the neuronal growth of PC12 cells seeded on collagen-coated plates under a light microscope over the course of seven days (days 1, 3 and 7). The growth of PC12 cells was compared based on the different composites of particles added. We measured and analyzed the neurites using the Neuron J Program, finding that there was a significant increase in the length of neurites of cells treated with the gold NPs as compared with that of neurites of the untreated cells.

Our research contributes to the field of neuronal repair, hoping to benefit pharmacological pursuits in the application and treatment of neurodegenerative diseases, such as Parkinson's, Alzheimer's and Cerebral Palsy.

Michelle Katz is a senior at Stern College majoring in the Physical Sciences, and minoring in Biology and Studio Art. In her spare time, she volunteers for Project START and hosts her own radio show on Israel. She'll be applying to dental school this summer, and hopes to attend after her studies at Stern.

The Effect of High and Low Molecular Weight Hyaluronan (HA) on the Synovium

Shoshana Mond, Claire Shortt, and Thorsten Kirsch
Department of Orthopaedic Surgery, Musculoskeletal Research Center, NYU School of Medicine, New York, NY

Osteoarthritis (OA), also known as degenerative joint disease, is the most common type of arthritis. OA is a disease not just of the cartilage but also of the joints. The synovial fluid is a thick viscous liquid that lines articular cartilage and prevents friction between subchondral bones. Synovial fluid contains hyaluronan (HA), proteinases, lubricin and collagenases that help coat the surface of the cartilage. Furthermore, synovial fluid is produced by the cells and contains all the inflammatory factors needed when studying inflammation.

Hyaloronan (HA) is a significant contributor to cell proliferation and migration. HA coats each cell (chondrocyte) in articular cartilage and is a major component of the cartilaginous extra cellular matrix. High molecular weight (HMW) HA contains several anti-inflammatory properties that can be useful during cartilage repair. In contrast, low molecular weight (LMW) HA may act as a strong inflammatory mediator. While HMW binds to the cell receptor CD44 to induce signaling, LMW binds to receptors such as RHAMM or toll like receptors (TLR 2, TLR 4) to induce signaling. LMW can also become fragmented by reactive oxygen species (ROS) and hyaluronidases within the body. LMW hyaluronan fragments were generated by treating the cells with IL-1 and bovine hyaluronidase. Additionally, LMW fragments of ~10kDa were also included in this study.

It is important to determine if using a unique RHAMM mimetic peptide, which has been shown to block signaling of HA fragments and reduce inflammation leading to a regenerative healing of skin wounds, works in synergy with HMW HA to prevent the development of OA. The current study investigates the effects of LMW HA and HMW HA on a synovial fibroblast cell line (SW982).

High concentrations of LMW HA were seen induce a catabolic effects on synovial fibroblasts. Additionally, TLR 2, an HA receptor on the SW982 cell line, showed high expression in the presence of IL-1. Importantly, the synovial fibroblast cell line showed a synergy between HMW HA (Orthovisc) and the peptide in vitro. This synergy reduced catabolic events, decreased inflammation and progression of cartilage degeneration of OA and interfered with the binding of LMW HA of synovial fluid. Additionally, HMW HA (Orthovisc) and the peptide together produce a protective effect when added to hyaluronidase. Treating patients with HMW HA (Orthrovisc) and the RHAMM mimetic peptide shows great promise in preventing OA and cartilage regeneration.

Shoshana Mond is a senior at Stern College for Women majoring in Biology. She did research at the NYU Hospital of Joint Disease in the summer of 2016.

Resistance to Tamoxifen in Breast Cancers with Hyper-Activation of mTORC1

Elana Perlow[1], Anya Alayev[1], Adi Berman[1], and Marina Holz[1,2]
[1]Department of Biology, Stern College for Women, Yeshiva University, NY, NY; [2]Department of Molecular Pharmacology, Albert Einstein College of Medicine, Bronx, NY

Over two-thirds of breast cancers are positive for the estrogen receptor ERα. Binding of estrogen to its receptor initiates a series of events that promote cell proliferation and growth. Normally, endocrine therapy is used to treat ERα positive breast cancers to inhibit estrogen activity. Tamoxifen, a commonly used anti-estrogen, is a competitive inhibitor of estrogen receptor.

Unfortunately, many patients develop resistance, either de novo or acquired, when treated with long-term endocrine therapy, which in some cases is due to hyperactivation of the mTOR (mechanistic target of rapamycin) pathway. The mTOR pathway circumvents the estrogenic activation of ERα and promotes cancerous cell proliferation.

By analyzing the mode of action of tamoxifen and the development of the resistance, new treatments can be developed and implemented. We generated tamoxifen resistant ER-positive MCF7 breast cancer cells by treatment with tamoxifen for two monthsr. A cytoplasmic and nuclear fractionation was performed to analyze the molecular changes associated with tamoxifen resistance. The results showed that expression and/or activation of proteins involved in the mTOR pathway increased in these cells. For example, an increase in levels of nuclear Raptor (regulatory associated protein of mTOR), which is critical in mTOR pathway activity, was found in these cells. This could promote estrogen independent ERα activity. These results show that tamoxifen resistance is associated with hyper activation of mTOR signaling in breast cancer cells.

Elana Perlow is a junior in Stern College for Women majoring in Biology. She is an editor of Derech HaTeva, Journal of Torah and Science and the Director of Mentorship of College EDge. In the coming years, Elana hopes to pursue an MD degree.

Investigating the Role of the CARD Domain of the Anti-Apoptotic Protein ARC in Chemotherapy Resistance

Jonathan Willner and Sumanta Goswami
Department of Biology, Yeshiva College, Yeshiva University, New York, NY

According to the American Cancer Society, approximately 90% of breast cancer deaths result from metastasis. Migratory cancer cell populations have previously been shown to express unique gene expression profiles and to exhibit resistance to standard chemotherapy treatments. The apoptosis and DNA repair pathways are commonly regarded as significant in the development of cancer. The Goswami Laboratory has previously shown that one of the proteins unregulated in migratory cancer cells, ARC, apoptosis repressor with caspase recruitment domain, is correlated with increases in the rate of DNA repair, single strand DNA repair proteins' mRNA expression, activity of select single strand DNA repair proteins, and resistance to apoptosis. Cells over-expressing ARC have been shown to express chemotherapy resistance both in vivo and in vitro.

In this study, the region of the ARC protein responsible for the results observed in previous studies is examined. Using flow cytometry and the single cell gel electrophoresis assay, it is shown that the observed correlation of ARC expression with increases in DNA repair rate and apoptosis resistance are mediated by the same protein domain, the CARD, or caspase recruitment domain. Further investigation is required to confirm that the CARD domain is responsible for the regulation of the single strand DNA repair pathway. Furthermore, additional study is required in order to evaluate the potential for synthesizing a small molecule inhibitor for the CARD domain of the ARC protein. Such a drug may combat the chemotherapy resistance observed in migratory cancer cells when combined with standard chemotherapy treatments, greatly enhancing the prognosis of hundreds of thousands of people diagnosed with breast cancer annually and decreasing the potential for relapse.

Jonathan Willner is currently in his fourth year at Yeshiva University majoring in Chemistry and Biology. After graduation he will attend the Albert Einstein College of Medicine.

Sleep Deficiency and Deprivation Leading to Cardiovascular Disease

Michelle Kohansieh[1] and Amgad Makaryus[2]
[1]Stern College for Women, Yeshiva University, New York, NY; [2]Department of Cardiology, North Shore-LIJ Health System, Hofstra North Shore-LIJ School of Medicine, Nassau University Medical Center, East Meadow, NY

Sleep plays a vital role in an individual's mental, emotional, and physiological well-being. Not only does sleep deficiency lead to neurological and psychological disorders, but also the literature has explored the adverse effects of sleep deficiency on the cardiovascular system. Decreased quantity and quality of sleep have been linked to cardiovascular disease (CVD) risk factors, such as hypertension, obesity, diabetes, and dyslipidemia. We explore the literature correlating primary sleep deficiency and deprivation as a cause for cardiovascular disease and cite endothelial dysfunction as a common underlying mechanism.

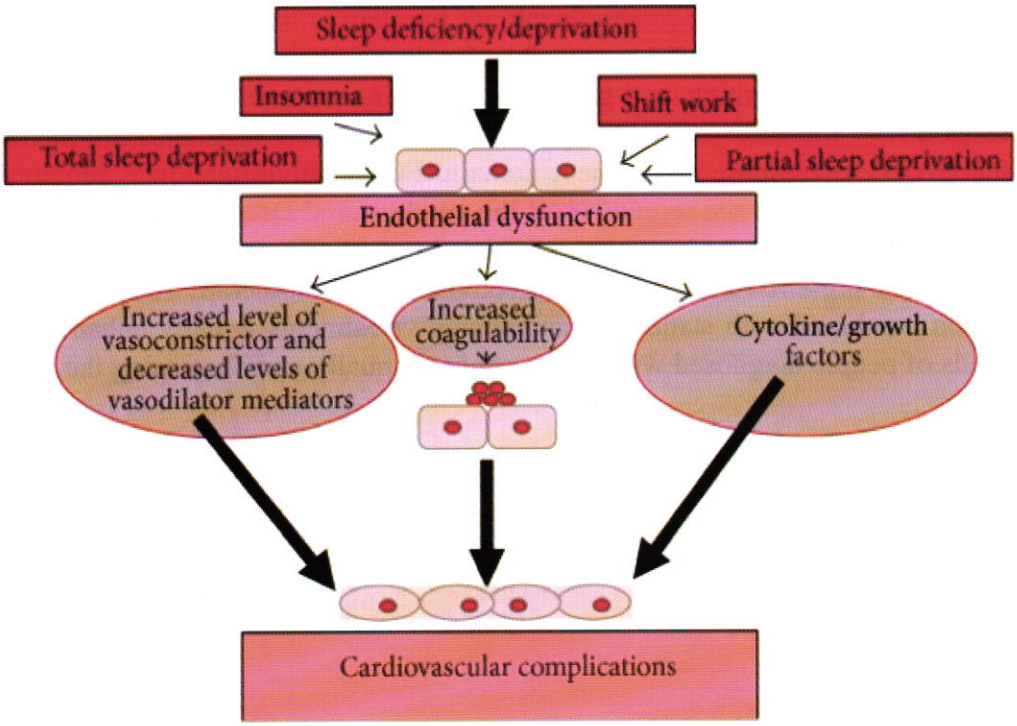


Figure 1. Mechanisms of endothelial dysfunction caused by sleep deprivation

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Michelle Kohansieh is a senior at Stern College for Women majoring in Biology. She currently has two publications on the PubMed database. She spends her free time volunteering for her local fire department as an EMT and she plans to pursue an MD degree in the coming years.

Implications of Gender Difference in Coronary Calcification as Assessed by CT Coronary Angiography

Amgad N Makaryus[1], Christina Sison[2], Michelle Kohansieh[3], and John N Makaryus[3]
[1]Department of Cardiology, NuHealth, Nassau University Medical Center, East Meadow, NY; [2]Biostatistics Unit, Feinstein Institute for Medical Research at the North Shore-LIJ Health System, Hofstra NSLIJ School of Medicine, Manhasset, NY; [3] North Shore-LIJ Health System, NSLIJ School of Medicine, Manhasset, NY

BACKGROUND: Arterial calcium as measured by 64-slice computed tomography coronary angiography (64-CT) is a reliable predictor of cardiovascular disease risk. Lipid-rich plaques with lower degrees of calcification may pose greater risk for adverse coronary events than more stabilized calcified plaques as a result of the increased risk of plaque rupture, migration, and subsequent acute coronary syndrome. We sought to examine coronary artery calcium scores as measured via 64-CT to assess the extent of calcification and plaque distribution in women compared to men.

METHODS: A total of 138 patients referred for 64-CT were evaluated. Computerized tomographic angiography was performed using the GE LightSpeed VCT. Subgroup analysis comparing male and female data (including demographic data) was performed. All major coronary arteries were analyzed for coronary stenosis/plaque characterization as well as total vessel calcium (Agatston) score quantification. Patient demographics and coronary risk factors were recorded.

RESULTS: Based on comparison of all total vessel calcium scores, males had a higher total mean calcium score than females in each individual vessel. The results were as follows for males versus females, respectively: left main total vessel calcium score 46.49 versus 16.71 ($P = 0.167$); left anterior descending 265.21 versus 109.6 ($P < 0.003$); left circumflex 130.5 versus 39.7 ($P < 0.004$); and right coronary 213.5 versus 73.8 ($P < 0.01$). The odds of having a total calcium score >100 (versus not) was 3.62 times greater in males relative to females, given that all the other cardiovascular risk factors are adjusted for (95% confidence interval: 1.37-9.54). On average, men had an average of 2.1 ± 1.5 epicardial vessels with a calcium score ≥ 11 compared to 1.3 ± 1.4 for women ($P < 0.005$).

CONCLUSION: There are clear differences between males and females regarding total vessel calcium scores and therefore risk of future adverse coronary events. Males tended to have higher average calcium scores in each coronary artery than females with a greater tendency to have multiple vessel involvement. Using this information, more large-scale, randomized controlled studies should be performed to correlate differences in the extent of coronary calcification with the observed variance in clinical presentation during coronary events between males and females as a means to potentially establish gender-specific therapeutic regimens.

Michelle Kohansieh is a senior at Stern College for Women majoring in Biology. She currently has two publications on the PubMed database. She spends her free time volunteering for her local fire department as an EMT and she plans to pursue an MD degree in the coming years.

Detection of Atrial Fibrillation in Ischemic Stroke Patients: A Systematic Review and Meta-Analysis of Secondary Stroke Prevention

Michelle Kohansieh
Stern College for Women, Yeshiva University, New York, NY

Evidence of atrial fibrillation (AF) is often sought in patients with ischemic stroke or transient ischemic attack (TIA), and confers a high risk of recurrent stroke. Guidelines for detection of AF following stroke vary. Therefore, we aimed to determine the frequency of newly detected AF by ambulatory cardiac monitoring through a systematic review and meta-analysis of the literature. We found a higher rate of AF detection by three modes of ambulatory cardiac monitoring – mobile cardiac outpatient telemetry, external loop reordering, and internal loop recording – when compared with rates of newly detected AF in inpatient bedside monitoring techniques. We suggest a systematic approach (Figure 1) that can be applied clinically to increase rates of AF detection, thus allowing for increased secondary stroke prevention through the use of anticoagulants.

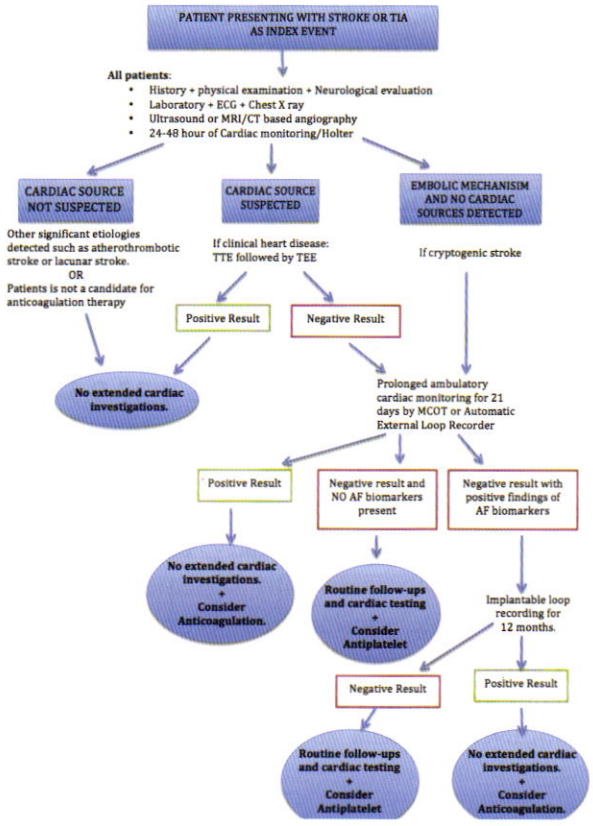

Figure 1. Systematic approach for detection of AF in stroke patients.

Michelle Kohansieh is a senior at Stern College for Women majoring in Biology. She currently has two publications on the PubMed database. She spends her free time volunteering for her local fire department as an EMT and she plans to pursue an MD degree in the coming years.

Determination of the Mitochondrial ATP Synthase Structure by Cryo-Electron Tomography

Nili Greenberg[1] and Zachary Freyberg[2]
[1]Stern College for Women, Yeshiva University, New York, NY; [2]Department of Psychiatry, Colombia University Medical Center, New York, NY

Although conventional electron microscopy (EM) has markedly improved our ability to resolve fine cellular features, is also carries significant limitations. Many methods may change or obscure important features within biological samples. To address this, our use of cutting edge cryo-electron microscopy (cryo-EM) and cryo-electron tomography (cryo-ET) approaches allowed us to avoid these potential experimental limitations and permitted visualization of cells in a near-native state. Instead of fixatives, living cells are plunge-frozen in liquid ethane, immediately preserving their native structures. The resulting images have sufficient contrast to produce incredibly clear and detailed images. The combination of these methods provided us with the capability to view a mammalian cell down to the level of single molecules and macromolecular complexes. Specifically, in cryo-ET, to determine the molecular structure of imaged objects, tomograms are first acquired from the samples. These are made up numerous individual images acquired at different angles relative to the sample, representing slices of the sample's z-plane. When pieced together, they provide a three-dimensional image of cellular organelles and their components. We applied these methods to the visualization of mitochondria within insulin-secreting pancreatic beta cells. In the process of our studies, we noticed small, regularly-spaced electron-dense structures on the membranes of the mitochondrial cristae. We hypothesized that these structures were mitochondrial ATP synthase dimers, which facilitate the production of ATP during oxidative phosphorylation. They are positioned on the cristae because their function requires a proton gradient, which exists between the matrix and the intermembrane space of a mitochondrion. Using a computer program, EMAN2, the coordinates of each of these individual structures were picked and recorded to facilitate the generation of an average structure, termed the sub-tomogram average. Sub-tomogram averaging is a computational device used to analyze the data for a tomogram, and essentially accounts and fills in information that is inherently missing from the three-dimensional structure. This gives a high resolution image of the structure that we are attempting to determine. If the structure of a molecule is already known from other methods, it can be used as a reference. To confirm whether the structure derived from the sub-tomogram average indeed conforms to a mammalian mitochondrial ATP synthase complex, we will compare our sub-tomogram average to the known ATP synthase complex structure which will be used as a reference. Ultimately, if the structure of these enzymes can be determined in healthy cells, similar approaches can be applied to cells carrying disease-causing mutations associated with these complexes. Such approaches open a new pathway for the study of mitochondrial diseases and facilitate development if fundamental new insights into the cause of such malfunctions on a structural level.

Nili Greenberg is a junior at Stern College. She is a Biochemistry major, and plans to pursue a career in biological research. She volunteers at Columbia University and is on the fencing team.

Cross-talk Between SUMOylation and Phosphorylation in Germ Cells

Elana Molcho[1], Xiao Yuxuan[1], Benjamin Lucas[1], and Margarita Vigodner[2]
[1]Department of Biology, Stern College for Women, Yeshiva University, New York, NY; [2]Department of Developmental and Molecular Biology, Albert Einstein College of Medicine, Bronx, NY

SUMOylation and phosphorylation are post-translational modifications that have been identified as important regulatory events that are implicated in several cellular processes.

The aim of this research was to study the cross-talk between SUMOylation and phosphorylation during the cell cycle, specifically by assessing the activity of various kinases known to be important for its regulation, including CDC2, ERK1 and 2, PLK1, AURKB, and AKT, upon inhibition of SUMOylation. SUMO conjugation was inhibited utilizing RNAi technology against the SUMO-conjugating enzyme, UBC9, (Figure 1) and we are currently generating a mouse model through which inhibition of SUMOylation in germ cells is achieved in vivo to be used for further research on this matter.

As shown in Figure 2, our data suggest that inhibition of SUMOylation decreased the inhibitory phosphorylation of CDC2, and increased the activating phosphorylation of ERK, AURKB, and AKT; therefore, their activity is normally inhibited by SUMOylation. In a different manner, inhibition of SUMOylation resulted in a slight decrease in the activating phosphorylation of PLK, hinting that its activity is activated by SUMOylation—an observation which was then confirmed in primary cells.

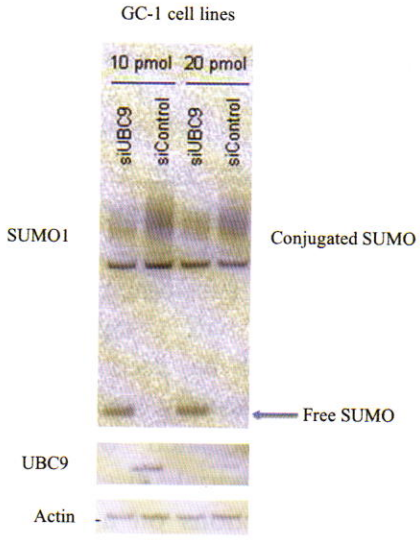

Figure 1

Figure 1. Anti-SUMO and anti-UBC9 western blotting confirmed the successful down-regulation of SUMOylation.

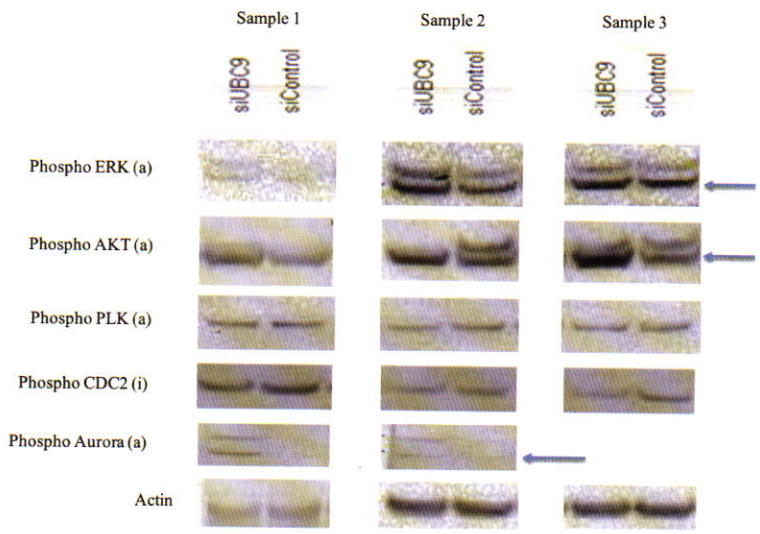

Figure 2

Figure 2. Running control and down-regulated samples on western blot revealed the effect of SUMOylation upon the phosphorylation of various kinases of interest.

Elana Molcho is a senior studying Biology at Stern College for Women. She founded and currently runs the Children's Preventative Care Foundation—a non-profit organization dedicated to teaching children living in marginalized communities about preventative health care techniques. She plans to pursue an MD in the coming years.

Leucine-Rich Repeat Kinase (LRRK2) Links Cellular Stress-Responses to KRAS Proto-Oncogene Expression in Lung Cancer

Jeffrey Sebrow[1], Subrata Manna[1], Michael Bouaziz[1], Gabriel Sturm[1], Jonathan Aivazi[1], and Yakov Peter[1,2]
[1]Department of Biology, Yeshiva College, Yeshiva University, New York, NY; [2]Department of Pulmonary Medicine, Albert Einstein College of Medicine, New York, NY

Lung cancer remains the number one cause of cancer-related deaths worldwide with an estimated 220,000 new cases and 150,000 deaths in 2015. Several recent studies suggest a link between lung cancer, cigarette smoking, and Parkinson's disease (PD). In one study the multifunctional leucine-rich repeat kinase (LRRK2/PARK8) gene, the most common genetic cause of PD, was shown to be overexpressed in a considerable portion of non-small cell lung cancer (NSCLC) tumors. With an established role in cell macroautophagy, the manner by which LRRK2, a large ~286 kDa protein, can affect NSCLC cell growth remains unknown. In this study, we stably overexpressed the human LRRK2 (WT) and variants (G2019S, N2081D, N551K, and a double N2081/N551 mutation) involved in disease, into the A549 NSCLC cell line. We investigated changes in viability, growth, and gene expression in untreated, serum starved (18 hours), and cigarette smoke extract (CSE; 48 hours; 10%) exposed cells. RT-PCR data demonstrated over a 39-fold (n=4) increase in LRRK2 transcript levels in the transfected A549 cells. However, like controls (empty vector), LRRK2 and its variants could be post-transcriptionally degraded, as seen by Western blot. Performing cell cycle analysis with propidium iodide, overexpression of WT and N551K ($P \leq 0.03$; $n \geq 5$), but not N2081D or the N2081/N551 mutations, enhanced cell proliferation increasing the percentage of A549 cells in the G2/M phase by 22.1±6.0%. The percentage of apoptotic cells was not significantly affected in the untreated clones. Full-length LRRK2 protein levels were stabilized in serum-starved cells, to suggest an important role for this protein in A549 stress-mediation. Serum starvation specifically reduced cell apoptosis in N2081D and N551K single mutations, but not in control, WT, or double N2081/N551 clones. In these cells, levels of KRAS expression were increased over 40% relative to control ($P=0.01$; $n \geq 3$). In contrast, while not affecting control and N551K expressing clones, CSE exposure significantly reduced levels of KRAS transcript by over 44% in WT, 2081 and N2081/N551 double mutant cell types. These data suggest that LRRK2 activation may indirectly signal KRAS proto-oncogene expression during cellular stress-mediated autophagy playing a major role in lung cancer cell proliferation and progression.

Jeffrey Sebrow is a recent Yeshiva College graduate where he majored in Biology. He is currently managing Dr. Peter's laboratory at Yeshiva College and will be applying to medical school this coming summer.

Jonathan Aivazi is a Biology major who recently graduated from Yeshiva College. He will be applying to medical schools this summer.

Investigating the Link Between Variability in Gene Expression and Protein Abundance in Ovarian Cancer Patients

Miriam Pearl Klahr, Samuel Zimmerman, Laurence de Torrenté, and Jessica Mar
Department of Computational Biology, Albert Einstein College of Medicine, Bronx, NY

While we understand that genes are up-regulated and down-regulated in a cellular phenotype, we are just beginning to recognize that variability in gene expression also has functional consequences. Limited research exists on the relationship between expression variability and average gene expression. There is also a lack of consensus regarding the best statistic to use when studying gene expression variability. Using many transcriptome-wide data sets from different microarray and RNA sequencing [RNA-seq] studies, conducted on both single cell and bulk tissue samples for different cell types, we investigated the nature of how average expression and expression variability are linked across the genome. We also evaluated the performance of three common variability estimators, standard deviation [SD], median absolute deviation and coefficient of variation. The evaluation was based on the degree of correlation between the average expression and expression variability. Our results collectively point to SD being the most stable estimator to use.

Using this information, we conducted an analysis of gene expression variability and protein abundance variability using data on 174 ovarian cancer patients from the Cancer Genome Atlas. We applied an F-test to identify genes that had significantly higher levels of variability at the transcriptional level than the protein abundance level, and vice versa. Additionally, we also identified a set of genes that had equal variability in both protein abundance and gene expression. We looked for enrichment of different pathways that was exclusive to each of these three sets of genes in attempt to understand the biological consequences of this variability. We used the NCI Pathway Database for this purpose, as well as other annotation sources, such as MSigDB. We discovered that many of the pathways among the three groups overlapped, meaning that within the same pathway we observed different levels of variability among different genes. This led us to hypothesize that perhaps certain genes are more or less critical to the pathway in that they are expressed at the protein level or transcript level, with greater or less variability in the ovarian cancer patients. This result may even suggest that there are different points of control in pathways that are used with greater consistency. Finding which genes and proteins these points of control correspond to may identify new targets for manipulating tumors.

Miriam Pearl Klahr is a second year student at Stern College for Women majoring in Physical Sciences and minoring in English and Judaic Studies. When not busy with school work, she enjoys volunteering as a College Edge mentor, reading and learning, playing piano, going on long walks, and spending time with friends and family.

Difference in Gene Expression in Parental and Transformed Epithelial Cells

Emily Chase, Michal Schwartz, and Ofir Hakim
Department of Life Sciences, Bar Ilan University, Ramat Gan, Israel

Altered gene expression is one of the major causes of carcinogenesis. Expression is commonly controlled by the binding of transcription factors to regulatory sites, which are likely to be found a few hundred kilobases around the gene. The goal of this study was to find transcription factors relevant to the altered gene expression by comparing changes in gene expression with changes in the activity of regulatory regions found in close proximity to the genes of interest. To accomplish this, we first had to quantitate the changes in gene expression. We performed RNA-seq on mammary epithelial cells transformed with a HRAS oncogene and on the parental cells in order to measure the difference in RNA levels. To validate the RNA-seq results, we picked a number of differentially expressed genes and tested them in real-time PCR. Using exon-exon primers to measure total mature RNA, we found that the fold change calculated from the RNA-seq correlated with the data from real-time PCR. The results from both data sets indicate that the genes CDH1, FN1, and LAMC2 are repressed and the genes IL6, PPARG, FGF2, and EGLN3 are activated in HRAS-transformed mammary epithelial cells when compared with normal mammary epithelial cells. Samples were run in electrophoresis gel following real-time PCR and the results confirmed the identities of the amplified DNA sequences as the intended genes. Biological repeats were performed and results were consistent with previous data. Using intron-exon primers with real-time PCR to measure nascent RNA, we found that nascent RNA levels correlate with changes in mature RNA levels determined in RNA-seq. This shows that most of the differences we see in total mature RNA levels between HRAS-transformed and normal mammary epithelial cells are due to transcriptional changes. The validation of the alteration in gene expression after transformation will be used to compare to changes in the activity of regulatory sites in order to identify regulatory sites that are associated with differentially expressed genes. From this data, we aim to determine transcription factors relevant to the changes in gene expression during cancerous transformation.

Emily Chase is a senior at Stern College for Women majoring in Biochemistry. Next year she plans to attend Albert Einstein College of Medicine.

MEIG1 Effect on B-Lymphocyte Development

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Meiosis, the fundamental evolutionarily conserved differentiative process by which haploid gametes are produced, is complex and tightly regulated. Of the many number of genes that are expressed during mammalian gametogenesis, the murine meiosis expressed gene 1 (MEIG1), was identified as a key component in meiosis, abundantly expressed in both male and female mice. The MEIG1 protein found in mice is also evolutionarily conserved in humans; the MEIG1 gene found in humans is eighty seven percent identical to the MEIG1 gene found in mice.

MEIG1 gene was also found to play a role in the function of the lymphatic system, specifically within the maturation of the B-cell lymphocytes. B-cell lymphocytes mature within bone marrow. They undergo a DNA rearrangement process, VDJ recombination, and display an ordered expression of genes and cell surface markers. To examine the possible involvement of MEIG1 in B-lymphocyte development, bone marrow cells of wild type and knockout mice without the MEIG1 protein were compared.

To follow the effect of MEIG1's presence during lymphocytic maturation, B-lymphocytes were separated using antibodies against various markers. Using fluorescence-activated cell sorting (FACS), B-lymphocytes were first separated from bone marrow cells using fluorescently labeled antibodies against CD19 marker. To differentiate between Pre-B cells from immature B cells, CD25 and IgM markers were used to detect the stage in development. If both markers were negative, VDJ recombination did not occur and these lymphocytes were still in the Pro-B stage of development. If CD25 was present but IgM was absent, the lymphocytes were Pre-B cells, but if CD25 was deficient and IgM was present, the B-lymphocytes had passed the BCR checkpoint and the lymphocytes present were more differentiated immature B cells. To observe the effects of the MEIG1 protein in the development of B-lymphocytes, the percentages of lymphocytes in the KO and WT mice present in each stage were compared using the markers mentioned above.

Despite the evidence that suggests the involvement of MEIG1 in DNA rearrangement processes, the results collected from FACS did not show any significant changes in the percent of lymphocytes in the subpopulations characterizing the differentiated stages of B cells during the two trials of this experiment.

Akiva Abramowitz is a senior in Yeshiva College majoring in Economics with a minor in Biology. Akiva will be applying to MD programs this summer.

Influence of Exercise-Related Energy Metabolites on Neurogenesis

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Adult neurogenesis is the process through which stem cells and progenitor cells proliferate, differentiate, migrate, and integrate into an existing neural network. Until recently, it was accepted that neurogenesis only occurred during pre-natal development and ceased once born. Although this holds true for the bulk of neural pathways, recent research has shown that adult mammalian neurogenesis occurs primarily in the subventricular zone and the hippocampus. The subgranular zone of the dentate gyrus, a portion of the hippocampus, is exceptionally notable for contributing to adult neurogenesis, with thousands of cells produced daily to initiate the neurogenesis process. However, very few cells actually reach the mature neuron state, and they depend on factors such as sleep, stress, age and exercise, which determine how conducive the microenvironment is for neurogenesis at any given time.

Aerobic exercise in particular has been shown to have a positive effect on hippocampal neurogenesis. This can be explained by the production of various metabolites that are byproducts of aerobic and anaerobic glucose metabolism. The proliferation of neural stem cells and neural progenitor cells are heavily affected by the availability of energetic sources in the brain. The aim of this experiment is to examine the relationship between an increase in exercise-related energy sources and neurogenesis. To achieve this, in-vivo research was conducted, in which the test group of mice was treated with energetic metabolites. After, a process of immunostaining, image acquisition, and stereological analysis was conducted to quantify neurogenesis. While the lab is presently in the process of stereological analysis, early results look promising. Current research is focusing on investigating the molecular basis of neurogenesis, which is the neuronal involvement in learning and memory, and in neurodegenerative disorders such as Alzheimer's Disease and Parkinson's Disease, as well as studying the recovery from trauma and stroke.

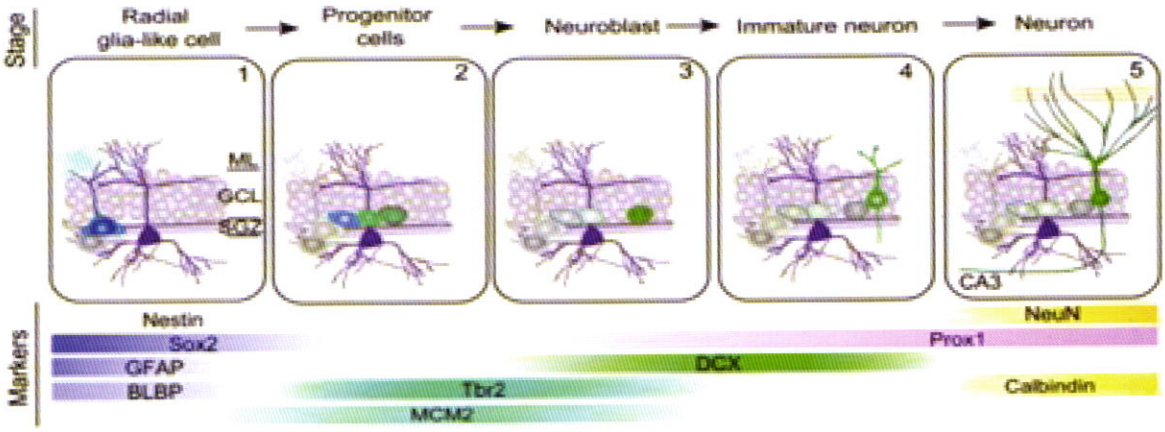

Figure 1. Schematic of neurogenesis processes in adult hippocampus with markers used for immunostaining at each respective stage.

Elizabeth Bitterman is a senior at Stern College for Women currently working towards her B.A. in Biochemistry and studying computer science. She enjoys being part of the research process and has recently been involved in neurogenesis and nutraceutical cancer research. In future years, Elizabeth plans to earn her M.D. and practice as a physician.

Age and Alzheimer's Disease Gene Expression Profiles Reversed by the Glutamate Modulator Riluzole

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Alzheimer's disease (AD) and age-related cognitive decline represent a growing health burden and involve the hippocampus, a vulnerable brain region implicated in learning and memory. To understand the molecular effects of aging on the hippocampus, this study characterized the gene expression changes associated with aging in rodents using RNA-sequencing (RNA-seq). The glutamate modulator, riluzole, which was recently shown to improve memory performance in aged rats, prevented many of the hippocampal age-related gene expression changes. A comparison of the effects of riluzole in rats against human AD data sets revealed that many of the gene changes in AD are reversed by riluzole. Expression changes identified by RNA-Seq were validated by qRT-PCR open arrays. Riluzole is known to increase the glutamate transporter EAAT2's ability to scavenge excess glutamate, regulating synaptic transmission. RNA-seq and immunohistochemistry confirmed an increase in EAAT2 expression in hippocampus, identifying a possible mechanism underlying the improved memory function after riluzole treatment.

Rina Leah Davidson is a senior at Stern College for Women, majoring in neurobiology. She did research at the Rockefeller University for a year and a half, and is planning on working at Columbia University over the summer.

The Effect of Sodium Orthovanadate on Neuronal Homeostasis in Experimental Cerebral Malaria

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Department of Pathology and Medicine (Infectious Diseases), Albert Einstein College of Medicine, Bronx, NY

Cerebral malaria (CM) is a neurological complication of infection with Plasmodium species, and accounts for approximately 1,000,000 deaths annually. One of the notable effects of CM is perturbation of the Blood Brain Barrier due to damage to the cerebrovasculature by lethal substances. CM has been shown to trigger alterations in the Akt cell survival signaling pathway and to induce profound alterations in the regulation of neuronal function and survival. It was hypothesized that Sodium Orthovanadate (NaOV) would indirectly phosphorylates Akt. This would increase its activity and result in the inhibition of the phosphorylation of tau, a protein vital in neurofibrillary tangle formation and neuronal degeneration. If tau phosphorylation is inhibited, cerebral function should return to normalcy. Mice with CM were treated either with NaOV or NaCl (as control). Behavioral testing was performed to see whether NaOV improved cognitive function. Western blot analysis was conducted to determine the specific signaling induced by NaOV. There was a difference in Akt levels for all groups, but no difference in Tau. PbA-infected mice had obvious declines in cognitive function, and although there was no significant improvement with NaOV treatment. This inconclusive data reveals the need for further testing of this compound. Many trends were observed with treatments that were close to significance. This study should be repeated with a larger sample, and further experiments examining transcription of Akt and Akt enzyme activity during malaria should be performed.

Joshua Rabanipour is a sophomore at Yeshiva College majoring in Jewish Studies with a minor in Biology. In his spare time, he volunteers for New York NCSY, Grace Plaza Nursing Home and Masbia Soup Kitchen. When not volunteering, he can be found in the Beit Midrash deliberating and reviewing medieval rabbinic manuscripts commenting on the Talmud.

Radioimmunotherapy with ^{225}Actinium Shows Promise

Michael Shavolian, Abdullah Norain, Ruth Bryan, Zewei Jiang, Ekaterina Revskaya, and Eka-terina Dadachova
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Recent biomedical research has demonstrated the newly discovered efficacy of α-emitting radioim-munotherapy (RIT) in targeting leukemias, which exhibit more radiosensitivity than their solid counterparts. In particular, RIT using ^{225}Ac has shown potential as an alternative to full-body mye-loblative radiation as a conditioning regimen prior to hematopoietic stem cell transplantation. CD45 is a pan-leukocytic antigen widely expressed to the measure of more than 10^5 sites per cell to which BC8, an immunoglobulin G monoclonal antibody, binds effectively. BC8 monoclonal antibody can be radiolabeled with ^{225}actinium using chelating agent DOTA in order to deliver theranostic radia-tion. In this experiment we aimed to evaluate the efficacy of conjugation, labeling and immunoreac-tivity as well as biodistribution of the BC8-DOTA-^{225}Ac complex.

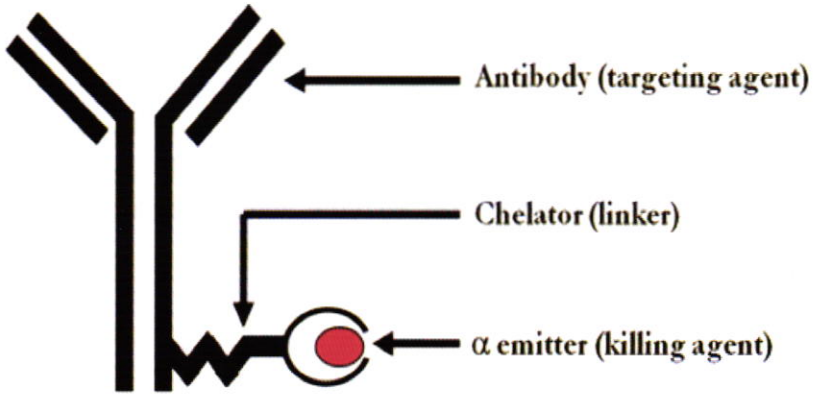

Figure 1: Schematic of BC8 MAb-DOTA-^{225}Ac Antibody

Both BC8 and 18B7, a control which binds to a polysaccharide in C. neoforman, were conjugated and labeled. We were able to achieve nearly 50% labeling efficacy, as demonstrated by High Per-formance Liquid Chromatography, and corroborated by flow cytometry showing that conjugated BC8 results in a significantly lesser degree of immunoreactivity than BC8 alone. While 18B7 ex-hibits negligible median fluorescence intensity, an immunoreactivity assay for the same MAb yielded a percent bound value of 19.4% of the labeled MAb. These apparently confounding results may be due to interactions between the secondary antibody and the DOTA-MAb complex. Instant Thin Layer Chromatography yielded a final purity of 78% for the sample utilized in the biodistribu-tion in healthy murine models at five different time points post injection.

We conclude that the absence of cancer in the healthy murine models may have led to a higher radi-oactivity biodistribution in the 11 tested organs. Additionally, the immunoreactivity of the radio-labeled MAb tested beforehand was below the ideal 90%. Lastly, this experiment allows the future calculation of effective dose per organ to assess the maximum theranostic dose of the treatment complex.

Michael lives in Great Neck, NY and studies biology at Yeshiva College. He enjoys exploring his interest in business and founded the Entrepreneurship and Biotechnology Club. This past summer he interned at a medical device start up and conducted research at the Albert Einstein College of Medicine. He hopes to affect policy and change from within the field of healthcare.

Stabilization of the eEF-2K Kinase Domain through Mutagenesis and Domain Interactions

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Department of Chemistry, City College of New York, New York, NY

Eukaryotic elongation factor 2 kinase (eEF-2K) is a non-canonical Ser/Thr kinase that phosphorylates elongation factor 2 (eEF-2), reducing its affinity for the ribosome and resulting in the decrease in global protein synthesis. eEF-2K activity has been associated with poor prognosis in certain types of cancer in addition to several neurological and inflammatory diseases. In spite of its importance, detailed structure/function analyses of the catalytic domain of eukaryotic elongation factor 2 kinase (eEF-2K) located on its N-terminus have been hindered by its tendency to aggregate at concentrations necessary for biophysical analyses. We are using two independent approaches to stabilize the kinase domain and render it soluble, monodisperse and suitable for structural analyses. The first is a mutational approach using both direct and random mutations. We have identified residues conserved across the crystal structures of related kinase domains but not conserved in eEF-2K and rationally mutated these residues to generate constructs with reduced ability to aggregate at high concentration. In addition, we have also used random mutagenesis to generate unbiased mutations and screened for solubility using an expression plasmid that encodes a C-terminal green florescent protein (GFP). The second approach is based on the observation that the kinase domain is stable in complex with various C-terminal fragments. We are optimizing these fragments to balance stability and overall size of the complex to allow their efficient characterization using biophysical/structural approaches.

We expect that using these two complementary approaches we will be able to generate constructs that are suitable for NMR and/or crystallographic analyses.

Isaac Snyder is a senior at Yeshiva College majoring in Biology with a strong interest in researching IBD. During his free time, he can be found reading IBD papers, cooking or something TEACH related.

Copper Promoted Aromatic Acyloxylation: The Formation of Aryl Esters through the Cross Coupling of Aryl Halides via Comproportionation of Copper II Reactants

Adam Goldstein, Steven Kohane, and Fabiola Barrios-Landeros
Department of Chemistry, Yeshiva College, Yeshiva University, New York, NY

Previous research has shown that Cu (I) complexes can successfully drive aromatic cross coupling reactions. New entry points for copper complexes were studied, with a particular focus on utilizing the ability of Cu (II) to comproportionate, as Cu (II) complexes are often cheaper and more stable. It was shown that Cu (II) acetate does, in fact, drive the copper- promoted acetooxylation of phenyl iodide in DMF following the proposed mechanism (Figure 1). Product development, as measured by gas chromatography, showed yields of 90%. Furthermore, the change in color of the copper as it transitions between the various oxidative states during the reaction allowed for an in- depth analysis of the rate of reaction, via flow cell spectroscopy. Additionally, Cu (II) sulfate, an even cheaper alternative, was able to drive the reaction in the presence of acetate salts, though with lower yields. Phase transfer agents, such as tetraflouroborate and hexaflourophosphate, increased the yield of the Cu (II) sulfate reactions. Ultimately, it was shown that the comproportionation of Cu (II) is a viable option for adding Cu (I) complexes into cross coupling reactions.

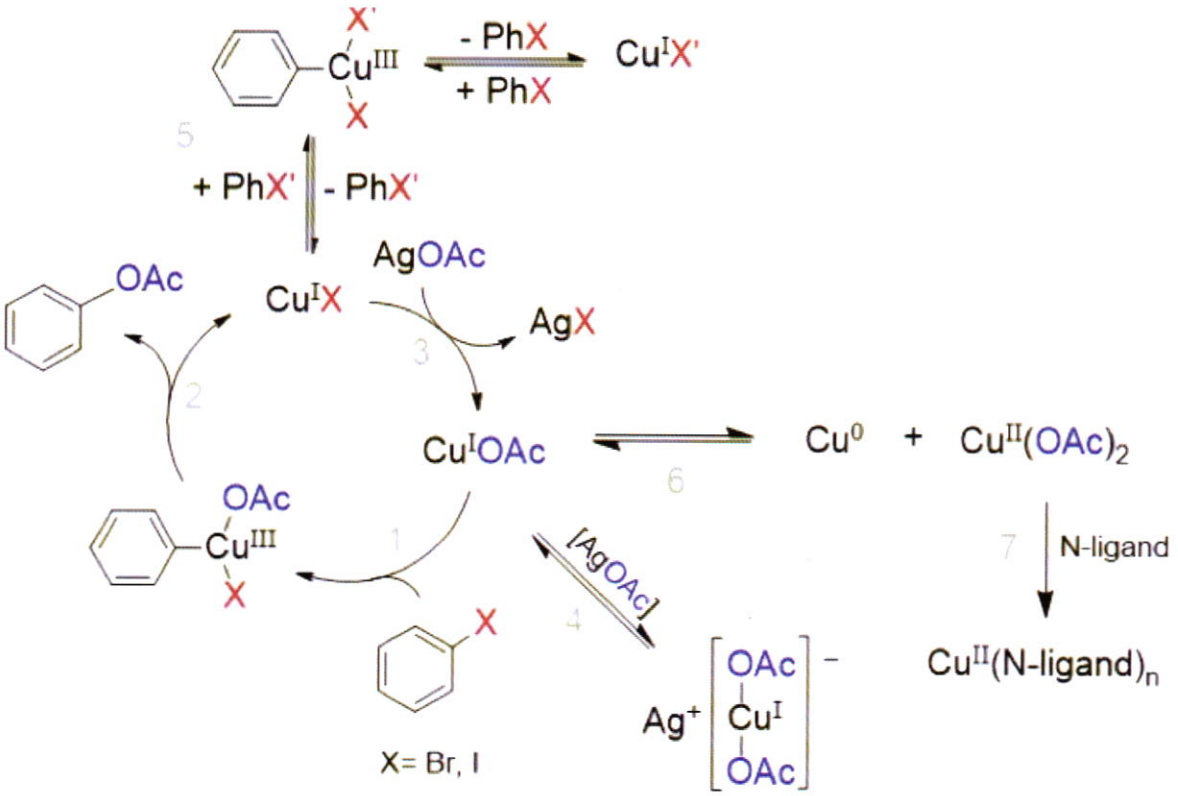

Figure 1: Proposed Mechanism for the Formation of Aryl Esters

Adam has recently graduated from Yeshiva College. While there, he completed a major in Mathematics. He will be attending medical school next year.

Protein TMEM16A: Opening Ion Channels for Cystic Fibrosis

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Novartis Institutes for Biomedical Research, Cambridge, MA

Cystic Fibrosis [CF] is the most common lethal genetic disease of Caucasians. It affects 1 out of 2500 live births and there are around 70,000 patients worldwide today. CF is systemic disease, which affects the cells that produce sweat, mucus and digestive enzymes in the entire body. In CF patients, these fluids become thick and clog the passageways in the body, which then triggers inflammatory responses. Lung pathogenesis is usually responsible for the death of the patient.

Cystic Fibrosis is a genetic disease caused by mutations in the gene encoding for the Cystic Fibrosis Transmembrane conductance Regulator [CFTR]. The CFTR protein channel plays a key role in maintaining the homeostasis of the airway surface liquid layer in the lungs. There is a fine balance between Cl^- secretion through the CFTR and Na^+ absorption through the Epithelial Sodium Channel, which controls the thickness of the periciliary fluid. Due to the dysfunction of the CFTR, Na^+ absorption prevails over Cl^- secretion. This imbalance dehydrates the mucus of the airway surface thereby impairing ciliary beating. Immobilized mucus then becomes a niche for bacterial survival and proliferation. Protein TMEM16A is an alternate calcium-activated pathway that needs to be potentiated in order to ideally replace the defective Cl^- secretion in the CF lungs. The proposed therapy would be an inhaled calcium-activated Cl^- channel opener, which would hydrate the airways, thus improving lung functions. It would also promote mucociliary clearance and reduce small airway occlusions and risks of repeated exacerbations.

CEN466 is a molecule found by HTS with modest capacities for activating TMEM16A. The challenge of the research is to improve the physical and chemical properties, and the activity of CEN466 by modifying the functional groups on its extremities. Multiple boronic acids were used to perform Suzuki reactions, followed by HATU couplings. The products from these reactions were then tested. The results of the Q-patch assay, which measures the reactivity of the molecules for potentiating TMEM16A, and of the Solubility and Rat Clearance assays, were too low. Therefore, these compounds were not advanced to further testing. However, other modifications of CEN466 through different chemical reactions were conducted and showed satisfying results from the same assays. These products are currently tested through more sophisticated pharmacological experiments. In the end, these outcomes helped us refine the possibilities of modification of CEN466 and demonstrated that solubility, potentiating capacities and metabolic clearance of the molecule can be improved by modifying the CEN466 scaffold.

Ayala Ouanounou is a junior at Stern College for Women of Yeshiva University, expecting to graduate in May 2017 with a B.A in Biochemistry.

A New Method of Correcting Error in Mass Spectrometry Metabolomics Data

Jordan Green[1], Irwin Kurland[1], and Van Kelly[2]
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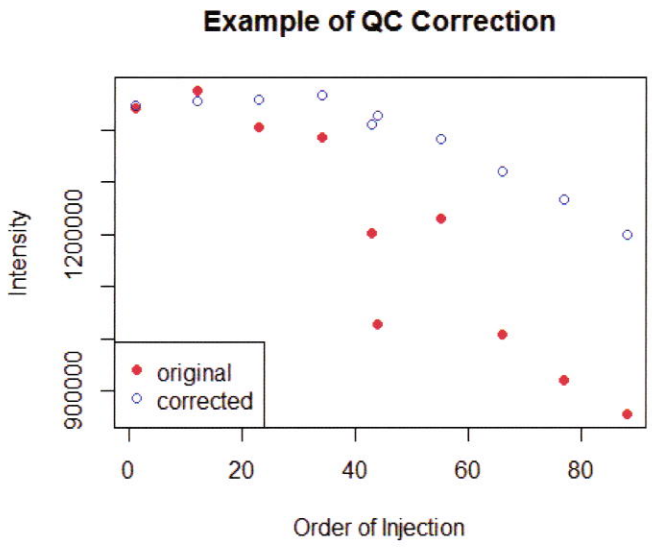
Metabolite intensity values are measured by extracting metabolites from plasma, urine, or tissue samples, and evaluating the spectra produced after injection into a gas chromatography/mass spectrometer (GC/MS) or a liquid chromatography/mass spectrometer (LC/MS) system. However, a number of outside factors during this process can negatively affect the end results. For example, the column for metabolite separation can become contaminated with metabolites that do not elute from the column, causing degrade in performance and introducing drift into the readings from continued usage.

To compensate for these factors, a set of identical quality control samples (QCs) are created by mixing together a small amount of each individual sample from a sample set, and one of these QCs is injected in between groups of samples at periodic intervals. The changes in their intensity values can be used to measure the amount of drift that has occurred in the samples themselves, in order to calculate a correction factor and produce more accurate data.

A previous study by Zhao et al. offers one such method for correcting gross error in GC/MS QC intensity values and using the resulting corrected values to correct systematic error in and normalize sample data. We believe that it is worth improving on this algorithm because of the extent to which correcting sample data depends on the intensity values of the QCs, and to this end we present an additional method of gross error correction for both GC/MS and LC/MS data.



We first correct QCs by observing the ratios between adjacent pairs of QCs and noting that since they are identical, these ratios should be close to 1. We then calculate linear regressions between n adjacent QCs to further adjust them. We subsequently measure improvement by counting the amount of ratios within a 5% deviation from 1, and the amount of relative standard deviations for all QCs in each given metabolite vector below .2, and confirm that our method causes these numbers to increase.

Figure 1. A set of QCs before (red) and after (blue) correction

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Jordan Green is a graduating senior at Yeshiva College majoring in computer science and mathematics. He plans to work in the technology industry in the near future.

Preferential Attachment and Real Models of Power Grids

Russell Spiewak[1], Yakir Forman[1], Sergey Buldyrev[1], Saleh Sultan[2], and Gil Zussman[2]
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We develop a preferential attachment based Degree and Distance Attachment (DADA) model of a grid and compare it to the US Western Interconnection (USWI). Using the DC power flows approximation, we find that simulations of our model and simulations of the USWI have similar distributions of degrees, resistances, and currents. We also investigate the behavior of both grids resulting from the failure of a single line. We find that our model and the USWI react very similarly to that failure. In many cases, failure of a single line can cause a cascade of failures which impacts the entire grid. We characterize the resilience of the lines by tolerance α, which is the ratio of the maximal load a line can carry to its initial load. We find that for many values of $\alpha < 2$ initial breaking of a single line can result with a high probability in a cascade of failures leading to a massive blackout with final yield less than 80% of the initial consumed power. The yield has a bimodal distribution, typical for a first-order transition, i.e. the failure of a single line leads either to an insignificant power reduction, or to a major blackout. In those cases in which a blackout occurs, we measure the propagation of the blackout across the grid as the cascade progresses. We find that there is a latent period in the cascade during which a few lines are overloaded, and yield remains high. The duration of the latent period is proportional to tolerance. The existence of the latent period suggests that intervention at the early stages of the cascade can significantly reduce the risk of a major blackout.

Russell Spiewak is currently a senior at Yeshiva College. He is a member of the honors program, a student research assistant, a peer tutor and an Eagle Scout. Russell is also currently in the YU RIETS Semikhah program and is considering participating in the YU Graduate Programs in Mathematics before furthering his education with graduate studies in physics.

Yakir Forman is finishing his third year at Yeshiva University. He plans on majoring in math and physics. In the past, Yakir has participated in START Science and has been a student chemistry research assistant. Yakir is also currently in the RIETS Semikhah program. After YU, Yakir hopes to continue his education in a graduate program in either math or physics.

ISC in MEG in Response to Engaging Videos

Joshua Blau, Abraham Goldstein, and Yuval Harpaz
Electromagnetic Brain Imaging Unit, Gonda Multidisciplinary Research Center, Bar-Ilan University, Ramat Gan, Israel

Magnetoencephalography (MEG) is a brain imaging technique that measures brain activity via magnetic fields perpendicular to the surface of the head. Because its data has much higher temporal and spatial resolution than other brain imaging techniques such as functional magnetic resonance imaging (fMRI), it is useful for studying specific frequencies and sections of the brain that may be undetectable using other techniques. Such results are important for cognitive psychophysiological studies, the main focus of our lab.

Functional magnetic resonance imaging (fMRI) studies have found large degrees of inter-subject correlation (ISC) for subjects watching movies in which they were emotionally engaged. However, fMRI is severely limited by the timescale of its hemodynamic responses. MEG, which instead uses neurophysiological imaging, measures brain activity directly, per millisecond over 248 channels, and can pick up much higher frequency signals. Using frequency analysis, this data can be separated into frequency bands, which can then be analyzed individually. For this reason, MEG serves as an excellent platform for testing and narrowing the conclusions of such fMRI studies.

We used short videos (around two and a half minutes) of charismatic speaking and non-charismatic speaking to approximate the effects of the emotional/psychological interest piqued by watching movies, and to serve as stimuli for measuring brain activity with MEG. By analyzing the data in MATLAB, we measured subject correlations in brain activity in alpha, beta, and gamma frequencies, particularly in the surface areas of the brain more relevant to specific frequency bands. After comparing both averaged and individual Fourier transformed time courses of MEG data against the audio track of the videos, it was clear that the charismatic video had a significant effect on ISC, thus corroborating the fMRI findings.

Joshua Blau is a junior at Yeshiva University double majoring in mathematics and computer science. His interests vary, and he participates in and occupies leadership positions at many university clubs in addition to working at the Wilf Campus Writing Center. He plans on pursuing a PhD after graduation.

Self-Disclosure of Positive Emotions in Close Relationships of Socially Anxious Individuals

Noa Choder, Elisheva Jakobov, and Eva-Gilboa Schechtman
Department of Psychology, Bar Ilan University, Ramat Gan, Israel

Social anxiety disorder, or social phobia, consists of experiencing excessive fear when placed in social and/or performance based situations. Socially anxious individuals tend to have fewer close relationships compared to non-socially anxious individuals because of their heightened fear of being judged or misrepresenting their true selves. Therefore, they tend to overly rely on these few close relationships, yet even within these friendships, they appear to disclose less information regarding their thoughts, emotions, and experiences, thereby decreasing the benefits of intimacy and support that they can potentially be receiving from their peers.

The goal of the study was to analyze patterns of self-disclosure focusing on positive experiences and emotions in close relationships of socially anxious individuals. Participants wrote about a positive experience they had within the past month. Prior to each experiment, each participant identified one individual with whom they felt close to, and rated how close they felt to that individual on a scale from 1-5 (not close – very close). Two studies were conducted: Study 1 (n=292) focused on the association between social anxiety and the tendency to disclose positive emotions and experiences. Participants composed narratives describing experiences of pride, joy, and closeness, first as if they were writing to a stranger, and after, they were asked to depict the same exact emotional experience to the person whom they previously designated as their close friend. Study 2 (n=226) focused on experiences of closeness and joy, and sought to determine whether patterns of self-disclosure are malleable in socially anxious individuals, and whether an increase in self-disclosure is associated with an increase of positive affect in socially anxious individuals. Narratives from both studies were then coded for words related to emotions and words indicating specificity of experience, including time, location, and duration. Phase one entailed determining coding criteria and training the coder in applying those criteria to judgments of the narratives. In phase two, the coding was completed and recorded. In the future, additional raters will code the data and interrater reliability will be assessed.

Although analysis of the data is at the very beginning stages, current results indicate that socially anxious individuals tend to use fewer emotional words in comparison to socially non-anxious individuals. Additionally, the analysis of specificity indicates that socially anxious males do not describe their experience in a specific manner. However, with females, this effect was not found. By better understanding patterns of self-disclosure in socially anxious individuals' close relationships, possible therapeutic interventions can assist these individuals in establishing stronger, closer, and more intimate relationships.

Elisheva Jakobov is a senior at majoring in psychology at Stern College for Women. Elisheva researches in a neuropsychology clinic at The New York State Psychiatric Institute during the week. She hopes to pursue a career working with underprivileged and at-risk populations.

He's Just Not That Into You: The Effects of Rejection in Close Relationships

Joshua Nagel and Jennifer Isaacs
Department of Psychology, Yeshiva College, Yeshiva University, New York, NY

This study investigates the cognitive and emotional effects when individuals are rejected by close friends. Although the effects of rejection have been studied extensively in the laboratory between strangers or new acquaintances, this study is one of the first to explore rejection within the context of adult friendships. In experiment 1, participants read a scenario in which they visualize themselves ostracized by a close friend or an acquaintance. Participants were then measured on the four primary needs and affect. In experiment 2, intimate and non-intimate relationships were artificially created through the Fast Friends exercise. Participants then either experienced rejection when their partner supposedly did not wish to continue working with them or were informed the study could not continue because their partner had to leave early. Participants then filled out self-evaluation measures on their ability to create friendships and their general affect. Both experiments failed to find significant results to support the hypothesis that rejection by a close friend hurts more than by an acquaintance. The lack of significant finding may be partially due to the experimental manipulation being too weak in experiment 1 and a failure to fully believe the experimental rejection manipulation in experiment 2.

Joshua Nagel is a super-senior at Yeshiva College majoring in Psychology and minoring in English. Along with his research, he dabbled in Yeshiva College student government, the Wilf Campus Writing Center, and the Yeshiva College Dramatics Society. He plans on pursuing a Ph.D in Industrial / Organizational Psychology in the coming years.


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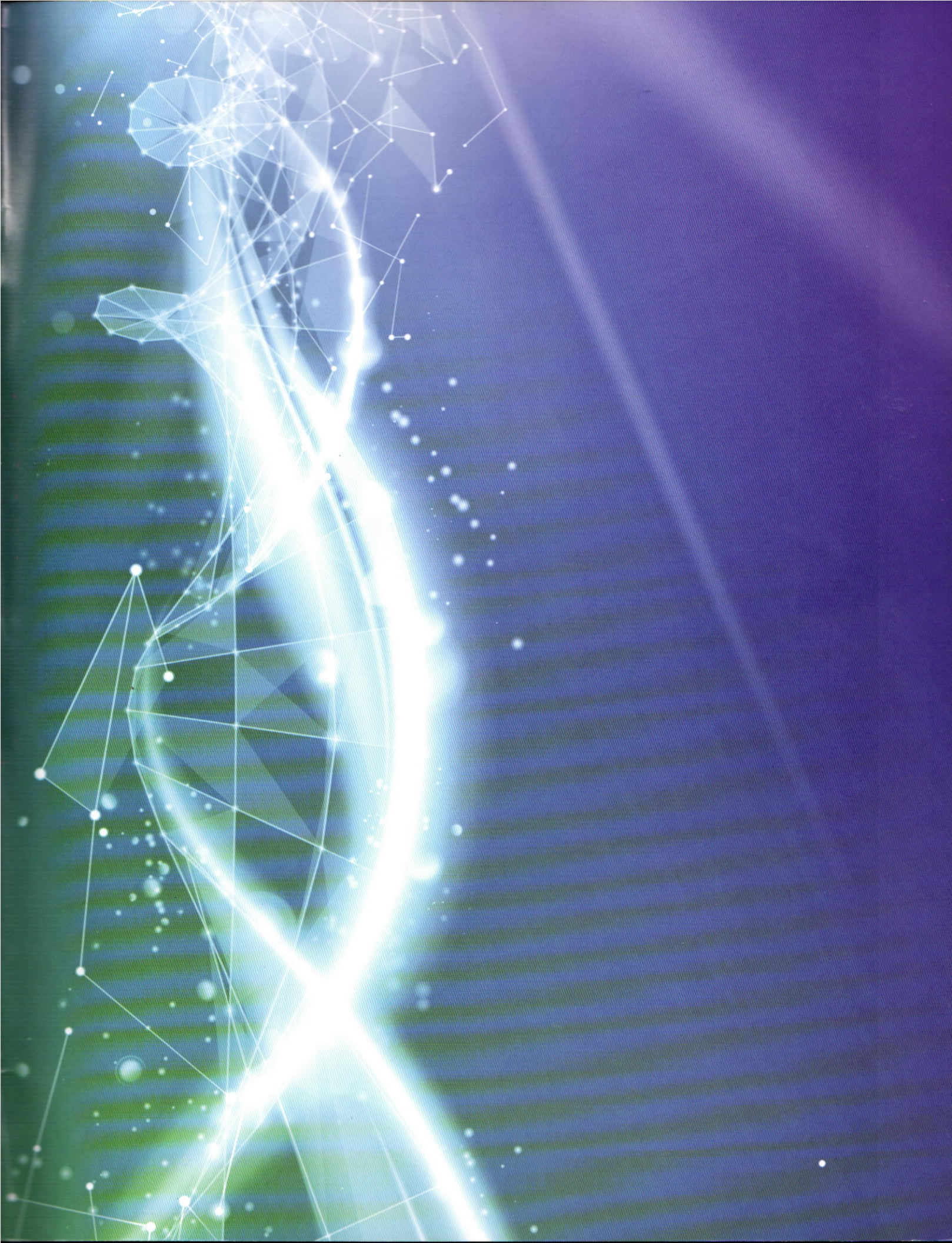

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