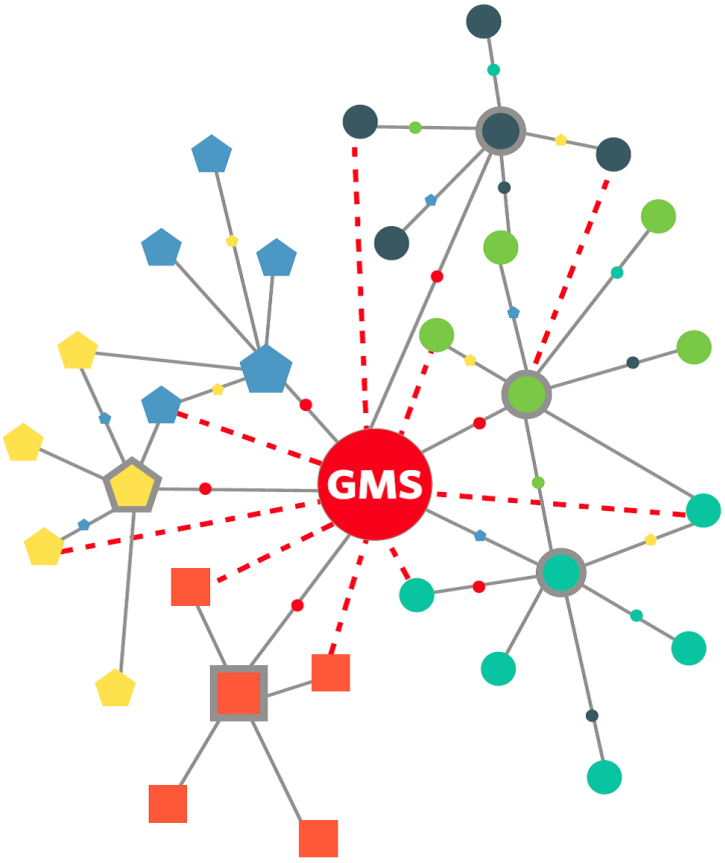
BU Division of Graduate Medical Sciences



Summer Undergraduate Research Program (SURP) Symposium

August 1, 2013

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Gelsolin Expression is Regulated during Brown and White Adipogenesis and is more prominent in White compared to Brown Adipose Tissue

By Jasmin Alves, Westminster College.

Mentors: Professor Stephen R. Farmer Ph.D and Ms. Lynes Torres.
Laboratory: Farmer Lab, Department of Biochemistry, Boston University School of Medicine

Obesity is pandemic. It gives rise to several life threatening comorbidities including insulin resistance, diabetes mellitus, cardiovascular disease, and osteoarthritis. Consumption of excess calories and their storage as triglycerides in adipose tissue is the major cause of obesity. There are two forms of adipose tissue, white (WAT) and brown (BAT). BAT is substantially different from WAT since it metabolizes rather than stores energy. This makes BAT a therapeutic target for weight loss. The ablation of Myocardian Related Transcription Factor-A (MRTFA), a co-activator of the Serum Response Transcription Factor (SRF), can promote the appearance of brown adipocytes in murine WAT. Gelsolin, a downstream target of SRF, is an actin filament severing protein and is required for white adipogenesis. Gelsolin expression has not been investigated in BAT. We hypothesize that gelsolin is enriched in WAT compared to BAT and its expression is regulated by factors other than SRF, which are involved in brown adipocyte formation.

3T3-L1 fibroblasts and brown preadipocytes were used to investigate the mechanisms controlling expression of gelsolin during white and brown adipogenesis. The 3T3-L1 fibroblasts were grown in 10% calf serum (CS) Dulbecco's modified eagle medium (DMEM) with penicillin and streptomycin. Two days post-confluence, the cells were treated with an adipogenic cocktail dexamethasone (D), methylisobutylxanthine (M) and insulin (I). The brown preadipocytes were cultured in 10% fetal bovine serum (FBS) DMEM with the antibiotics. These cells were treated with DMI plus indomethacin (INDO) and thyroid hormone (T3). This cocktail was replaced with 10% FBS DMEM plus insulin after 2 days. RNA and protein samples were isolated from adipose depots of wild type and MRTFA$^{-/-}$ mice and provided by Chendi Li. To analyze protein expression, Western Blot analysis was done with equal amounts of protein per a lane. Quantitative real-time PCR (qRT-PCR) was performed with SYBR green fluorescent dye on cDNA synthesized from sample RNA. C_t values were normalized to TATA binding protein (TBP), an endogenous control.

The data show that gelsolin mRNA and protein expression remain at fibroblast/preadipocyte levels during the first 48 hours of white and brown adipogenesis. Both parameters then drop several fold as the mature adipocytes form. The level of gelsolin mRNA and protein production was significantly lower in BAT compared to the inguinal or epididymal WAT depots in wild type (WT) and MRTFA-/- mice. There were no statistically significant differences in gelsolin expression in any of the depots of WT compared to MRTFA-/- mice, suggesting that gelsolin is not regulated solely by SRF and MRTFA.

The fact that gelsolin expression is higher in WAT than BAT suggests it could be positively regulated by transcription factors unique to WAT. It is also possible that BAT factors suppress its expression. Future studies could be directed towards identifying those factors and likely candidates include the nuclear hormone receptor, peroxisome proliferator-activated receptor (PPARγ) since its activation by synthetic ligands promotes brown adipocyte formation. Such activation suppresses as well as promotes gene expression.

Regulation of small RNA expression in *Neisseria Gonorrhoeae*

By Carolina Amorim, Weidner University.

Mentors: Dr. Caroline Genco Ph.D. and Dr. Ryan McClure Ph.D.
Laboratory: Genco Lab, Deparment of Microbiology and Section of Infectious Diseases, Department of Medicine, Boston University School of Medicine

The Gram-negative bacteria *Neisseria gonorrhoeae* is a strict human pathogen and is the etiological agent of gonorrhea, a sexual transmitted disease. Gonorrhea is found worldwide and is the second most common reportable disease in the United States. Currently, there is no effective vaccine for the disease. *N. gonorrhoeae* requires several important nutrients to grow during infection and must also contend with the host immune response. This makes transcriptional regulation in *N. gonorrhoeae* crucial for survival. Small RNAs (sRNAs) have been shown to play a role in transcriptional regulation of many pathogenic bacteria and can alter gene expression by acting as post-transcriptional factors that regulate translation of mRNAs or by binding to proteins directly. Interestingly, sRNAs are only beginning to be analyzed in detail in *N. gonorrhoeae*. The purpose of this study was to determine how individual sRNAs of *N. gonorrhoeae* that may be involved in the infection process are expressed and regulated under different conditions.

N. gonorrhoeae strain F62 was grown on gonococcal agar plates for 16 hours before culturing in chemically defined media (CDM). *N. gonorrhoeae* was then subjected to several different growth conditions relevant to infection including varying temperatures and levels of hydrogen peroxide and nitrite. Expression of sRNAs in the exponential and stationary phase of growth was also analyzed. In addition, several iron-regulated transcripts, including sRNAs, are known to be regulated by the gonococcal Ferric Uptake Regulator (Fur), a protein that is responsible for iron homeostasis. We therefore tested the regulation of sRNAs in wild-type and Fur mutant strains of *N. gonorrhoeae* to determine whether sRNAs were regulated by iron via the Fur protein. Bacterial RNA was collected via a phenol/chloroform method and iron and Fur regulation of sRNAs was determined via quantitative RT-PCR with regulation via other conditions being determined via Northern blot analysis.

Our results via Northern blot analysis show different patterns of expression for the different sRNAs tested. For several sRNAs the data show possible regulation via hydrogen peroxide concentration and temperature changes. Conversely, there did not appear to be remarkable regulation in response to nitrite concentrations or growth phase for the sRNAs tested. One sRNA showed a large variation in expression patterns over several conditions with some conditions revealing other RNA transcripts detected by Northern blot analysis. When examining iron and Fur regulation, several sRNAs showed higher expression under high iron conditions and may be activated by Fur. In other cases there was little regulation via iron or Fur.

These experiments show that sRNAs can be influenced under different growth conditions, including conditions relevant to infection. It is possible that these sRNAs affect the translation of mRNA targets that have critical roles in the infection process of *N. gonorrhoeae*. Future studies in our laboratory will focus on identifying such targets. It may also be productive to screen for potential protein targets of sRNA regulation to fully understand the mechanisms surrounding the life cycle of the pathogen and its infectious potential.

Alterations in Extracellular Redox States Affect Pancreatic β-Cell Insulin Secretion Levels

By Lynisha Farrell, University of the Virgin Islands.

Mentors: Dr. Barbara E. Corkey Ph.D. and Dr. Jude Deeney Ph.D.
Laboratory: Corkey Lab, Obesity Research Center, Department of Medicine, Boston University School of Medicine

Type 2 Diabetes Mellitus is a metabolic disease characterized by basal hypersecretion of insulin and insulin resistance, all features characteristic of a malfunction in the pancreatic beta-cell. Cellular redox states can be influenced by energy availability and reactive oxygen species (ROS). Recently, it has been suggested that an abnormal extracellular redox state could be linked to irregular cell function. The body regulates the redox state through a series of 'redox pairs', the primary extracellular pair being Cysteine (CyS) and Cystine (CySS). The effects of physiologically relevant alterations in redox potential on the function of the pancreatic beta-cell were thus investigated, with the hypothesis that there would be differences in insulin secretion with variations of the extracellular redox state.

Clonal rat beta-cells (INS-1 832/13) were cultured in 48-well plates to ~80% confluence. Acute incubations were performed in KREBS buffer under low (2mM) and high (8mM) conditions using various concentrations of CyS and CySS. Insulin secretion (HTRF) and glycerol release (NADH luminescence).

There were no significant differences in basal insulin secretion with respect to variations in different extracellular redox states. There were, however, significant changes in glucose/insulin secretion for the different redox states under a high KREBS buffer condition. Changes in Glycerol release were also observed, but the significance of this finding has yet to be fully understood. It was hypothesized that differences in redox potential would be characterized by differences in function. Changes seen at different millivoltage potentials confirmed that different redox states produced different output levels of insulin secretion. In relation to the different Cys/CySS ratios, insulin secretion levels were increased when the extracellular redox states were reduced.

A potential future study would be to determine the effects on redox states on ROS in the cell model, on HbA1c within Type 2 Diabetic patients, and to observe whether altering such redox states with pharmacological interventions provides a measurable benefit.

Protein translation inhibitor reduces TDP-43 inclusion formation

By Norma A. Hernandez, Georgia State University.

Mentors: Dr. Benjamin Wolozin, M.D., Ph.D. and Mr. Atsushi Ebata
Laboratory: Wolozin Lab, Department of Pharmacology and Experimental Therapeutics, Boston University School of Medicine

Cytoplasmic inclusions of transactive response DNA-binding protein 43 kDa (TDP-43) are pathological hallmarks of amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTLD). Past studies have suggested globally decreasing protein synthesis reduces the level of misfolded proteins. It has also been hypothesized that this can also lead to an increase in proteolytic and chaperone functions. Here, we hypothesized that mild inhibition of protein translation will reduce the TDP-43 inclusions through activation of protein homeostasis mechanisms.

A stable Pheochromocytoma (PC-12) cell line that inducibly expresses EGFP-tagged human TDP-43 was stressed with sodium arsenite and treated with varying concentrations of protein translation inhibitors, such as Guanabenz, PF4708671, Emetine, GC7, or Rapamycin. Fluorescence imaging was then used to analyze any changes in inclusion formation.

Among all compounds tested, Emetine significantly decreased the number of cells with TDP-43 inclusions dose dependently. There was also a 10% reduction of cells expressing the fluorescently tagged TDP-43 treated with high doses of Emetine. Even though high concentrations of Emetine affected protein translation, the magnitude of inhibition on inclusion formation surpassed that of translation inhibition. This suggests that the reduction of TDP-43 inclusions is not only due to the lowered protein translation, but also due to additional mechanisms, such as increased chaperone activities.

Evaluating the effects of Emetine on TDP-43 protein levels and its mode of action in chaperone activities will shed light on how the modulation of protein translation improves the proteostasis of pathologically linked TDP-43 inclusion formations.

Cockroach allergen changes the balance between ciliated and secretory cells in the respiratory epithelium

By Mayara Lorena de Souza, Washington and Jefferson University.

Mentors: Dr. William Cruikshank Ph.D. and Dr. Marina Tuzova M.D. Ph.D. M.P.H.
Laboratory: Cruikshank Lab, Pulmonary Center, Boston University School of Medicine

The early development of asthma is not completely understood. It requires both genetic predisposition and allergen exposure, with a strong correlation seen between viral (Respiratory Syncytial Virus, RSV) infection of ciliated cells in the lung epithelium with subsequent asthma development. Specifically, Notch signaling is published to be involved in the control of cell fate decisions, in the establishment of asymmetries and in the timing of differentiation during lung development. The goal of this project was to establish whether the exposure to cockroach allergen (CR) can affect proliferation and differentiation of epithelial cells, with a specific effect on ciliated cells that would lead to increased RSV infection.

Mouse primary epithelial cells and human epithelial cell line were used. After attachment, the cells were exposed to CR, incubated for a week and infected with RSV in different dilutions. Plates without CR were used as a positive control for infection, and plates with no RSV - as a negative control. A week after the infection, Air Liquid Interface (ALI) cultures were started by removing the media from the apical surface. At day 7, cells were fixed and double stained for ciliated cells and the presence of RSV using an immunofluorescent method. In order to quantify the signal, total mRNA from mouse and human samples was isolated with Trizol method and qRT-PCR was performed by standard protocol using FOXJ1, MUC5AC and SCGB1A1 primers.

CR exposure induced an increase in proliferation of ciliated cells compared to media controls, and these cells were more susceptible to RSV infection. Microscopy analyses of double stained epithelial cells from mouse and humans showed more ciliated cells in samples exposed to cockroach allergen, either infected or not. Ciliated cells demonstrated a higher rate of infection compared to the positive control. qRT-PCR showed unchanged numbers of goblet cells, a decrease in clara cells and increase in ciliated cells, when they were exposed to CR. The human samples also showed fewer clara cells and increased number of ciliated cells when exposed to CR. Goblet cells were not found in the human samples, and the experiments will be repeated in the future

CR exposure changed the differentiation pattern of the lung epithelial cells. The mechanism for this is not proved yet, but most likely involves the Notch pathway. The increase in ciliated cell leads to an increase in the rate of infection by RSV. The change in ciliated cell numbers in addition to the increase in RSV susceptibility indicate that CR exposure early in life can play a critical role in altering the lung phenotype, thereby increasing a predisposition for asthma development.

Role of the Aryl Hydrocarbon Receptor in Human Breast Cancer Cell Chemo-Resistance

By Victor E. Otaño-Riviera, University of Puerto Rico at Ponce.

Mentors: Dr. David Sherr Ph.D. and Ms. Elizabeth Stanford, M.A.
Laboratory: Sherr Lab, Department of Environmental Health. Boston University School of Public Health

The aryl hydrocarbon receptor (AhR) is a ligand-activated transcription factor that has been implicated in cancer pathogenesis and progression. When activated by environmental chemicals such as dioxin and Polychlorinated Biphenyls (PCBs), the AhR can activate the transcription of cytochrome P450 enzymes. P450 enzymes can metabolize environmental chemicals into mutagenic metabolites that eventually contribute to cancer development. AhR hyper-expression is prevalent in mammary tumors, including ER^-, PR^- and $HER2^-$ Triple Negative Breast Cancers (TNBCs). Results from our laboratory demonstrate that AhR-knockdown significantly inhibits mammary tumor invasion (*in vitro*) and tumor metastasis (*in vivo*). Given this background, AhR was investigated to see if modulation of its activity would affect the efficacy of chemotherapeutics by altering the chemo-resistance of cancer cells. Based on preliminary data, it was hypothesized that a reduction in AhR activity would increase chemotherapeutic efficacy.

MTT assays were performed with Hs578T cells, a TNBC tumor cell line, to measure cell viability after AhR modulation in conjunction with Adriamycin and Paclitaxel treatment. Here, cells were treated with different concentrations of chemotherapeutics for 24 hours, after which the MTT reagent was added and light absorbance was assayed as a measure of viability. In addition, quantitative Polymerase Chain Reactions (qPCR) were performed to determine if modulating AhR activity would affect mRNA expression levels of *ABCC1* and *ABCG2*, genes that are involved in chemotherapeutic resistance. The role of the AhR in the regulation of apoptotic pathways was also studied by quantifying *Bax1* and *BCL2* mRNA expression levels. Confluence testing was performed to study whether over-confluent cells, compared to non-confluent cells, had altered AhR activity.

Cell viability analysis showed that modulation of AhR activity by AhR antagonism increases cell death after chemotherapeutic treatment. Results from qPCR were inconclusive and require additional analysis. Data from confluence testing found that there were no significant differences between over-confluent and non-confluent cells with respect to their AhR activity levels.

In conclusion, it appears that decreasing AhR activity with a chemical AhR antagonist increases the effectiveness of chemotherapeutics Adriamycin and Paclitaxel in Hs578T cells. Future efforts will focus on elucidating the role that the AhR plays in the regulation of apoptotic pathways, as well as on performing *in vivo* assays to investigate the therapeutic potential of AhR antagonism via pharmacological interventions.

Age-Related Differences of Adult Neurogenesis in Zebrafish

By Carlo Quintanilla, San Diego State University.

Mentors: Dr. Irina V. Zhdanova M.D. Ph.D. and Mr. Alex Stankiewicz
Laboratory: Zhdanova Lab, Department of Anatomy and Neurobiology, Boston University School of Medicine

The Zebrafish has proved to be a valuable model for studying the timing and progression of adult neurogenesis within the central nervous system of diurnal vertebrates. In contrast to the two zones of new neuron formation in mammalian vertebrates, 16 distinct zones of cell proliferation in the adult Zebrafish have been identified and well characterized. Age-related decreases in the number of newly generated neurons have been shown in both rodents and some species of primates such as the marmoset. However, age related differences of adult neurogenesis have not been observed in Zebrafish.

To observe such differences, a 24-Hr Bromodeoxyuridine (BrdU) administration was carried out on 36 adult Zebrafish of three different age groups (6 month, 1 year and 1.5 year old) of both sexes. BrdU is a thymidine analog that becomes incorporated into the DNA of dividing cells, thus serving as a marker for proliferating cells. The animals were subsequently sacrificed and brains dissected, sectioned and immunostained to visualize BrdU positive nuclei. BrdU positive nuclei in five proliferative zones were quantified by manual cell counts under a light microscope at 40x magnification. Also, volumetric measures were taken of total brain tissue utilized in cell counting to normalize for differences in brain size. Cell counts for each of the five proliferative zones were divided by total brain volume (mm^3) collected, in order to acquire a measure of density (n/mm^3).

Although not statistically significant, mean densities for all five proliferative zones were greater in the 1.5 year old fish than in the 1 year old fish. Mean values for total number of BrdU positive cells did not differ significantly between the 1.5 and 1 year old fish. However, the current data represents fish from only two age groups (1.5 and 1 year old) in males only. Sexual dimorphism in particular regions of the adult Zebrafish brain such as the hypothalamus has previously been observed, which may also suggest differential patterns of cell cycle progression in Zebrafish females.

Further analyses should be conducted to observe differences in adult neurogenesis between males and females, and between 6 month old and 1.5 year old fish. Together, the results of such analyses will serve to fine tune the Zebrafish model for studying adult neurogenesis which can aid in the development of new therapeutics to curb the effects of neurodegeneration in humans.

GIV regulates integrin signaling pathways upon Extra Cellular Matrix stimulation

By Igor Rodrigues Ramos, Washington and Jefferson University.

Mentors: Dr. Mikel Garcia-Marcos Ph.D. and Dr. Anthony Leyme Ph.D.
Laboratory: Garcia-Marcos Lab, Department of Biochemistry, Boston University School of Medicine

GIV (Gα-interacting vesicle-associated protein) is a cytoplasmic protein that acts as a non-receptor GEF (Guanine-nucleotide Exchange Factor) allowing heterotrimeric G protein activation, leading to several cell responses, such as metabolic enzyme activation, plasma membrane receptor exposure, cell motility, and so on. Although little is known about GIV's mechanism of action and cellular functions, previous laboratory data has demonstrated that GIV enhances Akt and FAK phosphorylation upon Type 1 collagen stimulation. Therefore, this project analyzed whether GIV controls integrin signaling upon activation by the Extracellular Matrix components.

MDA-MB-231 breast cancer cells were cultured according to American Type Culture Collection (ATCC) guidelines. A subset of cells was transfected with a plasmid control (Scramble) or a plasmid that knocks-down GIV expression (ShRNA-GIV). A proliferation assay was performed by counting the cells of each plate over 5 days. Then the plated cells were re-suspended in media without Fetal Bovine Serum (FBS) to decrease protein phosphorylation rates. After one hour, cells were plated on plates coated with Fibronectin or Matrigel at five different time points. At this point, Western Blot analysis was performed to measure the amount of phosphorylated Akt (Ser 473) and FAK (Tyr 397). A PBD pull-down and a RBD pull-down were performed to detect the active form of Rac1 and RhoA, respectively.

In order to verify whether GIV plays a role in cell proliferation pathways in breast cancer cell lines, the proliferation assay was accomplished with the MDA-MB-231 Scramble and ShRNA-GIV but no significant difference was observed between the samples. Akt and FAK phosphorylation were analyzed in MDA-MB-231 Scramble and ShRNA-GIV cells upon Matrigel or Fibronectin stimulation to verify GIV influence on integrin signaling pathways; no difference was observed between the two subsets. To verify whether GIV is required for the activation of proteins responsible for cell motility, PBD pull downs and RBD pull downs were performed to detect the active form of Rac1 and RhoA in cells upon Type 1 collagen stimulation, but again, no difference was observed between the subsets.

In conclusion, GIV does not affect the proliferation of MDA-MB-231 breast cancer cells. Furthermore, in contrast to Type 1 collagen stimulation, GIV is not required for FAK and Akt activation upon Fibronectin or Matrigel stimulation. Finally, GIV's depletion does not affect Rac1 and RhoA activation upon Type 1 collagen stimulation.

Is Erythrocyte Spectrin really 190nm in Length?

By C. Breann Williams, Jackson State University.

Mentors: Dr. Esther Bullitt Ph.D. and Dr. C. James McKnight Ph.D.
Laboratory: Bullitt Lab, Department of Physiology and Biophysics, Boston University School of Medicine

Spectrin is essential for proper erythrocyte function. Mutations in spectrin can lead to spherocytosis, a disorder causing erythrocytes which are sphere shaped rather than the normal bi-concave disk. These mis-shaped erythrocytes are susceptible to hemolysis in capillaries, resulting in anemias.

The α_2, β_2 spectrin tetramer is a major part of the interlocking web that supports the plasma membrane of erythrocytes; the bi-concave morphology of the erythrocyte relies on this cytoskeleton. Unlike most cells, the erythrocyte cytoskeleton is comprised primarily of spectrin. Short protofilaments of actin in junctional complexes organize spectrin into a hexagonal grid that is attached to the membrane via ankrin and other junction complex proteins to shape and control cell morphology.

Spectrin tetramers isolated from spread erythrocytes have been measured to be ~190nm. However, other evidence suggest a much more compact length of 50-80nm in the intact erythrocyte.

We hypothesize that spectrin tetramers, when present in erythrocytes exhibit a 50nm length between actin filaments and extend, to the previously seen 190nm length when the red blood cell membrane is expanded in low salt. We predict that we will observe ~50nm spectrin tetramers in high salt in the presence of actin, by electron microscopy (EM).

Spectrin was a gift from Dr. David Speicher and Dr. Sandra Harper. Actin was purified from acetone powder by extraction, filtration, centrifugation, polymerization, high-salt wash, sedimentation of filamentous actin, depolymerization, and finally polymerization by dialysis. Purity of samples was confirmed by running a tricine gel to separate protein components based on molecular weight. EM was performed on negatively stained samples, frozen hydrated samples, and on freeze-dried samples of actin, spectrin, and actin-spectrin complexes.

Our preliminary studies show that EM images of negatively stained samples produce very low contrast images of spectrin. The visibility of spectrin is greatly enhanced in frozen hydrated samples. Furthermore, freeze dried samples provide additional image enhancement. In addition to the shorter spectrin filaments, we have observed a decrease in aggregation of spectrin. At higher concentrations, spectrin associates with other spectrin filaments as opposed to actin. When the concentration of spectrin is decreased it associates with the actin filaments. The data show spectrin comparable to the rungs of a ladder with actin forming the rails. In these data we see actin filaments ~50nm apart, cross linked by spectrin.

Our data indicate spectrin has a resting length of ~50nm. This finding is consistent with the hypothesis that in the presence of actin and moderately elevated salt concentration within the erythrocyte membrane spectrin is compact with a 50nm length and when the membrane is deformed the length of spectrin extends to 190nm.

In the future we will continue to test the proposed model. Another important avenue will be to add other proteins which are associated with spectrin to better imitate the physiological system of the erythrocyte.

History of the Summer Undergraduate Research Program (SURP)

The progression of SURP from pilot program to competitive summer internship for aspiring biomedical scientists began in 2010 under the leadership of Dr. Linda Hyman, Associate Provost. Dr. Hyman started the summer program (SURP) with ten academically talented undergraduate students from Xavier University (Louisiana) who were interested in careers as research scientists. The students conducted research full time for ten weeks under the guidance of GMS faculty mentors with support from lab managers, postdoctoral fellows and experienced graduate students. The SURP cohort lived in residential housing on Boston University's Charles River Campus, participated in weekly professional development activities, and enjoyed social events facilitated by a peer leader. At the end of the summer internship, students presented their research in a special symposium, and were encouraged to share their projects with faculty and peers at their home institutions and at national meetings.

The model of the GMS summer program has worked successfully for four years with minor adjustments based on feedback from students and mentors. Several accomplishments are noteworthy for SURP in 2013. First, the number of URM applicants has increased significantly over four years. In 2010 we received 12 applications from underrepresented minority students (URM) while in 2013 the number grew to 245. Key factors related to the aforementioned applicant growth include: a strong online presence, information provided by program directors at historically black and other minority serving colleges and scholar programs (i.e. MARC, RISE, McNair), postings at offices of career services, recommendations by faculty advisors, word-of-mouth, and conscientious follow up of all student inquiries. The rise in the quantity of URM applications parallel an increase in the average GPA of enrolled SURP students (2010 = 3.28 vs. 2013 = 3.58).

In terms of programmatic changes, GMS has experimented with different approaches to the professional development component of the program. During 2010 and 2011 we focused on providing faculty "science talks" to inform students

about the depth and breadth of research at BUSM especially research conducted by faculty members from underrepresented minority groups. In 2012, following a suggestion of Dr. William Cruikshank, we shifted the emphasis of professional development to skill-building within the framework of a Journal Club. Students met once a week and learned how to dissect and present a scientific journal article. The latter activity was focused on articles related to the research focus of the student's lab. The Journal Club assignment helped to build the students' personal confidence in public speaking. Also, a recent decision to offer two credits for completion of the ten week program has received positive feedback.

Finally, in keeping with opportunities provided by the GMS Office of Professional Development and Postdoctoral Affairs, SURP arranged speakers and field trips that encourage students to explore diverse research careers offered both within and outside a large academic research institution.

To date we are proud to announce that SURP has successfully "graduated" thirty-eight students who have utilized their summer research experience as a building block toward continued pursuit of their professional goals in biomedical research and health care.

Acknowledgements

Thank you to students who participated in the 2013 SURP program, faculty mentors, postdoctoral fellows, graduate students, departmental chairs and other supporters of the program from the research community at Boston University Medical Campus. A special thanks to Dr. Stephen Brady, Dr. Isabel Dominguez, Dr. Ron Corley, Beth Sommers, Dr. Shelley Russek, Sandra Grasso, Dr. William Cruikshank, Assistant Dean of Diversity and Multicultural Affairs for GMS, Sanghee Lim, SURP Program Assistant, the faculty and staff of the GMS office and Dr. Linda Hyman, Associate Provost.

"My first real taste of translational research with a human impact, and greatly influenced me to continue research in a medical setting. Plus I loved the SURP community." Carlos Rodriguez

"I truly enjoyed presenting my research at the end of the summer because it gave me more experience speaking in front of an audience and showed me that I would like to continue sharing my research with anyone who is willing to listen. My summer at BU was one of the best research experiences ever." Debra Garvey

Why SURP at BU?

Mentorship: Faculty mentors and lab members provide scientific guidance and community. Students work side by side with PhD students, postdoctoral trainees and faculty giving them a true sense of what it is like to conduct cutting edge research.

Networking: Research interns have the opportunity to build a professional network among like-minded peers.

Student Life: Boston University is the summer destination for hundreds of students. Your summer will be memorable, challenging, and filled with new experiences.

Boston: The city is a global center for medical research, health care, biotechnology and education; and culturally rich with history, art, music and sports.

Opportunity: Research interns gain new knowledge and skills and build confidence leading to opportunities to present research at national conferences.

Paid internship: Research interns are paid a stipend, housing and travel expenses.

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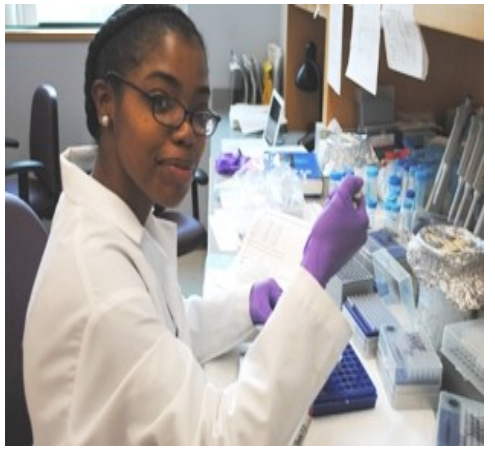
Boston University School of Medicine
Division of Graduate Medical Sciences

Summer Undergraduate Research Program



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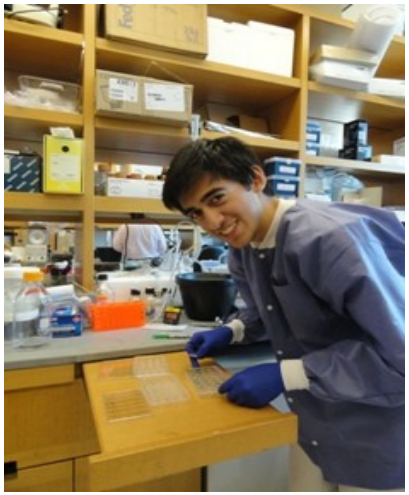
Summer Undergraduate Research Program

The Summer Undergraduate Research Program (**SURP)** is an internship sponsored by Boston University's Division of Graduate Medical Sciences (GMS), a recognized leader in graduate education and research in the biomedical sciences. The **SURP** program is located within GMS on the Boston University Medical Campus in the historic South End of Boston, a diverse and multicultural neighborhood.

SURP offers motivated and academically talented students a valuable opportunity to confirm a strong interest in doctoral study in the biomedical sciences and future professional roles as research scientists.



Program Highlights

The summer internship includes:

- Conducting research for 10 weeks in a lab engaged in biomedical research
- Participating in activities of the research community
- Development and presentation of an independent research project
- Weekly science talks
- Career discussions
- Journal club
- Professional development seminars
- Social activities in and around Boston University and the city of Boston
- Designated program staff and faculty advisor
- Opportunity to apply for travel awards for major national conferences such ABRCMS & SACNAS

Application Opens December 1st

Learn. Explore. Discover.

Living in Boston over the summer is a great opportunity to enjoy interesting field trips with the SURP program.

Boston is a unique college-oriented city within walking distance of shopping, restaurants and major attractions.