AWARD NUMBER:  
W81XWH-12-1-0043

TITLE: 
The South Carolina Collaborative Undergraduate HBCU Student Summer Training Program

PRINCIPAL INVESTIGATOR:  
Marvella E. Ford, PhD

CONTRACTING ORGANIZATION:  
The Medical University of South Carolina  
Charleston, South Carolina 29425

REPORT DATE:  
March 2014

TYPE OF REPORT:  
Annual Summary

PREPARED FOR: U.S. Army Medical Research and Materiel Command  
Fort Detrick, Maryland 21702-5012

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| 1. **REPORT DATE**  
March 2014 | 2. **REPORT TYPE**  
Annual Summary | 3. **DATES COVERED**  
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|
| **6. AUTHOR(S)**  
Marvella E. Ford, Ph.D.  
Omar Bagasra, Ph.D.  
Judith D. Salley, Ph.D.  
Leroy Davis, Ph.D. | **5e. TASK NUMBER**  
| **5f. WORK UNIT NUMBER**  
| **7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)**  
The Medical University of South Carolina  
Hollings Cancer Center  
Charleston, SC 29425  
Claihn University  
Orangeburg, SC 29115  
SC State University  
Orangeburg, SC 29117  
Voorhees College  
Denmark, SC 29042 | **8. PERFORMING ORGANIZATION REPORT NUMBER**  
| **9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)**  
Commander, U.S. Army Medical Research and Materiel Command  
ATTN: MRMC-IM  
504 Scott Street  
Fort Detrick, Maryland 21702-5012 | **10. SPONSOR/MONITOR’S ACRONYM(S)**  
|
Background: There is a critical need to increase the racial/ethnic diversity of prostate cancer researchers. The goal of the Training Program is to provide research training activities to 12 students over a 3-year period from three Historically Black Colleges and Universities (HBCUs) in South Carolina: Claflin University, South Carolina State University, and Voorhees College. The three aims of the Training Program are: Aim 1.) To provide training in the basics of research design and methods to 12 Student Fellows each year from the three HBCUs; Aim 2.) To immerse 4 Student Fellows per year in prostate cancer research; Aim 3.) To implement a unique dual-level research mentoring strategy for the students. 

Results: During the current reporting period, 4 Student Fellows were identified, recruited to participate in the program, and admitted to the DOD South Carolina Collaborative Undergraduate HBCU Student Summer Training Program. The Student Fellows were matched with Research Mentors at MUSC, with whom they conducted research in the summer of 2013. Each Student Fellow prepared a scientific paper, gave a scientific presentation at the end of the summer program, and completed a 9-week Princeton Review Graduate Record Examination Test Preparation Course. In the summer of 2013, students at SCSU participated in summer program lectures via videoconference. 

Conclusions: State-of-the art comprehensive prostate cancer research education and training opportunities were provided to 4 Student Fellows from HBCUs in South Carolina. Each Student Fellow prepared a scientific paper and gave at least 1 scientific presentation. Nine Student Fellows gave scientific presentations, two of which were presented at national scientific meetings. A cadre of scientists who are well-prepared to conduct research spanning the continuum from basic science to clinical science to population-based research was developed.
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INTRODUCTION

The Scientific Context of the Training Program
The South Carolina Collaborative Undergraduate HBCU Student Summer Training Program (referred to as the Training Program) will provide a biomedical research training experience to 12 students over a three-year period (2012-2015) from three Historically Black Colleges and Universities (HBCUs) – Claflin University (CU), South Carolina State University (SCSU), and Voorhees College (VC). Undergraduate students from the three HBCUs (defined as Student Fellows) will participate in research intensive summer internships in the laboratories/research units of senior prostate cancer research scientists at the Medical University of South Carolina Hollings Cancer Center (MUSC HCC). This new Training Program application builds upon the success of the previously funded Department of Defense (DOD) prostate cancer research training program (2009-2011) and the long standing NIH funded Summer Undergraduate Research Training Program at MUSC (1992-present). The inter-institutional leadership of these summer training efforts have carefully examined the formative and summative evaluations provided by previous Student Fellows, Mentors, and Advisors in order to maximize the ability of this new enhanced program proposal to reach its ultimate goal – to increase the racial and ethnic diversity of emerging scientists who may choose prostate cancer research careers in basic, clinical, and population sciences. In this new application, the Training Program has been improved with a built-in, dual-level research and career mentoring strategy involving current graduate students and post-doctoral trainees included on the mentoring team; the addition of a clinical shadowing experience in the MUSC/HCC multidisciplinary genitourinary clinics and tumor board; more year-round opportunities for which the Student Fellows will participate; and an opportunity for Training Program alumni to continue relationships with new trainees going forward. Measurable outcomes of the Training Program will include the number of Student Fellows who take the Graduate Record Examination (GRE), apply to graduate school, and give scientific presentations and publish their research results in peer-reviewed scientific journals based on their summer research experience. Efforts will be made to capture long term outcomes as well as to determine how many Student Fellows choose to pursue a medical or biomedical focused graduate and post graduate career.

The three Specific Aims are to:
Aim 1. To provide training in the basics of research design and methods to 12 Student Fellows each year from the three HBCUs;
Aim 2. To immerse 4 Student Fellows per year in prostate cancer research;
Aim 3. To implement a unique dual-level research mentoring strategy for the students.

Program Director and Training Team
Dr. Marvella E. Ford is the Program Director. Drs. Omar Bagasra (CU), Judith Salley (SCSU), and Leroy Davis (VC) are Associate Directors. This four-person leadership team collaborates closely in the management and administration of the award, as well as the continued development and enhancement of the Training Program. The Program Director and Associate Directors share scientific interests in health disparities, serve in other leadership roles within their institutions, and meet frequently, both formally and informally. These individuals form the Executive Committee for the Training Program. Each institution has appointed Faculty Advisors consisting of Dr. Ewen McLean (CU), Dr. James B. Stukes (SCSU), and Mrs. Gayle Tyler Stukes (VC).
Statement of Work

Task 1. Identify and Recruit the Student Fellows
   (a) Identify the pool of potential Student Fellows (Year 2, months 1-3)
   (b) Interview the potential Student Fellows (Year 2, months 1-3)
   (c) Select the top Student Fellows (Year 2, months 1-3)
   (d) Match the Student Fellows with their Research Mentors at MUSC (Year 2, months 1-3)
   (e) Hold the Kickoff Intensive and Luncheon (Year 2, months 4-6)

**Deliverables:** Four Student Fellows per year were identified, recruited to participate in the program, and matched with senior prostate cancer research mentors at MUSC.

Task 2. Provide Training in Biomedical and Prostate Cancer Research
   (a) Conduct Aim 1: Training in the Basics of Research Design and Methods through participation in the MUSC Summer Undergraduate Research Program (Year 2, months 6-8)
   (b) Conduct Aim 2: Prostate Cancer Research Training (Year 2, months 6-8)
   (c) Sponsor the Student Fellows’ Participation in a Graduate Record Examination (GRE) course (Year 2, months 6-8)

**Deliverables:** We provided state-of-the-art comprehensive prostate cancer research education and training opportunities for 4 students from three of South Carolina’s HBCUs. We have developed a cadre of scientists who are well-prepared to play a significant role in discovering and testing new prostate cancer biomarkers. These investigators will conduct research spanning the continuum from basic science to clinical science to population-based research. At least 75% of the Student Fellows will take the GRE and at least 75% of the Student Fellows will apply to graduate school.

Task 3. Prepare Tangible Scientific Products
   (a) Prepare and present scientific abstracts based on the Student Fellows’ prostate cancer research (Year 2, months 10-12)
   (b) Prepare manuscripts that will be submitted to peer-reviewed journals (Year 2, months 10-12)

**Deliverables:** At least 4 scientific presentations will be conducted by Student Fellows. At least 2 peer reviewed publications will result.

Task 4. Evaluate the Training Program
   (a) Assess the number of applicants to the Training Program (Year 2, months 1-4)
   (b) Assess the number of Student Fellows who apply to graduate school (Year 2, months 1-12)
   (c) Assess the number of Student Fellows who are admitted to graduate school (Year 2, months 1-12)
   (d) Assess the number of graduate schools to which Student Fellows are admitted (Year 2, months 1-12)
   (e) Employ several tracking mechanisms to monitor the scientific progress of the students, including:
      1. Searching the MUSC graduate program databases to identify whether any of the students applied, were offered, or accepted positions at MUSC.
      2. Contacting the participating universities’ alumni offices.
3. Employing other internet based search tools/communications (Google, MySpace, Facebook, and Historically Black College/University Connections, etc.) to identify students’ current locations, contact information, and academic achievements (Year 2, months 10-12)

(f) Identify the number of scientific abstracts presented and peer-reviewed publications that result (Year 2, months 10-12)

**Deliverables:** We will prepare a document assessing the tangible products that result from the Training Program.
**KEY RESEARCH ACCOMPLISHMENTS**

**Task 1. Identify and Recruit the Student Fellows**

(a) Identify the pool of potential Student Fellows (Year 2, months 1-3)

(b) Interview the potential Student Fellows (Year 2, months 1-3)

(c) Select the top Student Fellows (Year 2, months 1-3)

To accomplish Tasks 1(a) – 1(c), Dr. Ford, the Program Director worked with Associate Directors Dr. Rebecca Bullard-Dillard and her replacement upon leaving Claflin University, Dr. Omar Bagasra (Claflin University), Dr. Judith Salley (SC State University), and Dr. Leroy Davis (Voorhees College) as well as Faculty Advisors Dr. Ewen McLean (Claflin University), Dr. James Stukes (SC State University), and Mrs. Gayle Stukes (Voorhees College) to identify potential Student Fellows. The Associate Directors and Faculty Advisors issued a call for applicants to their student bodies and personally approached students whom they felt would be outstanding applicants for the summer research program. For example, Drs. Ford (Principal Investigator), Bagasra (Associate Director), Salley (Associate Director), and Davis (Associate Director) communicated via electronic mail to discuss the 2013 SURP application process and deadlines.

To cite another example, to broaden the pool of potential applicants, each Associate Director invited faculty and students from his/her institution to participate in the Ernest Just Symposium held on February 22, 2013 at MUSC. A total of 240 students participated, including 67 students from HBCUs in South Carolina (Table 1.). The 240 students represented 21 different high schools, colleges and universities. A total of 67 students from HBCUs in SC participated in the Symposium, as well as 75 students from HBCUs in other regions of the country. The agenda from the Symposium and the number of students from each institution are included in **Appendices A-B**. Dr. Salley was instrumental in recruiting HBCU students from across the U.S. The students who participated in the Symposium also received a tour of scientific research units at MUSC and met with MUSC faculty members who could become their future research mentors.
In Year 2, the Student Fellows were matched with their Research Mentors at MUSC based on the expressed interests of the Student Fellows as stated in their written MUSC Summer Undergraduate Research Program (SURP) applications. The following tables show the names of the students who participated in the DOD South Carolina Collaborative Undergraduate HBCU Student Summer Training Program, their Research Mentors at MUSC, and their research topics.

<table>
<thead>
<tr>
<th>Student Name</th>
<th>Academic Institution</th>
<th>MUSC Research Mentor</th>
<th>Research Topic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ms. Kiera Addison</td>
<td>SC State University</td>
<td>Dr. Danyelle Townsend</td>
<td>Redox Signaling is Deregulated in Cancer</td>
</tr>
<tr>
<td>Ms. Evelyn Martinez</td>
<td>SC State University</td>
<td>Dr. Steven Rosenzweig</td>
<td>Growth Factor Contribution to Epithelial Mesenchymal Transition</td>
</tr>
<tr>
<td>Ms. Tomesha Nesbitt</td>
<td>Voorhees College</td>
<td>Dr. Shikhar Mehrotra</td>
<td>The Effect of Vitamin D3 on T cell Activation and Death</td>
</tr>
<tr>
<td>Ms. Sadia Robinson</td>
<td>SC State University</td>
<td>Dr. David Turner</td>
<td>Examining the AGE-RAGE Signaling Axis as a Mechanism of Prostate Cancer Disparity</td>
</tr>
</tbody>
</table>
In addition to the students listed above, the Director and Associate Directors leveraged funding from two other grants to support an additional three students:

<table>
<thead>
<tr>
<th>Student Name</th>
<th>Academic Institution</th>
<th>MUSC Research Mentor</th>
<th>Funding Source</th>
<th>Research Topic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ms. Bobbie Blake</td>
<td>Claflin University</td>
<td>Dr. Jennifer Wu</td>
<td>DOD - Southeastern Virtual Institute for Health Equity and Wellness (PI: Slaughter; Project PI: Ford)</td>
<td>NKG2D Signaling Pathways Analysis</td>
</tr>
<tr>
<td>Ms. Franshawn Mack</td>
<td>SC State University</td>
<td>Dr. Marvella Ford</td>
<td>DOD - Southeastern Virtual Institute for Health Equity and Wellness (PI: Slaughter; Project PI: Ford)</td>
<td>Evaluating the Reliability of an Instrument Assessing Cancer Clinical Trial Perceptions in a Predominantly African American Sample in South Carolina</td>
</tr>
<tr>
<td>Ms. Jasmine Fox</td>
<td>SC State University</td>
<td>Dr. Victoria Findlay</td>
<td>NIH/NCI P20 South Carolina Cancer Disparities Research Center (PIs: Ford and Salley)</td>
<td>MiR-204 Negative Regulation of IGF2R as a Mechanism Driving Breast Cancer Disparity</td>
</tr>
</tbody>
</table>

(e) Hold the Kickoff Intensive and Luncheon (Year 2, months 4-6)

The Kickoff Intensive and Luncheon took place during the first meeting of the didactic training program in prostate cancer research. Dr. Debbie C. Bryant from the MUSC College of Nursing, who has a keen interest in working with summer undergraduate students, and Ms. Tonya Hazelton, who coordinates the DOD Training Program, gave an overview of the DOD South Carolina Collaborative Undergraduate HBCU Student Summer Training Program.

Task 1 Deliverables: Four Student Fellows (plus an additional three students who were supported using leveraged funds) were identified, recruited to participate in the program, and admitted to the DOD South Carolina Collaborative Undergraduate HBCU Student Summer Training Program. The Student Fellows were matched with Research Mentors at MUSC, with whom they conducted research in the summer of 2013.

Task 2. Provide Training in Biomedical and Prostate Cancer Research

(a) Conduct Aim 1: Training in the Basics of Research Design and Methods through participation in the MUSC Summer Undergraduate Research Program (Year 2, months 6-8)
The Student Fellows participated in an intensive training program in the Basics of Research Design and Methods through participation in the MUSC Summer Undergraduate Research Program (SURP). The following tables show the SURP curricula from 2013.
<table>
<thead>
<tr>
<th>Date</th>
<th>Topic</th>
<th>Lecturer</th>
</tr>
</thead>
<tbody>
<tr>
<td>May 28</td>
<td>The Development of a New Treatment and Diagnostic Test for Bladder Cancer: From Bench to Bedside</td>
<td>Perry Halushka, MD, PhD</td>
</tr>
<tr>
<td>May 29</td>
<td>Novel Therapies to Treat Acute Kidney Injury: From Bench to Bedside (*note: this lecture will be in BSB 302)</td>
<td>Rick Schnellmann, PhD</td>
</tr>
<tr>
<td>May 30</td>
<td>What is Translational Research?</td>
<td>Carol Wagner, MD</td>
</tr>
<tr>
<td>May 31</td>
<td>Human Subject Research Success Center: How Scientists Get Help Conducting Research/Examples of Translational Research</td>
<td>Susan C. Sonne, PharmD, Royce Sampson, MSN, RN</td>
</tr>
<tr>
<td>June 3</td>
<td>9:00-10:30am MANDATORY: Responsible Lab Citizenship &amp; Mentoring (lecture/discussion)</td>
<td>Ed Krug, PhD</td>
</tr>
<tr>
<td></td>
<td>10:30-11:30am Data Management/Data Manipulation (lecture/case study/discussion)</td>
<td></td>
</tr>
<tr>
<td>June 4</td>
<td>8:00-9:00am MANDATORY: Public Perceptions of Scientific Research (“And the Band Played On”)</td>
<td>Ed Krug, PhD</td>
</tr>
<tr>
<td></td>
<td>9:00-10:00am Questionable Research Practices (discussion of video)</td>
<td></td>
</tr>
<tr>
<td>June 5</td>
<td>8:00-9:00am MANDATORY: Moral Reasoning in Ethical Dilemmas (lecture/case study/discussion)</td>
<td>Ed Krug, PhD</td>
</tr>
<tr>
<td></td>
<td>9:00-10:00am Animal Use in Research (lecture &amp; discussion)</td>
<td>Alison Smith, PhD</td>
</tr>
<tr>
<td>June 6</td>
<td>8:00-9:00am MANDATORY: Authorship and Plagiarism (lecture/case study/discussion)</td>
<td>Ed Krug, PhD</td>
</tr>
<tr>
<td></td>
<td>9:00-10:00am Research Misconduct/Whistleblower Protections (lecture/case study/discussion)</td>
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<tr>
<td></td>
<td>10:00-11:00am Closing Comments/Exit Evaluation</td>
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</tbody>
</table>

Outside Assignment: Complete the University of Montana On-Line RCR training (link below) - you must score a minimum of 70% on all quizzes. Submit paper copies of quiz completion to Stephanie Brown-Guion (BE101F) no later than 4 PM Friday, June 15
(http://ori.dhhs.gov/education/products/montana_round1/research_ethics.html)
NOTE: The schedule on the following pages is color-coded. Lectures in the Black font are required of everyone. You
must select a lecture track for the remainder of the summer. Your choices are Cardiovascular (blue font), Cancer (red
font), Craniofacial biology (pink font), and Neuroscience (green font). If you are part of the OHH group, your lectures are
attached at the end of this schedule.

Lecture Time: 8:30-9:30; Place: Bioengineering Building Room 112

<table>
<thead>
<tr>
<th>Date</th>
<th>Topic</th>
<th>Lecturer</th>
</tr>
</thead>
<tbody>
<tr>
<td>June 7</td>
<td>Hepatic Steatosis in a Growing World: The Impact On Transplantation</td>
<td>Kenneth Chavin, MD, PhD</td>
</tr>
<tr>
<td>June 10</td>
<td>Lipidomics</td>
<td>Ashley Cowart, PhD</td>
</tr>
<tr>
<td>June 11</td>
<td>(C) Kinds of Cancer</td>
<td>Robert Gemmill, PhD</td>
</tr>
<tr>
<td>June 12</td>
<td>Cell Biology – Tissue Ultrastructure</td>
<td>Debra Hazen-Martin, PhD</td>
</tr>
<tr>
<td>June 13</td>
<td>Developmental Biology</td>
<td>Michael Kern, PhD</td>
</tr>
<tr>
<td>June 14</td>
<td>Proteomics Technology</td>
<td>Lauren Ball, PhD</td>
</tr>
<tr>
<td>June 17</td>
<td>Recombinant DNA</td>
<td>David Kurtz, PhD</td>
</tr>
<tr>
<td>June 18</td>
<td>Transcription</td>
<td>Steven Kubalak, PhD</td>
</tr>
<tr>
<td>June 19</td>
<td>(H) The Heart</td>
<td>Perry Halushka, PhD, MD</td>
</tr>
<tr>
<td>June 19</td>
<td>(D) Tooth Development – Room BSB 451</td>
<td>Michael Kern, PhD</td>
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<tr>
<td>June 20</td>
<td>(C) Cancer Cell Cycle</td>
<td>Cynthia Wright, PhD</td>
</tr>
<tr>
<td>June 21</td>
<td>Confocal/Multiphoton Microscopy of Living Cells And Tissues</td>
<td>John Lemasters, MD, PhD</td>
</tr>
<tr>
<td>June 24</td>
<td>Microarray Analysis</td>
<td>Jeremy Barth, PhD</td>
</tr>
<tr>
<td>June 25</td>
<td>(H) Electrical Properties of the Heart</td>
<td>Rupak Mukherjee, PhD</td>
</tr>
<tr>
<td>June 26</td>
<td>(C) Cytogenetics</td>
<td>Daynna Wolff, PhD</td>
</tr>
<tr>
<td>June 27</td>
<td>(N) Retinoids &amp; Vision</td>
<td>Masahiro Kono, PhD</td>
</tr>
<tr>
<td>June 27</td>
<td>(D) Salivary Diagnostics – Room BSB 451</td>
<td>V. Palanisamy, PhD</td>
</tr>
<tr>
<td>June 28</td>
<td>G Proteins</td>
<td>John Hildebrandt, PhD</td>
</tr>
<tr>
<td>July 1</td>
<td>Stem Cells</td>
<td>Amanda LaRue, PhD</td>
</tr>
<tr>
<td>July 2</td>
<td>(N) Dementia</td>
<td>Dr. Mark Kindy, PhD</td>
</tr>
<tr>
<td>July 3</td>
<td>(N) ADD/ADHD</td>
<td>Antonieta Lavin, PhD, Jonathan Dilgen, PhD</td>
</tr>
<tr>
<td>July 5</td>
<td>(H) Arterial Pressure Control &amp; High Blood Pressure</td>
<td>Perry Halushka, PhD, MD</td>
</tr>
<tr>
<td>July 8</td>
<td>Receptors</td>
<td>Steven Rosenzweig, PhD</td>
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<tr>
<td>July 9</td>
<td>(N) Spinal Cord Injury</td>
<td>Narendra Banik, PhD</td>
</tr>
<tr>
<td>July 10</td>
<td>(H) Aspirin &amp; NSAIDS</td>
<td>Perry Halushka, PhD, MD</td>
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<tr>
<td>July 10</td>
<td>(D) Temporomandibular Joint Biomechanics – BSB 451</td>
<td>Hai Yao, PhD</td>
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<td>July 11</td>
<td>(C) Smoking &amp; Cancer</td>
<td>Michael Cummings, PhD</td>
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<tr>
<td>July 11</td>
<td>(D) Periodontal Disease – BSB 451</td>
<td>Keith Kirkwood, DDS, PhD</td>
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<tr>
<td>July 12</td>
<td>(D) Oral Pharyngeal Cancer – BSB 451</td>
<td>Boyd Gillespie, MD</td>
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<tr>
<td>July 15</td>
<td>(C) Epidemiology of Cancer</td>
<td>Kristen Wallace, PhD</td>
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<tr>
<td>Date</td>
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<td>Subject</td>
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<td>-------------------------------------------</td>
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<tr>
<td>July 16</td>
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<td>Atherosclerosis</td>
</tr>
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<td>July 17</td>
<td>(C)</td>
<td>Cancer Chemotherapy</td>
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<tr>
<td>July 17</td>
<td>(D)</td>
<td>Oral Infections – BSB 451</td>
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<tr>
<td>July 18</td>
<td>(N)</td>
<td>Neuroimaging Lab Demonstration</td>
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<tr>
<td>July 18</td>
<td>(D)</td>
<td>Craniofacial Anomalies – BSB 451</td>
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<tr>
<td>July 19</td>
<td>(H)</td>
<td>Renal Regulation of Homeostasis</td>
</tr>
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<td>July 22</td>
<td>(H)</td>
<td>Imaging the Heart</td>
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<td>July 23</td>
<td>(N)</td>
<td>Addiction &amp; Alcohol</td>
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<tr>
<td>July 23</td>
<td>(C)</td>
<td>Cancer Disparities</td>
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<td>July 24</td>
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<td>Schizophrenia</td>
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<td>July 24</td>
<td>(D)</td>
<td>Oral Health Community Engagement – BSB 451</td>
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<tr>
<td>July 25</td>
<td>(N)</td>
<td>Addiction &amp; Drugs</td>
</tr>
</tbody>
</table>

**Key:**
- Black – mandatory for everyone
- Red or (C) – Cancer track
- Blue or (H) – Cardiovascular track
- Green or (N) – Neuroscience track
- Pink (D) – Craniofacial Biology
Conduct Aim 2: Prostate Cancer Research Training (Year 2, months 6-8)

The Student Fellows participated in an intensive training 10-week program in Prostate Cancer Research. Lectures focused on population science, statistical methods in prostate cancer research, prostate cancer clinical research, and basic science research. Other lectures described funding opportunities available to the students, career development opportunities, qualitative research methods, perspectives of prostate cancer among community members, and tips for preparing graduate school applications. In addition, as prostate cancer is a hormone-related cancer and some of the biological mechanisms that impact the etiology and treatment of prostate cancer are also relevant to breast cancer, the curriculum included information pertaining to breast cancer as well.

The schedule also provided time for students to rehearse their research presentations and gain input from their mentors and other scientists at the HCC. Disparities research was a cross-cutting theme in all of the lectures.

The structure of the curriculum also provides the students with a better understanding of the different population groups that were included in their research. Therefore, cultural enrichment activities were added to the curriculum, such as the Gullah tour of Charleston, in order to expose the students to the local and historic culture of the Charleston population. The Sea Island (Gullah) population is a subpopulation of African Americans indigenous to the coastal regions of the eastern seaboard. They are the most genetically homogeneous group of blacks in the U.S. Their particularly low rate of European American genetic admixture makes this a unique population for basic, clinical and population-based research. The following tables show the Summer 2013 cancer research training curriculum.
# 2013 Breast and Prostate Cancer
## Summer Undergraduate Research Curriculum

**May 28, 2013 - August 2, 2013**

<table>
<thead>
<tr>
<th>Week</th>
<th>Topic</th>
<th>Potential Instructor</th>
<th>Location and Date</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>WEEK 1</strong></td>
<td>Welcome and Overview of the Training Program</td>
<td>Leadership, Mentors and Planning Team</td>
<td>Tuesday, May 28, 2013</td>
</tr>
<tr>
<td><strong>WEEK 1</strong></td>
<td>Anatomy and the Function of the Breast</td>
<td>Rita Kramer, M.D., Associate Professor Hematology / Oncology</td>
<td>Wednesday, May 29, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 2</strong></td>
<td>Controversies in Breast Cancer Screening</td>
<td>Madeleine Lewis, M.D., Assistant Professor Radiology</td>
<td>Tuesday, June 4, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 2</strong></td>
<td>Hollings Cancer Center Outreach Mobile Unit &amp; Community Compass</td>
<td>Melanie Sian, MS, Juanita Brunson, MS Outreach Coordinators</td>
<td>Thursday, June 6, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 3</strong></td>
<td>Anatomy and the Function of the Prostate</td>
<td>Harry S. Clarke, M.D., Ph.D., Professor Urology Services</td>
<td>Monday, June 10, 2013 3-4pm BE402</td>
</tr>
<tr>
<td><strong>WEEK 3</strong></td>
<td>Funding Opportunities for Underrepresented Minority Scholars</td>
<td>Joann F. Sullivan, Ph.D., Assistant Dean for Extramural Program Development</td>
<td>Tuesday, June 11, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 3</strong></td>
<td>Epidemiologic Issues in Prostate Cancer Research</td>
<td>Anthony Alberg, Ph.D., Professor Cancer Prevention &amp; Control Program</td>
<td>Thursday, June 13, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 3</strong></td>
<td>Controversies in Prostate Cancer Screening</td>
<td>Jonathan Picard, M.D., Assistant Professor Urology Services</td>
<td>Tuesday, June 18, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 3</strong></td>
<td>Cultural Enrichment Event</td>
<td>Cultural Enrichment Event</td>
<td>Wednesday, June 19, 2013</td>
</tr>
<tr>
<td><strong>WEEK 4</strong></td>
<td>Epidemiologic Issues in Breast Cancer Research</td>
<td>Joan Cunningham, Ph.D., Research Assistant Professor Public Health Sciences</td>
<td>Thursday, June 20, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 4</strong></td>
<td>Biostatistical Issues in Breast and Prostate Cancer Research</td>
<td>Elizabeth Garrett-Mayer, Ph.D., Professor Public Health Sciences</td>
<td>Tuesday, June 25, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 5</strong></td>
<td>Community-based genetic research project among the Sea Islanders (Gullah) in SC</td>
<td>Ida J. Spruill, Ph.D., Assistant Professor College of Nursing</td>
<td>Thursday, July 11, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 5</strong></td>
<td>Improving Graduate School Admission Rates</td>
<td>Cynthia F. Wright, Ph.D., Associate Dean for Admissions and Career Development</td>
<td>Monday, July 1, 2013 BE402</td>
</tr>
<tr>
<td><strong>WEEK 5</strong></td>
<td>Qualitative Research Methods</td>
<td>Charlene Pope, Ph.D., Associate Professor College of Nursing</td>
<td>Tuesday, July 2, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 6</strong></td>
<td>Vitamin D and Prostate Cancer</td>
<td>Sebastiano Gattoni-Colli, M.D., Professor Radiation Oncology</td>
<td>Thursday, July 11, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 6</strong></td>
<td>Genetic Basis of Cancer</td>
<td>Dennis Watson, Ph.D., Professor Pathology &amp; Laboratory Medicine</td>
<td>Tuesday, July 9, 2013 BE 402</td>
</tr>
<tr>
<td><strong>WEEK 7</strong></td>
<td>“Receptor crosstalk leading to cancer cell invasion”</td>
<td>Steven Rosenzweig, Ph.D., Professor Pharmacology</td>
<td>Tuesday, July 16, 2013 BE402</td>
</tr>
</tbody>
</table>

**Legend:**
- **Core Course**
- **Breast Cancer Course**
- **Prostate Cancer Course**
<table>
<thead>
<tr>
<th>WEEK 8</th>
<th>Cultural Enrichment Event</th>
<th>Cultural Enrichment Event</th>
<th>Thursday, July 18, 2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>WEEK 8</td>
<td>Research Presentation Rehearsals</td>
<td>All Research Students and mentors</td>
<td>Tuesday, July 23, 2013 BE402</td>
</tr>
<tr>
<td>WEEK 0 (Rehearsals)</td>
<td>Research Presentation Rehearsals</td>
<td>All Research Students and mentors</td>
<td>Thursday, July 25, 2013 BE402</td>
</tr>
<tr>
<td>WEEK 0</td>
<td>Research Presentation Rehearsals</td>
<td>All Research Students and mentors</td>
<td>Tuesday, July 30, 2013 BE402</td>
</tr>
<tr>
<td>WEEK 10</td>
<td>Evaluations and Closeout Program</td>
<td>All Research Student and Staff</td>
<td>Wednesday, July 31, 2013</td>
</tr>
</tbody>
</table>

- **CORE COURSE**
- **BREAST CANCER COURSE**
- **PROSTATE CANCER COURSE**
(c) Sponsor the Student Fellows’ Participation in a Graduate Record Examination (GRE) course 
(Year 2, months 6-8)

In 2013, all four Student Fellows took the 10-week Princeton Review GRE Test Preparation Course. The Princeton Review is a standardized test preparation company. The course met on Wednesday evenings from 5:30 pm – 8:30 p.m. The course seamlessly adjusts classwork and homework to the skill level of each student. This is accomplished by focusing on the areas where each student needs the most improvement. The course provides instruction in test-taking skills, and provides opportunities for dynamic group discussions and collaborative drills.

Task 2 Deliverables: In 2013, state-of-the-art comprehensive prostate cancer research education and training opportunities were provided for 4 students from two of South Carolina’s HBCUs. Funds were leveraged from other federally funded training grants to provide the same level of education and training to an additional 3 students from HBCUs in South Carolina. We are developing a cadre of scientists who are prepared to play a significant role in discovering and testing new prostate cancer biomarkers. In the future, these investigators will likely conduct research spanning the continuum from basic science to clinical science to population-based research.

Task 3. Prepare Tangible Scientific Products
(a) Prepare and present scientific abstracts based on the Student Fellows’ prostate cancer research (Year 2, months 10-12)
(b) Prepare manuscripts that will be submitted to peer-reviewed journals (Year 2, months 10-12)
(c) Develop manuscripts to describe the scope and outcomes of the project (Year 2, months 9-12)

In 2013, each Student Fellow prepared a scientific research paper that will form the basis of a peer-reviewed publication. The Student Fellows are completing manuscripts with their research mentors. Each Student Fellow gave a scientific presentation based on the results of his or her work.

In addition, Ms. Franshawn Mack gave a presentation on November 15, 2013 at the Southeast Regional Research Conference in Little Rock, AR. The title of her presentation was “Evaluating the Reliability of an Instrument Assessing Cancer Clinical Trial Perceptions in a Predominantly African American Sample in South Carolina.” She was also a co-author of the following presentation:
Summaries of each Student Fellows’ research projects are included in Appendix C. A manuscript describing the scope and outcomes of the Training Program will be initiated in the spring of 2013.

**Deliverables:** A total of 9 scientific presentations were made by the Student Fellows, including two presentations at national scientific meetings.

**Task 4. Evaluate the Training Program**

(a) **Assess the number of applicants to the Training Program (Year 2, months 1-4)**

In the spring of 2013, 16 students from South Carolina’s HBCUs applied to the DOD South Carolina Collaborative Undergraduate HBCU Student Summer Training Program. As planned, four Student Fellows were selected who were funded through the DOD South Carolina Collaborative Undergraduate HBCU Student Summer Training Program enrolled in the Training Program in the summer of 2013. An additional three Student Fellows were selected. Their participation in the Training Program was supported through leveraged funds from a DOD Southeastern Virtual Institute for Health Equity and Wellness grant and an NIH/NCI P20 South Carolina Cancer Disparities Research Center grant.

(b) **Assess the number of Student Fellows who apply to graduate school (Year 2, months 1-12)**

The Student Fellows who participated in the 2013 DOD South Carolina Collaborative Undergraduate HBCU Student Summer Training Program were rising sophomores through seniors. As described below, we are employing several strategies to monitor the Student Fellows’ progression through their academic careers.

(c) **Assess the number of Student Fellows who are admitted to graduate school (Year 2, months 1-12)**

We are actively keeping track of the progress of the Student Fellows using the strategies that are described below.

(d) **Assess the number of graduate schools to which Student Fellows are admitted (Year 2, months 1-12)**

(e) **Employ several tracking mechanisms to monitor the scientific progress of the students, including:**

   1. **Searching the MUSC graduate program databases to identify whether any of the students applied, were offered, or accepted positions at MUSC.**
   2. **Contacting the participating universities’ alumni offices.**
   3. **Employing other internet based search tools/communications (Google, MySpace, Facebook, and Historically Black College/University Connections, etc.) to identify students’ current locations, contact information, and academic achievements (Years 2, 3, and beyond)**

We have implemented several steps for tracking student scientific progress. Communication and assistance from the Associate Directors and Faculty Advisors have proved to be very effective. Additionally, social media tools such as Facebook have also been useful for engaging the students and opening a venue for communication. Another method we have found useful is text messaging. We have found that students respond more quickly to text messages than to emails and telephone calls. We will utilize and build upon these methods to improve continued student tracking. These multiple tracking strategies will be used to update the table that is included in Appendix D, which lists the academic accomplishments of the Student Fellows.
(f) Identify the number of scientific abstracts presented and peer-reviewed publications that result (Year 2, months 10-12)

The Student Fellows gave a total of 9 scientific presentations, including two presentations at national scientific meetings. The mentors of the Student Fellows have confirmed that manuscripts that include some of the Student Fellows as co-authors are underway.

**Deliverables:** The Student Fellows are completing their sophomore and junior years of college and will apply to graduate or professional schools. The Student Fellows gave a total of 9 scientific presentations, two of which were made at two national scientific meetings. Also, each year, we ask the Student Fellows to evaluate the Training Program. The results from the 2013 Student Fellows are presented in the following table.
## SUMMARY RESULTS OF STUDENTS EVALUATIONS 2013 (n=7)

<table>
<thead>
<tr>
<th>Survey Item</th>
<th>Strongly Disagree</th>
<th>Disagree</th>
<th>Not Sure</th>
<th>Agree</th>
<th>Strongly Agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Overall, the summer program was a good research experience.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>2. The summer program helped me learn the fundamentals of breast and prostate cancer and research.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>3. The Princeton Review Graduate Record Examination (GRE) Course was effective in helping me to learn GRE test preparation strategies.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>4. The seminar schedule was convenient.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. The seminar topics were of interest to me.</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>6. Participating in the program helped to strengthen my desire for a career in cancer research.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. The Program Assistant (Ms. Hazelton) was accessible and assisted me when needed.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>8. My research mentor was accessible and assisted me when needed.</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>9. I would recommend this program to other students at my college/university.</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>6</td>
</tr>
</tbody>
</table>
REPORTABLE OUTCOMES

**Student Summer Research Summaries**

Each Student Fellow prepared a research paper and gave a scientific presentation to their peers, mentors and other faculty at MUSC. Details regarding the manuscripts and scientific presentations developed by the Student Fellows are included in Appendix C.
CONCLUSIONS

During past year of funding of the DOD South Carolina Collaborative Undergraduate HBCU Student Summer Training Program, the tasks outlined in the Statement of Work were successfully met. Twelve Student Fellows were recruited from Claflin University, SC State University, and Voorhees College. Each Student Fellow conducted research and prepared a research paper that was presented at the conclusion of the program. The Student Fellows also presented their work at national conferences and were included as co-authors on peer-reviewed scientific publications, based on their summer research.

As shown in the following tables, two additional students participated in the DOD South Carolina Collaborative Undergraduate HBCU Student Summer Training Program using funds leveraged from another DOD grant that was funded in 2010 (DOD Grant Number W81XWH-10-2-0057, Southeastern Virtual Institute for Health Equity and Wellness). The DOD SE VIEW grant provided funding for two additional students per year beginning in 2010.
<table>
<thead>
<tr>
<th>Student’s Name</th>
<th>Institution</th>
<th>MUSC Research Mentor</th>
<th>Research Title</th>
<th>Research Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Janielle Samuel</td>
<td>Voorhees College</td>
<td>Dr. Marvella E. Ford</td>
<td>Testing protein glutathionylation levels in MCF7 breast cancer cells expressing glutathione S-transferase Pi Isoforms</td>
<td>GSTpi has been implicated in the forward reaction of S-glutathionylation. Therefore, we are interested in understanding how polymorphism may alter cellular responses for both oxidative and nitrosative stress. As such, the four alleles of GSTpi have been transfected into MCF7 breast cancer cells and we are testing the rate and extend the S-Glutathionylation via western blot analysis.</td>
</tr>
<tr>
<td>Edward McMorris</td>
<td>Voorhees College</td>
<td>Dr. Christina Voelkel-Johnson</td>
<td>Acid ceramidase overexpression and its role in the activation of and addiction to Akt signaling in prostate cancer</td>
<td>Previous studies have demonstrated the role of the ceramide metabolizing enzyme acid ceramidase in promoting an aggressive cancer phenotype in prostate cancer cell lines. In addition, it has been found that greater than 80% of prostate tumors overexpress acid ceramidase, suggesting that acid ceramidase may be an important mediator of development and progression of prostate cancer. In this study, we demonstrate that the increased rate of proliferation in acid ceramidase overexpressing cells is dependent on signaling through the oncogenic PI3K/Akt pathway. In addition, we found that acid ceramidase overexpressing cells are more sensitive to Akt inhibition than control cells, suggesting that acid ceramidase overexpressing tumors are addicted to Akt signaling. These findings highlight the importance of investigating the Akt pathway as a potential therapeutic target in acid ceramidase overexpressing tumors.</td>
</tr>
<tr>
<td>Student’s Name</td>
<td>Institution</td>
<td>MUSC Research Mentor</td>
<td>Research Title</td>
<td>Research Summary</td>
</tr>
<tr>
<td>--------------------</td>
<td>-------------------</td>
<td>----------------------</td>
<td>---------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>CoDanielle Green</td>
<td>SC State University</td>
<td>Dr. Marvella E. Ford</td>
<td>Evaluating an intervention to increase cancer knowledge in racially diverse communities in South Carolina; as well as, the increase in cancer knowledge’s effect on cancer prevention activities.</td>
<td>To conduct a cancer education intervention with racially diverse communities in South Carolina. Then, to assess the impact that the cancer knowledge intervention is having on the cancer prevention activities of the residents.</td>
</tr>
<tr>
<td>De’Angelo Dinkins</td>
<td>SC State University</td>
<td>Dr. Christina Voelkel-Johnson</td>
<td>Thioredoxin 1 as a Therapeutic Target in Advanced Prostate Cancer</td>
<td>Prostate cancer is the 2nd leading cancer in men after lung cancer. Indolent disease can be treated fairly well and progresses slowly. However, the more aggressive form of prostate cancer spreads throughout the body and there are no curative treatments. We tested the hypothesis that increased expression of redox proteins is an underlying cause for the aggressive, therapy-resistant prostate cancer phenotype. In our project we looked at the expression of redox proteins and susceptibility to chemotherapy in ARCaPe and ARCaPm cells.</td>
</tr>
</tbody>
</table>
Ernest Just Scientific Symposium
February 22, 2013

Located in the James E. Clyburn Research Center Auditorium

Part 1: Introduction
8:00-9:00 am
Registration and Breakfast - Entrance to Auditorium

Opening: Stephen Lanier, Ph.D.
Associate Provost for Research
Professor of Pharmacology, Medical University of South Carolina

Etta D. Pisano, M.D., Dean, College of Medicine
Vice President for Medical Affairs, Medical University of South Carolina

Greeting: Perry V. Halushka, Ph.D., M.D.
Professor, Pharmacology and Medicine
Dean, College of Graduate Studies, Medical University of South Carolina

Title: "The Creativity of Ernest Everett Just"

9:10 - 9:40 am
William McDade, M.D., Ph.D.
Deputy Provost for Research and Minority Issues
Office of the Provost
University of Chicago

Part II: Role Models
9:40-10:10 am
Title: "Finding it in ALL Academic Medicine"
Samantha E. Kaplan, M.D., MPH
Assistant Professor of Obstetrics & Gynecology
Assistant Dean for Diversity & Multicultural Affairs
Director, Early Medical School Selection Program
Boston University School of Medicine

10:10-10:30 am
Break

Just Symposium Keynote
10:35-11:15 am
Title: "Science: A Powerful Tool for Justice"
Griffin P. Rodgers, M.D.
Director, National Institute of Diabetes & Digestive & Kidney Diseases
National Institutes of Health

Graduate Presenter
11:20-11:30 am
Title: "What's Wrong with my Heart? Improving left ventricular function following Myocardial Infarction"
Presenter: Denise Kimbrough, PhD candidate
Medical University of South Carolina

Molecular and Cellular Biology & Pathobiology
Department of Cardiology
Gazes Cardiac Research Institute

Undergraduate Presenter
Title: "Paralysis Due to Caffeine at the Neuromuscular Junction"
Presenter: Ms. Melissa Carr-Reynolds
Spelman College

28
11:45-12:55 pm  
**BREAKOUT SESSIONS/Lunch**
Campus tour for visiting students, Undergraduate Advisors meet with MUSC College Admissions Officers
(Drug Discovery Bldg. Rm 111)

### Part III Science

**1:00-1:50 pm**

**Title:** *The role of iRhom2/ADAM17 in EGFR receptor signaling and TNF-dependent pathologies*

**Carl Blobel, M.D., Ph.D.**

Professor: Departments of Medicine & Physiology & Biophysics
Center for Vascular Biology
Weill Cornell Medical College

**Title:** *Creating the Optimal Environment: Biomaterials in Regenerative Medicine*

**Jennifer Elisseeff, Ph.D.**

Professor of Ophthalmology and Biomedical Engineering
Director, Translational Tissue Engineering Center (TTEC)
Wilmer Eye Institute and Department of Biomedical Engineering
John Hopkins University

**Title:** *CANCER - The Close Cousin of Wound Healing*

**Kapil Mehta, Ph.D.**

Professor of Experimental Therapeutics Cancer Medicine (Biochemistry) The University of Texas MD Anderson Cancer Center
Appendix B: Ernest E Just Symposium Student Attendees
<table>
<thead>
<tr>
<th>Name of School</th>
<th># Students Who Participated in the February 22, 2013 Ernest E. Just Symposium at MUSC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anderson University</td>
<td>18</td>
</tr>
<tr>
<td>Benedict College</td>
<td>40</td>
</tr>
<tr>
<td>Bowie High School</td>
<td>1</td>
</tr>
<tr>
<td>Charles Herbet Flowers High School</td>
<td>11</td>
</tr>
<tr>
<td>Claflin University</td>
<td>14</td>
</tr>
<tr>
<td>Clark Atlanta University</td>
<td>23</td>
</tr>
<tr>
<td>Clemson University</td>
<td>6</td>
</tr>
<tr>
<td>Coastal Carolina University</td>
<td>6</td>
</tr>
<tr>
<td>Fayetteville State University</td>
<td>21</td>
</tr>
<tr>
<td>Gwynn Park High School</td>
<td>13</td>
</tr>
<tr>
<td>Lowcountry AHEC</td>
<td>7</td>
</tr>
<tr>
<td>Morehouse College</td>
<td>7</td>
</tr>
<tr>
<td>Savannah State University</td>
<td>6</td>
</tr>
<tr>
<td>Spelman</td>
<td>18</td>
</tr>
<tr>
<td>The Citadel</td>
<td>2</td>
</tr>
<tr>
<td>UMBC</td>
<td>4</td>
</tr>
<tr>
<td>Upstate AHEC</td>
<td>17</td>
</tr>
<tr>
<td>USC Aiken</td>
<td>4</td>
</tr>
<tr>
<td>USC Upstate</td>
<td>6</td>
</tr>
<tr>
<td>Voorhees</td>
<td>13</td>
</tr>
<tr>
<td>Winthrop University</td>
<td>3</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>240</strong></td>
</tr>
</tbody>
</table>

- **HBCU in SC**
- **HBCU in a Geographic Region Outside of SC**
Appendix C: Summaries of Students’ Abstracts from the
2013 Summer Research Program
Keira Addison

Redox Signaling is deregulated in Breast Cancer

Dr. Danyelle Townsend, PhD
Abstract

Reactive Oxygen Species (ROS) releases oxidative stress in cells which disturbs cellular immunity in the body leading to an unbalanced cellular environment and cancer. Factors that influence ROS are radiation, UV exposure, other environmental factors and the mitochondria in cells. When cells have high levels of oxidative stress, there are antibodies that are released to detoxify the cells, balancing out the cellular environment. Redox signaling is the process of reducing oxidative stress through the release of antibodies and the opening of different signaling pathways. In this work we studied the differential expression of antibodies (Thioredoxin, Sulfriridoxin, GSTπ and Peroxiredoxin) in breast cancer (MCF-7) and normal breast cells (MCF-10) by western blots. Our results show that the antibodies are expressed more in normal cells than breast cancer cells. According to these preliminary results, redox signaling is deregulated in breast cancer cells.
Redox Signaling is Deregulated in Breast Cancer

Keira Addison
Mentor: Danyelle Townsend, Ph.D.
Summer Undergraduate Research Program
What is Redox Signaling?

- Reactive Oxygen Species are high levels of oxygen that can pose cancerous risks to cells

- When a cellular environment has high levels of reactive oxygen species, it triggers the redox signaling process

- Redox signaling is the reduction of oxidative stress by the activation of antioxidants that work to provide a balanced cellular environment
**S-Glutathionylation**

- Post-translational modification on cysteine residues that alters structure / function / subcellular localization in response to oxidative or nitrosative stress

\[
\begin{align*}
\text{SH} & \quad \text{Cys} & \quad \text{[O]} & \quad \text{S-OH} & \quad \text{Cys} & \quad + \text{GSH} & \quad \text{S-SG} \\
\text{SH} & \quad \text{Cys} & \quad \text{+ GSSG} & \quad \text{S-SG} & \quad \text{Cys} & \quad + \text{GSH}
\end{align*}
\]
Hypothesis

- Redox signaling is deregulated in breast cancer.

Aim

- Evaluate the enzymes involved in redox signaling in normal and breast cancer cell models
**GSTπ, Sulfiredoxin, Peroxiredoxin, Thioredoxin**

- **GSTπ (Glutathione S-transferase)**
  - Catalyzes S-glutathionylation reactions

- **Sulfiredoxin (Srx)**
  - Catalyzes S-deglutathionylation of proteins
  - Catalyzes reversal of sulfenic acid residues (Prdx)

- **Peroxiredoxin (Prdx)**
  - Protects cells from oxidative stress by reducing hydrogen peroxide
  - Regulates cells proliferation

- **Thioredoxin (Trx)**
  - Plays a role in de-glutathionylation and de-nitrosylation of cysteine residues
  - Inhibitor of apoptosis
Cell Model of Normal and Cancer Breast Cancer Patients \(\sim 75\% \text{ ER}^+\)

**MCF-7**
- Breast Cancer cells by the Michigan Cancer Foundation
- Expresses estrogen receptors and responds to anti-estrogen therapy

**MCF-10**
- Normal breast cells
- No estrogen receptors present
Materials & Methods

1. Culture and incubate cells at 37°C
2. Harvest cells with Trypsin
3. Extract proteins from cells with 1X Lysis Buffer
4. Odyssey Blocking Buffer 1 hour at room temperature
5. Fast transfer of proteins on gel to PVDF membrane
6. Gel Electrophoresis using 25μg of sample in a 12% gel, 115V for 1 hour
7. Add primary antibodies, incubate overnight at 4°C
8. Add secondary antibodies, incubate 2 hours at room temp.
9. Scan on Odyssey Scanner
Materials & Methods

Stripping Membrane

Two 5 minute washes in PBST → Incubate in stripping solution for 20 min @ 50°C → Wash 4 times in PBST for 10 minutes

Incubate in Primary Antibody and continue Western Blot Protocol → Block for 1 hr room temperature
Results

Expression of GSTπ in Normal and Breast Cancer Cells

Expression of Trx in Normal and Breast Cancer Cells
Results

Expression of Prx in Normal and Breast Cancer Cells

Expression of Srx in Normal and Breast Cancer Cells

Peroxiredoxin

Sulfiredoxin

22kDa

14kDa
Conclusions

- MCF7 breast cancer cells do not express detectible levels of GSTP where as “normal” MCF10 have high levels
  - MCF7 cells are likely to have diminished enzyme mediated S-glutathionylation reactions
- MCF7 breast cancer cells have significantly lower levels of Thioredoxin relative to normal
  - De-nitrosylation / glutathionylation is potentially impaired
- Sulphiredoxin and Peroxi-redoxin levels are not changed in MCF7 breast cancer cells

** Enzymes involved in the S-glutathionylation signaling pathway are blunted in MCF7 breast cancer cells **
Acknowledgements

- Dr. Danyelle Townsend and Leticia Reyes, Lab Manager
- Dr.’s Townsend, Tew and Uys lab members
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Evelyn Martinez  
Cancer Research  
Dr. Rosenzweig  

Introduction  

Head and Neck squamous cell carcinoma (HNSCC) is the sixth most common cancer worldwide, with over 600,000 new cases each year. In the United States alone, HNSCC accounts for 10,000 cancer related deaths per year ("All About Head & Neck Cancer," n.d.). Anatomically, head and neck cancers are found above the collar bone in the oral cavity, pharynx, mouth, and tongue (Rothenberg & Ellisen, 2012). Squamous cell carcinoma accounts for 90 percent of the malignant tumors found in the head and neck region, starting as a malignancy of the squamous cells which are the flat cells found in the upper layers of the skin like the lining of the nose, mouth, and throat. Environmental factors such as chronic tobacco use and excessive alcohol consumption are known to induce HNSCC. Evidence also shows that Human Papilloma Virus (HPV), particularly type 16 and 18 are linked to oropharyngeal cancer ("Squamous Cell Carcinoma," 2012). Prognosis variables of HNSCC include the presence of distant metastases and presence of lymph node metastases. Although there are multiple aggressive treatments for HNSCC such as surgery, chemotherapy, and radiation therapy, nearly 50 percent of patients with advanced disease have recurrences (Chung et al., 2004).

Epithelial-mesenchymal transition (EMT) has been associated with therapeutic resistance and contributes to tumor growth, invasion, and metastases (Krisanaprapornkit & Iamaroon, 2012). It is a process in which epithelial cells lose their cell to cell adhesion, restructure the cytoskeleton, and take on a mesenchymal phenotype. Epithelial cells line the human body and are characterized as closely packed cells connected together by cell junctions and play a role in diffusion and secretion (Ananth, 2013). Mesenchymal cells are spindle-shaped cells that only interact with each other through focal points.
Epithelial cells express a high level of E-cadherin while mesenchymal cells express N-cadherin. As epithelial cells undergo EMT there is a loss in E-cadherin and an increase in N-cadherin known as “cadherin switching.” This gives transformed epithelial cells mesenchymal-traits such as loss of cell adhesion, allowing them to more effectively invade nearby structures.

Vascular endothelial growth factor (VEGF) and endothelial growth factor (EGF) have been suggested to stimulate EMT in oral squamous cell carcinoma (OSCC). VEGF is a signaling protein that stimulates vasculogenesis, blood vessel formation during embryonic development, and angiogenesis. Because cancers cannot grow without an adequate blood supply, cancers that overexpress VEGF are able to grow and metastasize (Lucas et al., 2010). VEGF binds to and activates a receptor tyrosine kinase (VEGFR) through transphosphorylation ("VEGF Signaling Pathway," nd). Once activated, the VEGFR induces processes common to growth factor receptors including cell migration and proliferation.

Many articles state that VEGF and EGF have been widely accepted to stimulate migration and metastasis in pancreatic and colon cancer. In this study, we hypothesized that VEGF and EGF stimulate EMT in OSCC in order to initiate metastasis. Cells from human tongue oral cancer cell lines (SCC9) and laryngeal cancer cell lines (SCC22A) were treated with VEGF and EGF. Western blot analyses were used to determine changes in the expression of the molecular markers, E-cadherin and N-cadherin, of EMT after growth factor treatment.
Growth Factor Contribution to Epithelial Mesenchymal Transition

Evelyn Martinez
Mentor: Dr. Rosenzweig
Department of Pharmacology
SURP 2013
Head and Neck Squamous Cell Carcinoma (HNSCC)

- 6th most prevalent cancer worldwide – 600,000 new cases each year

- What is squamous cell carcinoma?

- Etiologic factors:
  - Chronic tobacco smoking
  - Excessive alcohol consumption
  - HPV-type 16 and 18
Epithelial-Mesenchymal Transition (EMT)

- EMT: process in which epithelial cells lose their cell to cell adhesion
  → restructure the cytoskeleton → take on a mesenchymal phenotype

- Epithelial Cells –
  - Closely packed cells connected by cell junctions that line the human body

- Mesenchymal Cells-
  - Spindle-shaped cells that only interact with each other through focal points

clincancerres.aacrjournals.org
Cadherin Switching

N-cadherin
- Cell adhesion molecule that induces
- scattered morphology
- higher motility, invasion, & metastasis

E-cadherin
Cell adhesion molecule involved in
- regulating cell-cell adhesion
- mobility
- proliferation of epithelial cells
GROWTH FACTORS

- Biological factors
  - Regulate the division and proliferation of cells
  - Influence growth rate of some cancers

- Vascular Endothelial Growth Factor (VEGF)
- Epithelial Growth Factor (EGF)

http://www.amepc.org/tgc/article/view/76/68
Hypothesis

- To determine if VEGF and EGF stimulate EMT in oral squamous cell carcinoma (OSCC) in order to initiate metastasis

- Specific Aim:
  - To identify changes in the expression of the molecular markers of EMT after growth factor treatment using Western blot analyses
Methods

Cells from human tongue oral cancer cell line (SCC9) and laryngeal cancer cell line (UM-SCC22A) were split into a 6-well plate

Treated with 100 ng/mL of VEGF and EGF at 6 different time points
Extraction of protein

Lysis buffer → Sonicate cells → Protein assay
Western Blot

1. Load 40 mg of protein
2. Run gel
3. Transfer onto membrane
4. Block with 5% milk TBST
5. Probe with primary, incubate overnight
6. Wash
7. Probe with secondary
8. Expose blots
9. Strip and repeat steps 4-8 for N-cadherin and Actin
Results-VEGF

SCC9

E-cadherin → 130 kDa

Actin → 40 kDa
Results-EGF

SCC9

UM-SCC22A

E-cadherin 130 kDa

Actin 40 kDa
Conclusion & Discussion

- VEGF and EGF did not stimulate EMT and induce migration in our oral cancer cell lines
- Literature states VEGF and EGF stimulate migration and metastasis
  - Concentration of VEGF and time points were the same as literature
- Variables:
  - Cell density too high/too low
  - Contamination we didn’t catch
Future Direction

- Snail, a transcription factor, has been involved in the progression of tumors by regulating E-cadherin
  - Suppresses expression of E-cadherin

Membranes could be probed with snail to possibly stimulate EMT
Acknowledgements

- Dr. Rosenzweig
- Casey Holmes and the Rosenzweig lab
Acknowledgements (cont...)

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  - Dr. Ford
  - Ms. Brown-Guion
The Effect of Vitamin D3 on T cell Activation and Death

Name: Tomesha Nesbitt
MUSC Summer Undergraduate Research Program
Mentor: Dr. Mehrotra
Date: July 31, 2013
Abstract: Vitamin D plays an important role in the human body. It helps the body absorb the calcium and phosphate needed. In humans, the most important compounds in this group are vitamin D3 also known as cholecalciferol and vitamin D2, which is also known as ergocalciferol. Vitamin D3 stops the growth of T cells. Active T cells up regulate vitamin D and non-active T cells do not. In T cells, vitamin D expression is triggered through engagement of T cell receptor leading to activation of an mitogen- activated protein kinase pathway, and the expression of vitamin D in T cells correlates with greater T cell responsiveness. Up regulation of vitamin D, like CD69 is an early response to stimulation that occurs in T cells. Vitamin D3 stops the growth of T cells; vitamin D3 also has the ability to decrease T cell activation. Vitamin D regulates the expression of more than 900 genes involved in a wide array of physiology functions.
The Effect of Vitamin D3 on T Cell Activation and Death

Name: Tomesha Nesbitt
Program: Student Undergraduate Research
Mentor: Dr. Mehrotra
Date: July 31, 2013
Introduction

- Vitamin D3, also known as cholecalciferol is one of the most important compounds in humans. [Hollick et.al., Mayo Clinic Proc., March 2006]
- Vitamin D3 inhibits differentiation, maturation, and functions of dendritic cells leading to impaired immune responses. [Song et.al., The Journal of vitamins and hormones, 3, 1 May 2003, Pages 235-247]
- Vitamin D3 exerts a marked inhibitory effect on adaptive immune cells. [UK essays, Immunomodulatory Effects Of Vitamin D Health Essay, 2003-2013]
Introduction Continue

- Vitamin D3 (VD3), the most physiologically relevant form of vitamin D, is synthesized in the skin from 7-dehydrocholesterol, a process which depends on sunlight. [Mora et al., The Journal of Nat Rev Immunology, 2008 September; 8]

Experimental Aim

• To elucidate the effect of vitamin D3 on the T cells activation and death by using various cellular assays.
Hypothesis

- Vitamin D3 will suppress T cell activation.
- Vitamin D3 will enhance the death of T cells.
Methods and Materials

![Diagram of JURKAT CELLS process]

4 x 10^6 cells

0.5 x 10^6 cells

24 hrs

+ Phytohaemagglutinin (PHA)

- Phytohaemagglutinin (PHA)

Bar chart showing the results of the experiment.
Experiment 1

CD69

0nM VD3 10nM VD3 100nM VD3

+PHA

46.1 62.6 65.3

6.31 7.70 11.1

Experiment 1

Cell number

Stimulated
Unstimulated

0nM VD3 10nM VD3 100nM VD3
Experiment 2

Experiment 3
Experiment 3
Experiment 4

0nM VD3

10nM VD3

+PHA

-PHA

CD69

Bar graph showing the comparison between stimulated and unstimulated conditions for 0 nM VD3 and 10 nM VD3.
Results

- Vitamin D3 enhanced the up-regulation of Cluster of Differentiation 69 (CD69) in activated T cells.
- Vitamin D3 caused a reduction in cell number of activated T cells at higher doses.
Conclusion

- Vitamin D3 does not suppress T cell activation.
- Vitamin D3 enhances death in activated T cells.
The study helped me understand the regulation of T cell activation and death by Vitamin D3.
Acknowledgements

- Dr. Shikhar Mehrotra
- Dr. Krishnamurthy Thyagarajan (TK)
- Mrs. Stephanie Brown-Guion
- Dr. Marvella Ford
- Ms. Tonya Hazelton
- Ms. Juanita Brunson
- All the lab techs
Examining the AGE-RAGE Signaling Axis as a Mechanism of Prostate Cancer Disparity

Sadia M. Robinson, Dion Foster, David P. Turner

Department of Pathology & Laboratory Medicine

Abstract

Nationally, African American prostate cancer patients are two and a half times more likely to die of prostate cancer than their European counterparts. However in the State of South Carolina, minority African Americans are three times more likely to die from prostate cancer. It is now apparent that a racial disparity in cancers exists due to molecular variances in tumor biology as well as consequence of stress, socioeconomic and environmental problems.

Glycation is the non enzymatic glycosylation of sugar moieties to macromolecules which produces vastly reactive metabolites known as advanced glycation end products (AGE’s). Elevated AGE levels drive the serious complications observed in diabetes and Alzheimer’s patients and AGE’s are now emerging as possible intermediaries of cancer. Cancer and dietary sugars are possible mechanisms of cancer health disparities because of associated biological and socioeconomic links. Research studies have been found that lack of exercise and high fat and sugar filled diets aid greatly to the aid of AGE pools. Foods containing abundant AGE’s promote obesity and men who are obese are more likely to die because of prostate cancer than thinner men. A higher proportion of African American are overweight or obese and do not exercise compared to European American men.

Harmful effects of AGE’s are facilitated in part through its transmembrane receptor RAGE (receptor for advanced glycation end products) which can activate signaling cascades promoting signaling pathways such as NFkB and AKT. This increases excretion of pro-inflammatory cytokines and increases oxidative stress which both promote aggressive cancer.
“The difference in the incidence, prevalence, mortality, and burden of disease and other adverse health conditions that exists among specific population groups”
Cancer Health Disparities

- Overall, African Americans are more likely to develop and die from cancer than any other racial or ethnic group.

- In South Carolina African American men are at the highest risk to be diagnosed and die of prostate cancer in America.
What are Advanced Glycation End Products?

- Endogenous AGE’s are reactive metabolites produced during natural metabolism via glycosylation

- Exogenous AGE’s are derived from our diet, and environmental factors such as tobacco smoke

- High levels of AGEs are implicated in many chronic diseases
What is Glycation?

Advanced Glycation End Products (AGEs) are the end products of glycation reactions in glycosylation.

PROTEINS \(\rightarrow\) SUGARS \(\rightarrow\) GLYCATED PROTEINS

NH₂ \(\rightarrow\) HC=O

ADVANCED GLYcation END PRODUCTS

RESTROOMS
AGE’s are in our Food

Exogenous AGEs are also accumulated in the body through the ingestion of food, smoke and alcohol

High fat foods

Heavily cooked foods

Smoking

Sugary foods

Alcoholic Beverages

Red Meats
AGE content in western diets has steadily increased over the last 50 years.
AGE’s and Human Diseases

- Heart Disease
- Stroke
- Hypertension
- Atherosclerosis
- Pathogenic
- Diabetes
- Insulin resistance
- Hyperglycemia
- Cataracts
- Macular degeneration
- Alzheimer’s
- Kidney & Renal failure
- All diseases associated with health disparity in African Americans
- Cancer
What is RAGE?

- AGE metabolites are ligands for the RAGE receptor resulting in the activation of signaling pathways.
- RAGE is a 35kDa polypeptide transmembrane receptor of the immunoglobulin superfamily.
- Increased expression of RAGE is known to promote multiple cancer types, i.e., Breast Cancer, Prostate Cancer.

Diagram:
- 1: AGE binds to RAGE
- 2: NFkB activated
- 3: Tumor cell
- 4: Pro-inflammatory cytokine release

Pro-inflammatory phenotype
Increased RAGE activation is a consequence of elevated AGE levels and is a biological mechanism promoting prostate cancer disparity
Specific Aims

- Develop molecular models with which to target RAGE expression in prostate cancer
- Examine the effects of targeting RAGE expression on cancer associated pathways
- Examine the effect of AGE treatment on cancer associated pathways
Tumor AGE assessment

Control  EA-LG  AA-LG

Normal prostate

![Bar graph showing average tissue fluorescence (AU) for different conditions.](chart.png)
Tumor RAGE assessment

Normal prostate

EA-LG

AA-LG

EA-HG

AA-HG

Average tissue fluorescence (AU)

0 1000000 2000000 3000000 4000000 5000000 6000000

EA-LG AA-LG EA-HG AA-HG
Methods- to assess AGE & RAGE in cell lines

a) Dot Blot experimentation used to examine cell lines DU145 and PC3
b) shRNA used to reduce RAGE expression levels
c) Mammalian expression vectors to increase RAGE expression in prostate cancer cell lines
d) To assess RAGE protein reduction and increased expression, western blot analysis
Methods – to examine cancer associated pathways

a.) Sulforhodamine B (SRB) proliferation assays were used to assess cell growth
b.) Transwell assays will be used to assess cell migration
Dot Blot Results

1= PC3; 2= DU145; 3=CWR22rv1; 4= Buffer control
5= WPMY1; 6= VCaP; 7= LNCaP; 8= LNCaP C4-2

Dot blot quantification
Targeting RAGE Expression

**RAGE shRNA mediated knockdown**

PC3 W7

RAGE

GAPDH

PC3 cells

**RAGE overexpression**

Exogenous

GFP, 0.025 μg

GFP, 0.05 μg

GFP, 0.1 μg

RAGE, 0.025 μg

RAGE, 0.05 μg

RAGE, 0.1 μg

Endogenous

RAGE

GAPDH

PC3 cells
Cell growth & migration assays

PC3 shRAGE SRB assay

- 878
- 528
- 165
- wild type

PC3 shRAGE Migration Assay

p = <0.0001 compared to WT
AGE treatment

PC3 WT 100ng/ml AGE 10ng/ml AGE

pAKT

AKT

99
Summary

1) AGE’s are reactive metabolites that accumulate in our organs and tissues as we grow older
2) They accumulate endogenously during normal metabolism and exogenously through the foods we eat
3) AGE accumulation is associated with diseases associated with growing older
4) Due to Western diets, dietary AGE’s now contribute significantly to the accumulation pool and therefore disease phenotypes
5) Our work shows that:
   - AGE levels are elevated in cancer tissue and cell-lines
   - Highest AGE levels are observed in African American cancer patients
   - RAGE promotes cancer associated processes
Conclusion

- The AGE-RAGE interaction is important in prostate cancer development, and inhibition of this interaction has potential as a new molecular target for cancer therapy or prevention.
- As AGE and RAGE levels are highest in African American cancer patients, the AGE-RAGE signaling axis may be a mechanism of cancer disparity.
AGE Reduction = Disease Prevention

Raw chicken
800 AGE kU/100g

Poached chicken
1,000 AGE kU/100g

Fried chicken
8,000 AGE kU/100g

Big Mac
7,801 AGE kU/100g

Bacon, fried 5min, no oil
91,577 AGE kU/100g

Chicken Nuggets
8,627 AGE kU/100g
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- Dion Foster
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- South Carolina State University
Bobbi Blake
Mentor: Dr. Jennifer Wu
Funding Source: DOD Southeastern Virtual Institute for Health Equity and Wellness (PI: Slaughter; Project PI: Ford)
NKG2D Signaling Pathways Analysis

Hypothesis
Stimulation of NK cell lines with purified soluble MIC will lead to NK cell activation in a dose-dependent manner as measured by pAKT and DAP10/12 expression.

Introduction
The response of natural killer cells are initiated by cellular surface receptors and regulated by signaling pathways that function in activation or inhibition. The balances between the signals are the difference between cytokine production or immune escape. Activation of NK cells occurs through the interaction of NKG2D receptor and MIC molecules. NK cell kill cells that lack MIC “self-recognition” receptors (Lanie, 2003).

Previous studies have shown positive correlation of patients with different tumors and high level of soluble MIC in serum samples (Roda-Navarro & Reyburn, 2009). This relation suggests the occurrence of immune evasion.

Two targets, pAKT and DAP10/12 have been identified to indicate cell proliferation. pAKT, protein kinase b is a main factor in cellular survival pathways in which it has the ability to inhibit apoptosis and promote the production of growth factors. DAP10/12 is an adaptor on the NKG2D receptor that aids in costimulation and enhances activation (Jiang, Zhong, & Ritchey, 2002).

In this study, NK cells are exposed to soluble MIC cells and pAKT and DAP10/12 expression is examined. The impact of soluble MIC molecules on positive and negative signaling in NK cells is a promising area in understanding the mechanisms of NK cell homeostasis.
NKG2D Signaling Pathways Analysis

Bobbie Blake
Mentor: Dr. Jennifer Wu
Summer Undergraduate Research Program
Natural Killer (NK) Cell

• Cytotoxic lymphocytes in the immune system
• Antibody-dependent cell-mediated cytotoxicity
• Cytolytic granule mediated cell apoptosis
• Missing 'self' hypothesis
  – NK cells recognize the lack of self-ligands on infected cells or cells undergoing other types of stress

Tumor cell surveillance
NK Cell Function
NK Cell Receptors

- NKG2D receptor activates NK cells after engaging ligands induced by cellular stress

Vivier et al., Science Jan 2011
NKG2D Receptor and MIC Ligand

- Activating receptor expressed by
  - All NK cells
  - Most NKT cells
  - Certain subsets of γδ T cells
  - All human CD8+ cells
  - Activated mouse CD8+ cells
  - Activated macrophages
  - IFN-producing killer DCs

- Distinct ligands and signaling adaptors (DAP10/12) for human and mouse
- NKG2D receptor binds to MIC ligands
- MICA - MHC class I related polypeptide sequence A
Soluble MIC in Cancer

- Cancers adopt diverse strategies for immune evasion to ensure their survival.
- Epithelial tumors shed soluble MIC in later stages of cancer.
- Shedded MIC = serum sMIC
Signal Pathway of NKG2D

• Proteins of Interest
  – DAP10-adaptor proteins, which activate signaling pathways for cytotoxicity and cytokine production
  – AKT-pAKT, protein kinase b is a main factor in cellular survival pathways in which it has the ability to inhibit apoptosis and promote the production of growth factors.
Hypothesis and Specific Aims

• Hypothesis
  – Stimulation of NK cell lines with purified soluble MIC will lead to NK cell activation in a dose-dependent manner as measured by pAKT and DAP10/12 expression.

• Specific Aim 1: Determine whether sMIC lead to AKT phosphorylation in NK cell lines NKL and NK-92MI

• Specific Aim 2: Determine if changes in AKT phosphorylation occur through the NKG2D signaling protein DAP10/12
Methods

• Cell Culture – NKL and NK-92MI cell line will be cultured in a 1 x $10^6$ x 1 ml per well, 24 well plate
  – NKL and NK-92 cells were cultured in the presence of IL-2 and during the experiment IL-2 was removed.
• Cell Harvest- Cells were exposed to soluble MIC at the concentrations 0, 50, 100 ng/ml and harvested at 1, 4, 24 and 48 hour time points.
  – Cells were lysed for proteins in the presence of protease and phosphatase inhibitors
• BCA Protein Assay- Quantify protein concentrations
• Western Blotting- Quantify expression of AKT and DAP10 proteins at varied time points of exposure to sMIC
Expected Results

DAP10 Proteins
• Increase expression of proteins with increase in sMIC concentration and time

Akt Proteins
• Decrease in expression of phospho Akt protein with an increase in sMIC concentration and time.
Future Directions

• The current study involves NK cell interacting with soluble ligand
• Future studies will involve NK cell interacting with tumor cell expressing soluble ligand.
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• MUSC Hollings Cancer Center
Franshawn Mack
Mentor: Dr. Marvella Ford
Funding Source: DOD Southeastern Virtual Institute for Health Equity and Wellness (PI: Slaughter; Project PI: Ford)
Evaluating the Reliability of an Instrument Assessing Cancer Clinical Trial Perceptions in Predominantly African American Populations in South Carolina

Franshawn Mack  
Dr. Marveilla E. Ford  
Dana Burshell  
Wei Wei  
Dr. Elizabeth Garrett-Mayer  
July 19, 2013

**Background:** African Americans (AA) are disproportionately affected by cancer mortality compared to their European American (EA) counterparts. While greater participation in cancer clinical trials among AA could help to reduce this disparity, negative perceptions of trials may play a role in negatively impacting trial participation in this population.

**Objective:** To evaluate the reliability of the Attitudes towards Randomized Trial Questionnaire (ARTQ) in assessing perceptions of cancer clinical trials in predominantly AA populations in South Carolina (SC). The ARTQ was developed in Europe and has not yet been tested for use in an AA sample.

**Methods:** Principal Component Analysis and Cronbach’s alpha estimates were used to assess the reliability of the ARTQ in a convenience sample of 315 participants (81.4% AA), from 2008 to 2013, who lived in SC counties with high racial disparities in cancer mortality rates.

**Results:** Slightly more than half of the participants had at least a college diploma (60.8%), 84.8% were female, and 53.4% had an annual income of $40,000 or more. In this study, Cronbach’s alpha for the ARTQ was 0.86.

**Conclusion:** The ARTQ displayed strong evidence of high statistical reliability. This analysis has great implications for future research because it represents the first test of reliability of the ARTQ in a predominantly African American sample and lays the groundwork for use of the ARTQ in future studies in diverse populations.
Evaluating the Reliability of an Instrument Assessing Cancer Clinical Trial Perceptions in a Predominantly African American Sample in South Carolina

Franshawn Mack, Rising Sophomore, South Carolina State University
2013 MUSC Summer Undergraduate Research Program
Funding Source

Mentor: Dr. Marvella Ford
Grant: Department of Defense- Southeastern Virtual Institute for Health Equity and Wellness (DOD-SE VIEW)
Presentation Outline

Introduction
  - Statement of Problem
  - Purpose of Study
Methods
  - Study Sample
  - Measures
  - Analysis
Results
  - Demographic Characteristics
  - Instrument Responses
  - Reliability Analysis
Discussion
Conclusions
Acknowledgements
Introduction
Introduction (continued)

For the majority of cancer types African Americans (AA) have the highest cancer mortality rate of any other racial or ethnic group in the United States. This is also true in South Carolina.
Cancer Mortality For South Carolina
County: All Counties In South Carolina, 2008

Breast Cancer

Prostate Cancer

All Cancers

Lung Cancer

Colorectal Cancer
Statement of the Problem

- Despite being disproportionately impacted by cancer, AA participate in cancer clinical trials less frequently than European Americans (EA).
- Negative perceptions of clinical trials could affect AA participation.
Conceptual Framework

Behavioral Beliefs → Attitude Toward the Behavior → Behavior


In order for health disparities research to be conducted in a meaningful manner, it is important to determine first whether measures developed among nonminority populations perform in the same way when applied to minority populations (Ramirez et. al 2005)
The Attitudes to Randomized Trial Questionnaire (ARTQ) is widely used in Europe to assess perceptions of trials. However, the reliability of this instrument has never before been tested in an AA sample, so its applicability in this population is unclear.
Reliability in this context refers to the repeated use of an instrument, over time, with consistent results.
Purpose of the Study

Purpose: To evaluate the reliability of the ARTQ in a predominantly AA sample. This is the first study to do so

Hypothesis: The ARTQ will show evidence of high statistical reliability
Methods
Study Sample

- Study participants were residents in South Carolina communities with high racial disparities in cancer mortality rates
- 17 sites in 11 counties
- Majority of counties along the I-95 Corridor
- Male or female
- Any race or ethnicity
- Ages 21 years or older
Study Sample (continued)

- The study sample participated in an educational program using a National Institutes of Health PowerPoint presentation that describes cancer clinical trials
  - 30-minute presentation focusing on cancer clinical trials information
- The study investigators modified the NIH presentation to make it more culturally appropriate:
  - Inclusion of lay language
  - Use of images with ethnically/racially diverse people
  - Review of previous atrocities (Tuskegee Syphilis study)
  - Description of safeguards developed to protect trial participants
Study Sample (continued)

- The clinical trial education program was part of a larger evidence-based, 4-Hour Cancer Education Program developed by the South Carolina Cancer Alliance
- Incorporated a Train the Trainer Design and a Pre-Test/Post-Test Design
Measures

❖ Pre-test data, interviewer-administered
❖ General sociodemographic information
  ❖ Age, race, education level, income
❖ 7-Item Attitudes to Randomized Trial Questionnaire (ARTQ) (Fallowfield et al. 1998)
❖ Responses include Yes, No, DK
Measures (continued)

Fallowfield Study (1998): Development of the ARTQ
- Seven-item instrument used to assess perceptions of cancer clinical trials
- Consecutive sample of 323 patients with cancer in the United Kingdom (UK)
- 315 patients completed the ARTQ, unassisted
Measures (continued)

Summary of the 7-item ARTQ Questions:
Response Categories: Yes, No, Don’t Know

1. Do you think patients should be asked to take part in medical research?

Would you be prepared to take part in a study:
2. comparing different treatments?
3. where the treatment was chosen at random?
Measures (continued)

Would you be encouraged to take part in a randomized study:

4. where either treatment would be suitable for you?
5. if you could leave the study if the treatment was not suitable for you?
6. if before you agreed to participate, your doctor would tell you all about both treatments being compared?
7. If you knew all of the following things were taken into account, would you change your mind and agree to take part in the study?

- Both treatments were completely suitable
- You could leave the study if the treatment did not suit you
- There was plenty of information before the random choice was made
Analysis

Reliability Analysis

- Principal Component Analysis (PCA) was used to evaluate the dimensionality of the ARTQ
- Cronbach’s alpha was used to measure internal consistency, indicating survey reliability
Results
Demographic Characteristics
(N=315)

Gender* (n=211)
- Majority of the study participants were female (84.8%)

Race* (n=296)
- Most participants were African American (81.4%)
- Compared to the state population of 0.5% Native American/Alaskan Native (NA/AN), our study included a significant population of NA/AN (5.1%)

Racial Distribution of Participants*

* = missing data
Demographic Characteristics (N=315)

Education* (n=298)
- More than half of the study participants had at least a college degree (77.9%)

Income* (n=286)
- Slightly more than half had an annual household income equal to or greater than $40,000 (53.1%)
Survey Responses

Proportion of Participants Who Responded Yes/No/Don’t Know for the Seven Items in the Attitudes to Randomized Trial Questionnaire
Statistical Tests

- PCA confirmed unidimensionality of the ARTQ
- Cronbach’s alpha=0.86

<table>
<thead>
<tr>
<th>Cronbach's alpha</th>
<th>Internal consistency</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\alpha \geq 0.9$</td>
<td>Excellent (High-Stakes testing)</td>
</tr>
<tr>
<td>$0.8 \leq \alpha &lt; 0.9$</td>
<td>Good (Low-Stakes testing)</td>
</tr>
<tr>
<td>$0.7 \leq \alpha &lt; 0.8$</td>
<td>Acceptable (Surveys)</td>
</tr>
<tr>
<td>$0.6 \leq \alpha &lt; 0.7$</td>
<td>Questionable</td>
</tr>
<tr>
<td>$0.5 \leq \alpha &lt; 0.6$</td>
<td>Poor</td>
</tr>
<tr>
<td>$\alpha &lt; 0.5$</td>
<td>Unacceptable</td>
</tr>
</tbody>
</table>
Discussion
Discussion

Study results show the ARTQ to be reliable in this predominantly AA South Carolina sample
Conclusions

This is the first study to employ the ARTQ to evaluate perceptions of trials in a predominantly AA sample - the majority of studies using the ARTQ were conducted in Europe with primarily EA samples.
Conclusions (continued)

- Additional testing of the ARTQ is required in other samples of diverse population groups from different geographic regions of the US to confirm the reliability of the instrument in these groups:
  - Male vs. Female
  - Low Education Level vs. Higher Education Level
  - Low income Level vs. Higher Income Level
  - Subgroups of AA (e.g., Sea Islands, Haitians, Nigerians)
  - Latinos/Hispanics
  - Non-English speaking populations in the U.S. who have low trial participation rates
Acknowledgements

Dr. Marvella Ford
Ms. Dana Burshell
Mr. Wei Wei
Dr. Elizabeth Garrett-Mayer
Mrs. Tonya Hazelton
Ms. Juanita Brunson
Mrs. Stephanie Brown-Guion
MUSC Summer Undergraduate Research Program
MiR-204 Negative Regulation of IGF2R as a Mechanism Driving Breast Cancer Disparity

Jasmine Fox, South Carolina State University

Victoria Findlay, Ph.D., Mentor

2013 MUSC Summer Undergraduate Research Program (SURP)

Abstract

Breast cancer accounts for 22.9% of all cancers in women in the world (http://breastcancersymptoms.net). Approximately 1 in 8 women will develop breast cancer, and it causes 13.7% of cancer death in women. In the US, African American (AA) women have a significantly higher rate of mortality due to BC compared to Caucasian American (CA) women.

MicroRNAs are small non-coding RNAs that function to silence gene expression by translational repression or mRNA target degradation (2). The microRNA of interest in this study is miR-204: we identified the Insulin Growth Factor 2 Receptor (IGF2R) as a direct target of miR-204. The IGF-2R is a multifunctional receptor that binds IGF-II resulting in its internalization and degradation via lysosomes. The IGF-2R does not have tyrosine kinase/signaling activity, instead it functions to sequester IGF-II away from the IGF1R signaling pathway, resulting in its proposed role as a tumor suppressor.

Studies have shown that the levels of IGF2R are lower in AA women compared to CA women with breast cancer. To assess whether miR-204 levels are disparate in AA women with breast cancer, we performed real time PCR in serum samples from 20 patients: 10 AA and 10 CA. Our analysis shows that there is a significant increase in the levels of miR-204 in the AA breast cancer patients when compared to the CA patient samples. We have previously shown that miR-204 over-expression results in an increase in migration and invasion. Therefore, to assess whether this miR-204 mediated increase is through the negative regulation of the IGF2R we transfected miR-204 expressing and scrambled control breast cells with the IGF2R ORF. IGF2R levels were confirmed by western blot analysis. We found that expression of exogenous IGF2R was able to restore invasive levels of the cells back to control level. We also performed immunofluorescence to demonstrate that IGF2R when exogenously expressed was localized to the cell membrane. These studies are being optimized.
MIR-204 NEGATIVE REGULATION OF IGF2R AS A MECHANISM DRIVING BREAST CANCER DISPARITY

Jasmine Fox
Mentor: Victoria Findlay, Ph.D.
Summer Undergraduate Research Program (SURF) 2013
Breast Cancer Overview

- Breast Cancer (BC), is the development of cancerous tumors in the breast tissue that arise from epithelial cells
- Breast cancer accounts for 22.9% of all cancers in women in the world
- Approximately 1 in 8 women will develop breast cancer
- BC causes 13.7% of cancer death in women
Breast Cancer Disparities

- AA women have the highest BC death rates of all races and ethnic groups
- EA women have the highest incidence rate for BC, but AA are most likely to die from BC
- AA women often have fewer socioeconomic resources than other women such as:
  - Lack of medical coverage
  - Barriers to early detection/screening

Who has health coverage?
Percent of Americans with health coverage, by race

- Whites: 88%
- Asian Americans: 82%
- Native Hawaiian or other Pacific Islander: 79%
- African Americans or blacks: 79%
- Hispanics: 68%
- American Indians and Alaskan Natives: 68%

Note: Percentages for Native Hawaiian or other Pacific Islander and American Indian and Alaskan Native is based on 2005–2007 data; all other percentages based on 2009 data.
The Sea Island Population of SC

- African Americans from the Sea Islands of SC are direct descendants of blacks from the “rice or windward coast” of West Africa
- The Sea Island descendants are primarily from the country of Sierra Leone
- The Sea Island population is genetically and culturally distinct due to previous geographic isolation and low rates of genetic admixture
- The low rates of admixture in the Sea Island population makes them uniquely positioned to allow genetic studies
MicroRNAs (miR-204) Background

- MicroRNAs are small non-coding RNAs that function to silence gene expression by translational repression or mRNA target degradation

- The microRNA of particular interest in this study is MicroRNA 204 or miR-204

- miR-204 is a novel oncomir

- Over-expression of miR-204 in immortalized non-transformed and non-invasive breast cancer cell lines results in increased migration, invasion, and cellular transformation
IGF2 and IGF2R Background

- Insulin Growth Factor 2 Receptor (IGF2R) has been identified as a novel direct target of miR-204
- Insulin growth Factor 2 receptor (IGF2R) is a transmembrane receptor that binds IGF-2, resulting in its degradation via internalization and transport to lysosomes
- IGF-II is a mitogenic and anti-apoptotic peptide
- IGF-II influences the proliferation of various cell types including normal and transformed breast epithelial cells
IGF2R in Cancer

- Low levels of IGF2R expression were shown to correlate with poor patient prognosis in breast cancer patients and a recent study showed significantly higher levels of IGF2R in EA compared to AA tumor samples
Key components of the IGF-1R pathway.
Hypothesis

- We hypothesize that miR-204 mediated negative regulation of the IGF2R is a mechanism promoting breast cancer disparity.
1) To evaluate the levels of miR-204 in serum samples from AA and EA women with breast cancer
2) To demonstrate that miR-204 mediated increase in migration is through the negative regulation of the IGF2R
Specific Aim 1 - Methodology

- Transfection with different ratios of Transfection Reagent:IGF2R plasmids
- Transfection in MCF10A cells stably transfected with scrambled and miR-204
- Protein extractions using RIPA buffer to lyse the cells and run on western blot:
  - the levels of IGF2R using protein specific antibody
- RNA Extraction, Reverse Transcription and Real Time PCR
- Migration assay
- Immunofluorescence assay - localization
MCF10A Transfection Optimization

Screening of Transfection Reagent, Ratio = µl Reagent:µg DNA
DNA= pmiR-EV (cherry)

Ratio 1:1

Ratio 2:1

Ratio 3:1

Ratio 4:1

Ratio 5:1

Ratio 6:1
MCF12A stables

scr

miR-204

IGF2R  |  Hoescht  |  2' only

166
IGF2R “Rescue”

Migration

<table>
<thead>
<tr>
<th>Condition</th>
<th>Average # cells/field</th>
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<tbody>
<tr>
<td>NC</td>
<td>5</td>
</tr>
<tr>
<td>miR-204</td>
<td>20</td>
</tr>
<tr>
<td>IGF2R</td>
<td>10</td>
</tr>
<tr>
<td>miR-204/IGF2R</td>
<td>15</td>
</tr>
</tbody>
</table>

p < 0.04
p < 0.001

Western Blot

IGF2R
EV
GADPH
Specific Aim 2 Methodology

- Serum samples were obtained from the HCC tissue Biorepository
- 10 EA & 10 AA (3 non-SI & 7 SI)
- RNA extraction from serum samples using Trizol
- miR reverse transcription
- Real Time PCR assay
miR-204 levels are disparate in serum from breast cancer patients

serum samples

\[ p < 0.05 \]

SI separation

\[ p = 0.09 \]
\[ p = 0.04 \]

non-Si: Si

3:7
CONCLUSIONS

- We demonstrated that miR-204 increases migration via direct negative regulation of IGF2R
- We showed that levels of miR-204 expression in serum samples were higher in AA women than EA women
- This decrease in IGF2R expression may contribute to the increase risk of malignant transformation in AA breast cancer patients
Acknowledgements

- Victoria Findlay, Ph.D., mentor
- Lourdes Nogueira
- Qi Guo
- Dr. Marvella Ford
- MUSC’s Summer Undergraduate Research Program (SURP)
- Hollings Cancer Center
Appendix D: Academic Accomplishments to Date of the 2013 Student Fellows
<table>
<thead>
<tr>
<th>Student Name</th>
<th>Summer Research Project</th>
<th>Funding Source</th>
<th>Publications, Presentations and Honors</th>
<th>GRE Status</th>
<th>Graduate School Admission</th>
</tr>
</thead>
</table>
| Ms. Jasmine Fox    | **Research Project:** MiR-204 Negative Regulation of IGF2R as a Mechanism Driving Breast Cancer Disparity | National Institutes of Health/ National Cancer Institute | **Publication:** No publications to date  
**Presentation:** 2013 MUSC Summer Undergraduate Research Program | Has not taken the GRE | Still enrolled at SC State University. |
| SC State University| **Mentor:** Dr. Victoria Findlay                                                         |                                         |                                                                                                       |            |                            |
| Ms. Sadia Robinson | **Research Project:** Examining the AGE-RAGE Signaling Axis as a Mechanism of Prostate Cancer Disparity | Department of Defense (HBCU)            | **Publication:** No publications to date  
**Presentation:** 2013 MUSC Summer Undergraduate Research Program | Has not taken the GRE | Still enrolled at SC State University. |
| SC State University| **Mentor:** Dr. Dave Turner                                                              |                                         |                                                                                                       |            |                            |
| Ms. Tomesha Nesbitt| **Research Project:** The Effect of Vitamin D3 on T cell Activation and Death             | Department of Defense (HBCU)            | **Publication:** No publications to date  
**Presentation:** 2013 MUSC Summer Undergraduate Research Program | Has not taken the GRE | Still enrolled at Voorhees College. |
| Voorhees College   | **Mentor:** Dr. Shikhar Mehrotra                                                         |                                         |                                                                                                       |            |                            |
| Ms. Keira Addison  | **Research Project:** Redox Signaling is deregulated in Breast Cancer                    | Department of Defense (HBCU)            | **Publication:** No publications to date  
**Presentation:** 2013 MUSC Summer Undergraduate Research Program | Has not taken the GRE | Still enrolled at SC State University. |
| SC State University| **Mentor:** Dr. Danyelle Townsend                                                       |                                         |                                                                                                       |            |                            |
| Ms. Franshawn Mack | **Research Project:** Evaluating the Reliability of an Instrument Assessing Cancer Clinical Trial Perceptions in a Predominantly African American Sample in South Carolina | Department of Defense (SE VIEW)         | **Publication:** No publications to date  
**Presentation:** 2013 MUSC Summer Undergraduate Research Program | Has not taken the GRE | Still enrolled at SC State University. |
| SC State University| **Mentor:** Dr. Marvelsa E. Ford                                                         |                                         | **Presentation:**  
| Ms. Bobbie Blake   | **Research Project:** NKG2D Signaling Pathways Analysis                                  | Department of Defense (SE VIEW)         | **Publication:** No publications to date  
**Presentation:** 2013 MUSC Summer Undergraduate Research Program | Has not taken the GRE | Still enrolled at Claflin University |
| Claflin University | **Mentor:** Dr. Jennifer Wu                                                              |                                         |                                                                                                       |            |                            |
| Ms. Evelyn Martinez| **Research Project:** Growth Factor Contribution to Epithelial Mesenchymal Transition   | Department of Defense (HBCU)            | **Publication:** No publications to date  
**Presentation:** 2013 MUSC Summer Undergraduate Research Program | Has not taken the GRE | Still enrolled at SC State University. |
| SC State University| **Mentor:** Dr. Rosenzweig                                                               |                                         |                                                                                                       |            |                            |
| **Southeast Regional Research Conference in Little Rock, Arkansas on November 15-17, 2013 (oral presentation) | | | | | |