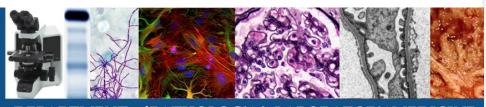
THE PATH WAY

August 2013





Volume 4, Issue 2

DEPARTMENT of PATHOLOGY & LABORATORY MEDICINE



Mary S. Richardson, M.D., D.D.S.

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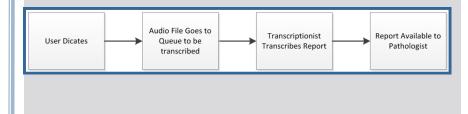
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Voice Recognition in Anatomic Pathology Is it ready for prime time use?

By: James Madory, D.O.

Unfortunately there is not a simple yes or no answer to the question. The answer is dependent on which voice recognition technology will be used and in what areas it will be expected to function. Voice recognition for every aspect of an anatomic pathology type case from gross to sign out will not work in our current workflow. There are, however, areas in anatomic pathology where voice recognition can play a role and increase productivity while decreasing turn-around time. For the purpose of this discussion anatomic pathology type cases include all types of cases in our department utilizing Cerner CoPath as their reporting application.

Currently the dictation software utilized in pathology and laboratory medicine is part of a digital system. The user dictates the case which is recorded as a digital file on a server. Transcriptionists listen to the digital recording and type directly into Cerner CoPath Plus. Benefits of the current system include the capturing of a digital audio file, allowing the user to dictate freely and having transcriptionists perform corrections and formatting of the report.



This newsletter is made possible from the generous contributions of MUSC's Pathology and Laboratory Medicine Faculty and Staff. The success of this publication is dependent upon this support. Thank you for your interest, time and information. For inquiries, suggestions or submission information please contact Lori Roten (roten@musc.edu).

Cover Story Cont'd

Voice recognition of dictated text can be done in two different ways. Front end voice recognition takes place the moment the dictating person is speaking. With front end dictation the voice recognition software translates the speaker's voice almost instantaneously as they speak. The translated text is displayed on the screen. When errors in the translated text are encountered, the speaker corrects the text and formatting as they continue with their work. We have previously used this type of system in our gross pathology areas with variable amounts of success.

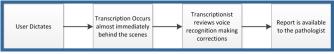
When using front end voice recognition there are two options for dictating the text into the report. With free text dictation, any text dictated by the speaker is translated into text while with templated dictation the speaker dictates answers to the empty spaces within the template. Benefits of free text dictation include the ability of the speaker to use whatever words or phrases they would like. Problems with free text dictation include the need for more accurate voice recognition and the need to format the recognized text. Benefits of templated dictation include uniformity of dictations, reminders of elements to include, and easier recognition on part of the voice recognition engine. Problems with templated dictation are usually



found with the cases that do not fit nicely into a template. It is also possible to combine the free text as well as templated dictation allowing cases that fit into the template to be readily completed while those that do not fit can have free text added.

Front end voice recognition does not come without problems. With front end dictation the user must not only focus on the job they are attempting to complete, but also on the translation of their text on the screen. Often the user spends as much time correcting the dictation as they do working on the case. Attempting to make the corrections by voice commands while working on a case can cause significant frustration. This can result in the user giving up and switching to the keyboard to correct the results by direct entry. By using direct entry the system does not learn from its mistakes and will continue to make them in the future.

The second option for voice recognition in dictated text is known as back end dictation. In back end dictation the speaker dictates into a digital recording analogous to the system in place today. Once the recording has been completed, a server behind the scenes analyzes the digital recording using the voice recognition software. After the text has been analyzed, transcriptionists proof read the recognized text, correct errors in the voice recognition system and then format the text to the correct format. Benefits of this type of system include allowing the dictating user the ability to free text whatever they would like, almost instantaneous initial transcription and invisibility of the voice recognition process to the dictating user. Voice recognition in this type of system does not work well for templated dictation, however those cases can be flagged to go directly to a transcriptionist. With back end dictation the role of the transcriptionist is modified to becoming a proof reader.



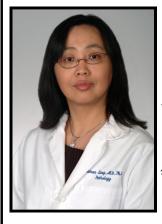
Choosing a voice recognition system for pathology is not an easy process and many decisions will need to be made prior to implementation of a voice recognition system. The Medical University is currently evaluating their current transcription services in the clinical environment as well as areas like radiology and pathology and a discussion on voice recognition will be included. While the technology has improved significantly since we stopped using front end voice recognition in 2006, I do not believe it has improved enough to work with our current workflow without preventing significant workflow delays. Back end recognition may prove to be a little more useful.



PROMOTIONS

WELCOME!

CONGRATULATIONS!



Hainan Lang, M.D., Ph.D. Associate Professor (7/1/13) Angie Duong, M.D. Assistant Professor will join Pathology and Laboratory Medicine on 9/1/13

> Dr. Duong will serve as the Assistant Medical Director in Hematopathology.

Dr. Duong completed her residency at MUSC.

We are excited to be working with her again!

CONGRATULATIONS!

Stephen Guest, Ph.D.

Research Assistant Professor

Promoted from Post Doc to Research Assistant Professor in Dr. Ethier's Lab on 7/1/13. ALL HANDS MEETING

TUESDAY, AUGUST 20TH

9:30-10:30 AM

IN

2W AMPHITHEATRE



MUSC 2-Factor Authentication Information

By: Tony Eisenhart

In order to prevent phishing attacks and further strengthen access to sensitive data, MUSC is implementing two-factor authentication for remote accessto MUSC resources.

Off campus access to MUSC applications like Outlook Web Access (http://exchange.musc.edu), Citrix Virtual Applications (http://webapps.musc.edu) and VPN (http://vpn.musc.edu) will now be protected with two-factor authentication. Our system will automatically contact you when you try to log into a MUSC resource from a computer that is not on the clinlan domain.

If you do not have access to a phone you will need to use a one-time bypass. When you register you will be asked a series of security questions. These security questions are how you will be able to gain access should you lose your phone, not have cell coverage, or if you prefer not to be called to authenticate.

A one-time bypass is completed by going to http://2factor.musc.edu and Logging In with your NetID and password. The system will try to contact your phone and after 2 minutes time out, then your security questions will be presented. Once you have successfully answered your security questions you can select "One-time Bypass" for access.

If you are going to be working from home and utilizing multiple resources at once, utilizing VPN for a secure and continuous connection is recommended.

Further information is available through an FAQ link at https://2factor.musc.edu/PhoneFactor/ and you can contact the OCIO-IS Help Desk at 843-792-9700 any time for assistance.

Mobile Device Management information

Mobile devices are widely used by faculty, staff, students and other authorized individuals to access a variety of MUSC systems that contain sensitive data, including administrative and financial records, educational records, and protected health information. While mobile access can provide valuable benefits, there is a significant risk of unauthorized access to sensitive MUSC data if a mobile device is lost or stolen, or otherwise leaves the control of its owner or authorized user.

Who: Mobile Device Management is required for All users who connect to the MUSC Exchange server (MUSC email) from a smart phone.

Why: By requiring Mobile Device Management (MOM) software on smart phones, the following goals will be achieved:

-Force Password lock

-Require Inactivity Timeout

-Enforce Encryption of MUSC data including email, contacts, and calendar information.

-Push <muscsecure> wireless and VPN settings to our users' devices and remove them if necessary.

-Push Exchange Email, Contacts, and Calendar settings to users devices and remove them if necessary.

-Add the Ability to Wipe, Selectively Wipe MUSC Data only, locate lost devices if users choose to allow their device to be located. The physical security of mobile devices must be maintained at all times. In particular, these devices should not be left unattended in any location where loss or theft, or any access to the device by an unauthorized party, could be a reasonably anticipated and avoidable risk.

If devices are lost, users will have the ability to log into a self-service portal at http://www.musc.edu/myphone and do the following:

-lock their device

-locate their device (If they have location services turned on and GPS enabled on their device)

-Wipe their device completely

-Wipe only MUSC data from the device (Exchange Email, Exchange Contacts, Exchange Calendar, MUSC VPN Settings, muscsecure wireless settings, passcode policy)

Please Note: MDM does NOT allow administrators to see users' private data stored on their phone such as messages in MUSC or personal email accounts, pictures, videos, phone calls, and text messages. MOM does allow administrators to see device data such os version, Battery Life, Phone Number of Device, Location of Device (only with written permission of the user when users allow their device to be located}, Applications installed on the device, and Passcode and Encryption Compliance.

All users who connect to the MUSC Exchange server from their phone will be required to install the Xen Mobile MOM client.

ARRIVALS / DEPARTURES

ARRIVALS:

Yang Zhao, joined Dr. Cheung's Lab as a Research Specialist I on April 16, 2013.

Dorinda Andrews, joined Dr. Zhu's Lab as a Temp-Lab Tech IV on May 20, 2013.

Connor Stanley, joined Dr. Watson's Lab as a volunteer on June 3, 2013.

Matt Paul, joined Dr. Watson's Lab as a volunteer on June 3, 2013.

Jeremy Morgan, joined Dr. Watson's Lab as a volunteer on June 3, 2013.

Ashley Cross, MD, joined Pathology and Laboratory Medicine as a PGY 1 Resident on July 1, 2013.

Kate Eichel, MD, joined Pathology and Laboratory Medicine as a PGY 1 Resident on July 1, 2013.

Jonathan Gullett, MD, joined Pathology and Laboratory Medicine as a PGY 1 Resident on July 1, 2013.

Daniel Skipper, DO, joined Pathology and Laboratory Medicine as a PGY 1 Resident on July 1, 2013.

Mike Stump, MD, joined Pathology and Laboratory Medicine as a PGY 1 Resident on July 1, 2013.

Chris Wenzinger, MD, joined Pathology and Laboratory Medicine as a PGY 1 Resident on July 1, 2013.

Joseph, Bergeron, MD, joined Pathology and Laboratory Medicine as a Cytopathology Fellow on July 1, 2013.

Courtney Ingram, MD, joined Pathology and Laboratory Medicine as a Cytopathology Fellow on July 1, 2013.

Kate Lindsey, MD, joined Pathology and Laboratory Medicine as a Cytopathology Fellow on July 1, 2013.

Jessica Sugianto, MD, joined Pathology and Laboratory Medicine as a Dermatopathology Fellow on July 1, 2013.

Darren Monroe, MD, joined Pathology and Laboratory Medicine as a Forensic Pathology Fellow on July 1, 2013.

Kalli Faulkner, DO, joined Pathology and Laboratory Medicine as a Hematopathology Fellow on July 1, 2013.

ARRIVALS CONT'D:

Lixia Zhang, joined Dr. Moussa's Lab as a Postdoc Scholar on July 22, 2013.

Jianning (Jason) Zhang, joined Dr. Lang's Lab as a Visiting Scholar on August 5, 2013.

Satya Nandana Narla, Ph.D., joined Pathology and Laboratory Medicine as a Clinical Chemistry Postdoctoral Fellow on August 15, 2013

Rachel B. Mariotti, will join Pathology and Laboratory Medicine as a Pathologist Assistant on August 26, 2013.

DEPARTURES:

Xinping Hao, Visiting Scholar, left Dr. Lang's Lab on May 19, 2013.

Christopher Hensley, Research Specialist I, left Dr. Lang's Lab on May 31, 2013.

Ana Medina, M.D., Assistant Professor, left the Department on May 31, 2013.

Julia Kuhnert, Postdoc Scholar, left Dr. Moussa's Lab on June 17, 2013.

David Hurray, MD, Clinical Assistant Professor, left Stephens County Hospital on June 28, 2013.

Ashlyn Boserup, Lab Tech II, left on June 20, 2013.

Carlene Brandon, Research Specialist III, left Dr. Ethier's Lab on June 30, 2013.

Su Zengliu, Clinical Chemistry Fellow, left the Department on June 30, 2013.

Hu Yuan, Visiting Scholar, left Dr. Sha's Lab on June 30, 2013.

Hon-Wei Zheng, Visiting Scholar, left Dr. Sha's Lab on June 30, 2013.



RESEARCH DIVISION UPDATE

Statistics for the Division of Research from April through June. Nine grant proposals were submitted requesting \$1,864,444 in total first year costs. Also, during this period fifteen grants were awarded totaling \$1,954,992.

Congratulations and many thanks to everyone involved in obtaining these awards.

Bradley Schulte, Ph.D., Vice Chair of Research

SUBMITTED 4/1/2013 – 6/30/2013:

Stephen Ethier, Ph.D. Title: Breast Cancer Oncogenes on the 8p11 Amplicon \$344,317 – Proposed Start Date 6/1/13

Victoria Findlay, Ph.D. Title: miR-204 Regulation of Cas-1 as a Mechanism Driving Breast Cancer Disparity \$224,251 – Proposed Start Date 4/1/14

Hainan Lang, M.D., Ph.D.Title: Auditory Nerve Degeneration and Repair\$368,750 – Proposed Start Date 7/10/13

Meenal Mehrotra, Ph.D. Tile: CSSG-Flow Cytometry \$22,094 – Proposed Start Date 4/1/13

Frederick Nolte, Ph.D. Title: Evaluation of the Investigational Simplexa MRSA Direct Assay \$380,000 – Proposed Start Date 7/15/13

Chandrakala Puligilla, Ph.D. Title: MEKK4 Signaling in Sensory Cell and Neuron Formation within the Mammalian Cochlea \$20,000 – Proposed Start Date 6/28/13

Demetri Spyropoulos, Ph.D. Title: A Lung Cryopreservation Approach that Maintains Cell Viabillity and Tissue Architecture \$423,176 – Proposed Start Date 12/1/13

Demetri Spyropoulos, Ph.D. Title: Development of Stem Cell Based Tools to Identify and Measure "Obesogens" in S.C. Coastal Waters \$56,990 – Proposed Start Date 2/1/14

Demetri Spyropoulos, Ph.D. Title: The Role of SOX4 in Triple Negative Breast Cancer Disparity \$24,866 – Proposed Start Date 3/1/14

AWARDED 4/1/2013 – 6/30/2013:

Stephen Ethier, Ph.D.Title: Amphiregulin Signaling in Human Breast Cancer\$69,767—Start Date 5/1/13

Stephen Ethier, Ph.D.Title: Amphiregulin Signaling in Human Breast Cancer\$209,301—Start Date 5/1/13

Stephen Ethier, Ph.D. Title: Breast Cancer Oncogenes on the 8p11 Amplicon \$323,658—Start Date 6/1/13

Hainan Lang, M.D., Ph.D. Title: Auditory Nerve Degeneration and Repair \$350,313 – Start Date 7/1/13

Meenal Mehrotra, Ph.D. Tile: CSSG-Flow Cytometry \$22,094 –Start Date 4/1/13

Meenal Mehrotra, Ph.D. Tile: Role of Hematopoietic Stem Cells in Establishing the Osteosarcoma Microenvironment \$30,000 – Start Date 5/1/13

Omar Moussa, Ph.D. Tile: The Role of Thromboxane A2 (TP) Receptor Beta in Bladder Cancer \$145,472 – Start Date 4/1/13

Frederick Nolte, Ph.D. Tile: Evaluation of the Investigational Simplexa MRSA Direct Assay \$380,000 – Start Date 6/20/13

Chandrakala Puligilla, Ph.D. Title: Role of Sox2 in Specification of Prosensory and Hair Cell Fate in Mouse Cochlea \$12,449 – Start Date 12/1/13

RESEARCH DIVISION UPDATE, continued

AWARDED 4/1/2013 - 6/30/2013:

Su-Hua Sha, M.D.

Title: Molecular Mechanisms in Noise-Induced Hearing Loss \$17,787 - Start Date 4/1/13

Avtar Singh, M.D. Title: Mechanisms of Krabbe Disease Pathobiology and Therapy \$20,973 – Start Date 4/1/13

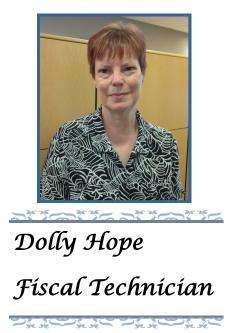
Avtar Singh, M.D. Title: Nitrosylation Mechanisms for Protection Against Neurovascular Inflammatory Injury \$290,391 – Start Date 5/1/13

Dennis Watson, Ph.D.

Title: Building Next-Generation Bioinformatics Cyberinfrastructure for Genomics-enabled Research and Education in the Charleston Scientific Community \$11,680 – Start Date 5/1/13

Je-Seong Won, Ph.D. Title: Development of S-Nitrosothiol-Based Therapy for Alzheimer's Disease \$49,014 – Start Date 5/1/13





Nomination: Thank you very much for your help.

Other Nominees: Eowyn Corcrain, Tony Eisenhart, Christopher Hensley, Jarvis Jenkins, Sonya Jordan, and Linda McCarson

National Medical Laboratory Week 2013







2013~2014 Fellows

CYTOPATHOLOGY



My name is **Joey Bergeron**. I graduated from LSU Medical School in New Orleans, LA in 2009. I then completed my AP/CP pathology training at VCU Medical Center in Richmond, VA. I have been married to my wife, Christina, for just a little over four years. My wife and I are looking forward to living in a beach town where we plan to spend a lot of our free time. We enjoy the outdoors and spend most of our vacations in national parks. I also enjoy drawing when I find enough free time. I very much look forward to training at MUSC in cytopathology and hope for a great year!

My name is **Courtney Ingram**. I was born in Atlanta, Georgia and attended Hampton University in Virginia for my undergraduate degree. I attended Emory University School of Medicine, and initially began a pediatric residency at the University of Alabama in Birmingham. However, as a new



mother, I found it very difficult to see such precious patients deal with such serious illnesses. I subsequently changed my emphasis to pathology and completed my pathology residency at Baptist Health System in Birmingham, Alabama. I met my husband, Carlos Ingram, in high school. We were married while I was in medical school and he was beginning his career as a financial advisor. We have three beautiful daughters, Caley (9), Cameron (7), and Caris (1). We are, without question, <u>extremely</u> busy with three children; but in the rare event that we have spare time, we love to explore new places. Our family is very excited about our move to Charleston, and I am particularly excited to be a part of MUSC!

Kate Lindsey is a native of South Carolina, or as much of a native as an "Air Force Brat" can be. She just completed residency here and is stay-



ing as one of the three Cytopathology fellows this year. The most wonderful news she has to share is the birth of her second child, John Russell. He was born at the end of May, joining his sister Ella Grace. Kate's husband is the Director of Chemistry at a small pharmaceutical company stationed here in Charleston. Kate plans to follow her Cytopathology fellowship with an additional fellowship in Hematopathology at Baylor University Medical Center in Dallas, Texas.

DERMATOPATHOLOGY



I am **Jessica Sugianto**. I grew up in Katy, TX, and went to Texas A&M University. I went to medical school at UT Southwestern Medical Center in Dallas, TX, where I also did my pathology residency. I enjoy playing piano, ballroom dancing, and teaching my mischievous cat tricks. I'm excited about starting my fellowship in Charleston!

FORENSIC PATHOLOGY

I am **Darren Monroe**. I'm from Louisville, KY and obtained a master's degree in mechanical engineering from the University of Louisville. I then went to medical school at the University of Kentucky and stayed there for residency. I was born and raised a UK fan, and I will always bleed blue for the Wildcats, but I'll also pull for UL if they aren't playing UK. My hobbies include fencing epee and foil, swing and ballroom dancing, shooting, playing bass and guitar, riding motorcycles, and ripping phone books in half. I really like pizza, too. I can't wait to explore Charleston and all of its history and am looking forward to moving there and working with all of you! It's not my dog in the picture, but I wish it was.

HEMATOPATHOLOGY

I am Kalli Faulkner. I am originally from Southeast Missouri, where I grew up on a farm in the middle of no-

where. During high school I met my husband, Drew, and we have been inseparable since. I attended medical school in Kansas City, during which we added our amazing daughter, Maddi, to the mix. When it came time for residency, my husband (who is from Georgia) had only one request: South! So, we moved to

Mobile, Alabama and have spent the last four years enjoying the beautiful Gulf Coast. During my spare time I love to paint, especially with my daughter (who is a budding artist). Both of my parents are artists and were a bit surprised when I went to medical school. So, anytime I have the chance, we pull out our easels and Maddi and I add to our collection. I am quickly running out of wall space! We are all excited to move to Charleston for my fellowship...my daughter especially. We spent a week taking her to every kid friendly attraction in the area and as a result, Charleston is a magical, wondrous place in her mind. I am looking forward to working at MUSC and cannot wait to get started!



SURGICAL PATHOLOGY

I am **Keels Allen.** I went to undergrad at Wofford College in Spartanburg South Carolina, before going on to Medical School and residency at the Medical University of South Carolina. I enjoy the daily challenges of surgical pathology, and the chance it gives me to interact with my clinical

colleagues. In my free time I enjoy water skiing, fishing, golf and reading, as well as spending time with my wife Sarah, and our two girls, Ada Cathryn age 3 and McCauley age 1.



Evelyn T. Bruner, MD, is originally from Columbia, SC. She graduated *cum laude* with a Bachelor of Arts degree in Studio Art at the University of South Carolina. She spent several years working as a graphic artist. Dr. Bruner then went on to earn her medical degree at the Medical University of South Carolina in Charleston, SC, graduating in 2009. Dr. Bruner finished her pathology residency training at MUSC this year and began her surgical pathology fellowship in July. She hopes to pursue a career in academic medicine and enjoys teaching medical students and residents. Evelyn is happily married to Michael Bruner, a local fishing charter guide, with two children, a thirteen year old son,

Caleb and a twenty month old daughter, Mattie Mae. She enjoys cooking, spending time with her family, exploring Charleston's beaches and parks and is an avid Gamecock fan.



My name is **Laura Spruill** and I am the subspecialty Gynecologic Surgical Pathology Fellow this year. I am originally from Rochester, NY and I went to college at SUNY Geneseo where I received a BS in biochemistry. After graduation, I started the Medical Scientist Training Program at MUSC and graduated with my MD and PhD in 2008. I then completed residency and a year of general surgical pathology fellowship at MUSC. I am excited to continue the work I started last year and I hope to pursue a career in academic medicine.

I was married a year ago to Pal Suranyi, a diagnostic radiologist at MUSC. We have four children, Kinga (14), Benett (11), Samantha (8) and Luke (5). We spend our family time enjoying Charleston parks or traveling. When not at work or with family, I am trying to learn Hungarian to be able to converse with my new in-laws.

2013~2014 Residents (PGY1)



My name is **Ashley Cross** and I am an incoming PGY-1. I grew up in Virginia Beach and attended the University of Virginia for my undergraduate degree. While there, I met my husband, Mack, a realestate development professional from Winston-Salem, NC. I have enjoyed the last 4 years at Eastern Virginia Medical School in Norfolk, VA. Mack and I have been married for 2 years and have loved being parents to our Cavalier-mix puppy, Sailor. We recently purchased a home in Mt Pleasant, in the same neighborhood where my siblings and I were born. It's thrilling to begin my residency at MUSC, just as my dad did in 1985. I am excited about the

next 4 years in Charleston.



I am **Kate Eichel**. I am originally from Moscow, Russia but have been happy to call South Carolina my home for most of my life. After receiving a BS in Microbiology from Clemson University, I attended medical school at MUSC. My awesome husband Carl and I enjoy ballroom dancing, cooking, wine tasting, sailing, and hiking with our energetic dog Tatze. We are extremely excited to call Charleston our home and look forward to making lots of new friends in the upcoming years!



I am **Jonathan Gullett**. I was born and raised in beautiful Orlando, FL. I received a BS from the University of Florida - Go Gators! (Exercise Physiology), an MS from The College of St. Scholastica (Exercise Physiology), and another BS from the University of Central Florida (Molecular Biology and Microbiology). After several years of research as an independent contractor for a biomedical research center, I attended the University of Central Florida College of Medicine as a member

of its Charter Class. My grandmother, Vernette, lives with me now and will be moving with me for residency. Also, I have two labs, Toby and Rocky. We're all in credibly excited to move to Charles ton and to start a wonderful new chapter our lives!

2013~2014 Residents (PY1), continued



I am **Daniel Skipper**. "I was born and raised in Conway, SC, and received my B.S. in Biology from Coastal Carolina University. Prior to entering medical school, I worked as a firefighter-paramedic in Myrtle Beach, SC. I completed my medical school training in TN at Lincoln Memorial University-DeBusk College of Osteopathic Medicine. I had a very interesting childhood, as I am an identical twin. My brother currently works for the South Carolina Highway Patrol. My wonderful wife Jessica is a middle school math teacher. Outside of medicine, I love to play piano, read, go to the movies, cook, and most of all, eat! I'm very excited to begin the next chapter of my life here at MUSC."



I am **Michael Stump**. I am originally from Greenwood, IN. I graduated from DePauw University in Greencastle, IN, and from there attended Indiana University School of Medicine in Indianapolis. I have a wonderful girlfriend, Jamie Stevenson, whom is a horse trainer and eventer. During my free time, I like to compete in triathlons, trail runs, and road races. Other interests include reading science fiction and adding to my music collection. I cannot wait to explore all Charleston has to offer and to meet everyone at MUSC!

I am **Chris Wenzinger**. I'm originally from the Shenandoah Valley region of Virginia. I graduated from UVA in 2009, and from there moved to Norfolk, VA for medical school. That

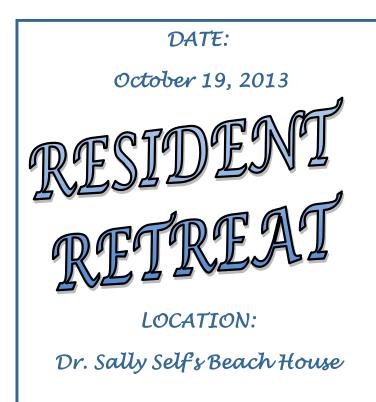
was where I met my fiancé Sarah, who is beginning her internship with Pediatrics. Sarah and I were delighted to learn that we matched at MUSC. We moved to James Island just a few weeks ago and couldn't be happier to be living here. We have had a great time exploring the area, and have especially loved kayaking through the tidal marshes. I am thrilled to be a part of MUSC and look forward to meeting & working with everyone here.

JANE K. UPSHUR POST-SOPHOMORE FELLOW



I am **Tariq Rashid** and I am currently in between my second and third year of medical school here at MUSC. I spent most of my life in Minnesota, only having moved down to South Carolina after college 4 years ago for AmeriCorps in Greer, SC. After AmeriCorps, I spent a year working for Delta Airlines and Kaplan Test Prep teaching the MCA before coming to MUSC for medical school. My hobbies include cooking food from all over the world, biking Charleston, hiking mountains, running, and traveling to new places. My life goal is to use the immense amounts of knowledge gained from schooling and apply it in many contexts, here and abroad. I am incredibly excited for all that I will learn here as a Post-Sophomore Fellow here in the Department of Pathology.







Pathology Residency Update: June 2013

David Lewin, M.D.

July 1 will see six new residents start the pathology residency program. Once again we have a good representation of the southeast US and beyond. One incoming resident is from MUSC (Dr. Kate Eichel), two are from East Virginia Medical School (Dr. Ashley Cross and Dr. Christopher Wenzinger), one from Lincoln Memorial Osteopathic school (Dr. Daniel Skipper), one from University of Central Florida (Dr. Jonathan Gullet), and one from Indiana University (Dr. Michael Stump). A number of the incoming residents have South Carolina roots. Dr. Kate Eichel went to North Charleston High School and Clemson University. Dr. Daniel Skipper is originally from Myrtle Beach, SC and went to Costal Carolina University. Dr. Ashley Cross did a month long rotation at MUSC last year. The average USMLE scores for our incoming residents are 230 Part I and 245 Part 2 (well above the national average of 222 on both parts). Additionally we have our third Jane Upshur Post Sophomore fellow (Tariq Rashid from MUSC) starting in July as well. The first-year residents and post sophomore fellow will begin the year with an introductory month, paired with senior residents in surgical pathology and autopsy. We wish them all well for the start of their careers in pathology.

We have five residents graduating from the residency program and all are going on to fellowship programs:

Dr. Keels Allen: MUSC Selective Surgical Pathology

Dr. Matthew Bernstein: Medical College of George Surgical Pathology

Dr. Evelyn Bruner: MUSC Surgical Pathology Fellowship

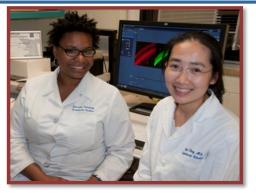
Dr. Kate Lindsey: MUSC Cytopathology Fellowship

Dr. Emily Ogden: Dallas Medical Examiner Forensic Pathology Fellowship

The residency program recently underwent it's reaccreditation visit from the Accreditation Council for Graduate Medical Education (ACGME) and received no educational citations and a 5 year reaccreditation cycle (the maximum). The ACGME will shortly be moving to the Next Accreditation System (NAS) starting in 2014 for pathology. This will potentially extend the site visits to every 10 years, however will require more semiannual reporting from the program regarding the progress of the residents with respect to newly developed milestones and more annual surveys of the residents and faculty.



A Novel Role for Microglia in the Developing Auditory Nerve



LaShardai Conaway and Yazhi Xing, MD

LaShardai Conaway, a first year graduate student, and Yazhi Xing, a Postdoctoral Fellow, from Dr. Hainan Lang's lab, are working collaboratively on a research project regarding the role of macrophages in the onset of hearing.

Hearing is initiated by the mechanical movement and vibration of stereocilia located on cochlear hair cells. These vibrations are then carried across the auditory nerve to the brain where they are interpreted as sound. Normal hearing acquires the establishment of fine wiring pattern between the auditory nerve and hair cells. In the Two types of nerve fibers are present in the mature mammalian ear the auditory nerve. Each inner hair cell is innervated by 20-30 type I fibers, while each type II fiber can innervate several outer hair cells. The proper establishment and maturation of innervation to the hair cells is critical for the onset of auditory function.

In the mouse cochlea the development and refinement of neuronal connection occurs through postnatal day 12, before the onset of hearing, and proceeds in 3 distinct phases. First, the neurites of the afferent fibers elongate and extend to the inner and outer hair cells. Next, the neurites are refined and finally, the neurites undergo retraction and synaptic pruning to eliminate type I spiral ganglion neuron innervation of outer hair cells [1]. The mechanisms underlying auditory nerve refinement and pruning are currently unknown.

Microglia, a subset of glial cells, are resident macrophages in the central and peripheral nervous systems and are responsible for debris clearance and eliciting inflammatory responses. During development, microglia are actively surveying surrounding cells and responding to changes within their microenvironment. In the mature ear, resident microglia become inactive or "resting," only converting to an active phenotype when stimulated by pathogens or the presence of cellular debris and apoptotic cells [2]. With late age, microglia can become dysfunctional and hyperactive, resulting in neuronal degeneration. This process has been linked to the onset of many age-related neurodegenerative disorders. Although the phagocytic role of microglia in the nervous system has been documented, the mechanism of microglial dysfunction remains unknown. Further understanding of the role of microglia during development is critical for understanding their dysregulation with old age.

In the central and peripheral nervous systems, physical association between microglia and synapses is observed in the retina and several locations of the brain during development. A recent article by Paolicelli et. al. (2011), reported that microglia are involved in the synaptic pruning of developing dendrites in the mouse hippocampus [3]. This was the first study to demonstrate that microglia play a role in the sculpting of connectivity in the central nervous system. Additionally, microglia also have been shown to sculpt the neural circuits in the retina in an activity-dependent manner [4]. These events are similar to the neural refinement that occurs in the cochlea, suggesting that microglia could play a role in auditory nerve maturation.

The focus of Dr. Hainan Lang's lab is the investigation of mechanisms associated with auditory nerve degeneration and regeneration. One ongoing project in our lab is to determine the role of microglial cells during auditory nerve development and maturation.

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Our hypothesis is that microglia, which are present in the auditory nerve, are involved in the refinement of the postnatal auditory nerve and play a role in auditory nerve degeneration in the aged cochlea.

Our investigations of the postnatal auditory nerve have lead to the observation of a unique pattern of microglia expression during auditory nerve maturation. Immunohistochemistry to detect microglial cells was performed in two cochlear structures. Rosenthal's Canal and the osseous spiral lamina, at postnatal days 0, 3,7,14, and 21 [Figure 1]. This study reveals that the peak of IBA-1⁺ cell presence corresponds to the timepoint of neurite retraction during auditory nerve refinement. Microarray analysis further revealed a unique pattern of microglia-associated gene expression during cochlear development. These findings suggest that microglia play an important role in the refinement and maturation of the auditory nerve. Interestingly, we have seen an increase in microglia expression between the young adult and aged mouse cochlea, suggesting that microglia may be contributing to the spiral ganglion neuronal degeneration characterized in sensorineural hearing loss with age.

Future studies will be aimed at answering the following questions: are microglia necessary for proper refinement of the auditory nerve? What are the proliferation patterns of microglia during development and in the aged cochlea. Is increased expression of microglia in the aged cochlea contributing to the neuronal loss associated with sensorineural hearing loss?

References:

- 1. Huang L, (2012). Synaptic profiles during neurite extension, refinement and retraction in the developing cochlea. Neural Dev. 7:38.
- 2. Ekdahl CT, (2012). Microglial activation- tuning and pruning adult neurogenesis. FPHAR 3:1-9.
- Paolicelli, RC and Gross, CT (2011). Microglia in development: linking brain wiring to brain environment. Neuron Glia Biol. 7: 77-83.
- Schaffer, DP et. al. (2012). Microglia sculpt postnatal neural circuits in an activity and complement-dependent manner. Neuron 74 (4): 691-705.

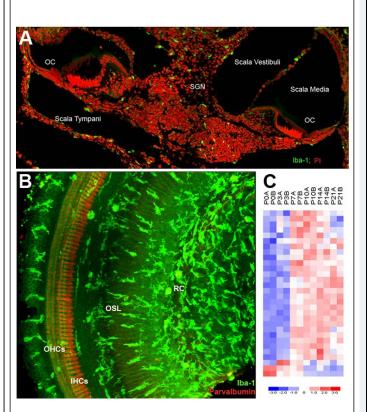


Figure 1. Macrophages/microglia cells engraft in the postnatal day 7 mouse cochlea. A. Immunohistochemistry reveals the presence of macrophages/microglial cells in the middle turn of the postnatal day 7 cochlea. Microglia were labeled with IBA-1, shown in green, and nuclei were counterstained with propidium iodine, PI. Scale bar: 25µm. **B** . Numerous IBA-1⁺ cells are found throughout a P7 cochlea. The cochlear specimen was processed for whole-mount preparation and stained with Iba-1 (green, a macrophage/microglial marker) and parvalbumin (red, an inner hair cell marker). C. Heat map of differentially expressed macrophage/microglia-associated genes. Targeted genes shown have differential expression between P0 and P7 samples with an expression fold change greater than 1 1/2 and Student *t*-test of *p*<0.05. **OC**: organ of Corti; SGN: Spiral Ganglion Neuron; RC: Rosenthal's Canal. **OSL**: Osseous Spiral Lamina; OHCs: Outer Hair Cells; IHCs: Inner Hair Cells



AGE'S AND DISEASE: YOU ARE WHAT YOU EAT !!

David P. Turner, Ph.D.

Abnormal metabolism is a driving factor at the center of many of the modern day epidemics in America today including obesity, diabetes, cardiovascular disease, metabolic syndrome and cancer. Research in the Turner lab is centered upon examining the link between the reactive metabolites found in our diet with disease phenotypes. Advanced glycation end products, or AGE's as they are known, are a diverse group of reactive metabolites produced during normal metabolism and the food we eat. They are formed by a non-enzymatic reaction between carbohydrates and proteins known as glycation (Fig 1). Due to poor clearance, AGE metabolites accumulate in our organs over time causing many of the diseases associated with growing older and is even thought to drive the aging process itself. The pathological effects of AGE's lie in their ability to denature functional proteins and promote cell signaling pathways which leads to increased inflammation and stress responses. AGE's are most studied for their contribution to complications associated with diabetes but also play a role in a wide variety of other diseases including Alzheimer's, cardiovascular disease, renal and kidney failure, arthritis, and cancer to name a few.

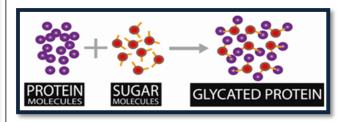


Fig 1 - AGE's (glycated proteins) are formed by a reaction between sugars and proteins and accumulate in our organs as we grow older with pathogenic effects.

A significant source of exogenous AGE accumulation in our bodies is from the food that we eat. The western diet is characterized by high intakes of red meats, highly manufactured and high fat foods and sugar laden desserts all of which are a significant source of reactive AGE metabolite. The increased consumption of these

foods over decades has led to a significant increase in the number of exogenous AGE's that accumulate in our organs and tissues. Heat processing for example is heavily used by food manufacturers to improve food safety, bioavailability and taste. Such heat treatment accelerates the reaction between sugars and proteins and therefore significantly accelerates dietary AGE formation. In addition, food manufacturing companies also directly add AGE's to some foods to enhance flavor, improve color and increase aroma. Foods high in protein and fat have the highest AGE content whereas grains, vegetables and fruits have the lowest. The AGE content in our diet depends not only upon nutrient composition but also the way we prepare our foods. As mentioned above heat accelerates AGE formation and therefore how we cook our foods can also have significant effects on dietary AGE content. It is estimated that frying, broiling and grilling can increase dietary AGE formation between 10 to 100-fold compared to uncooked food. For example, raw chicken breast contains approximately 800 AGE units/100g. Poaching the chicken for 20min increases the AGE content to around 1,000 AGE units but frying the same piece of chicken increases the AGE content to around 9,000 AGE units/100g (Fig 2).



800 AGE units/100g 9,000 AGE units/100g 1,000 AGE units/100g

Fig 2 - The way we cook our foods can also have significant effects on dietary AGE content. Pan frying chicken breast for example increases age content 10-fold.

While the pathogenic effects of AGE metabolites have been extensively studied in diseases such as diabetes, their role in cancer is less well known. Given their role in promoting inflammation and stress responses we examined if they also altered these biological pathways to promote cancer.

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We examined the circulating levels of the AGE metabolite called carboxymethyl-lysine (CML) in biological samples from prostate cancer patients. CML is one of the more common AGE metabolites and is used as a tasting agent by food manufacturers. In serum from low grade (less aggressive) and high grade (more aggressive) prostate cancer patients we found that CML levels were consistently higher in the patients with aggressive prostate cancer (Fig 3 upper). We also examined the levels of AGE metabolite in the prostate cancer tumors themselves and again saw the highest AGE levels in the more aggressive tumor samples (as indicated by the higher green fluorescence in Fig 3 lower).

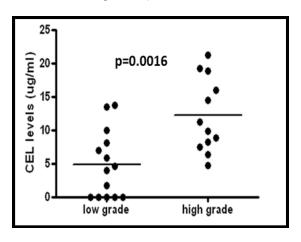




Fig 3 – Above: Levels of the AGE metabolite carboxyethyl-lysine are increased in serum from low grade and high grade prostate cancer patients. Below: Levels of the AGE metabolite carboxyethyl-lysine are also increased in tumor tissue from the same patients as shown by higher green fluorescence.

To expand these data further we examined the AGE signaling pathway (Fig 4) in the same prostate cancer tissue samples. AGE's can alter cell function by activating cell signaling cascades mediated by its receptor, known as the receptor for advanced glycation end products or RAGE for short. AGE activates RAGE to increase the expression of many factors involved in inflammation and stress responses (Fig 4). One such factor is known as nuclear factor kappa B (NFkB) and is associated with increased inflammation in multiple diseases. We found that like AGE metabolite levels both RAGE and NFkB are also elevated in tumor tissue compared to normal tissue with the highest levels being observed in the more aggressive high grade tumors as shown by higher red fluorescence in Fig 5 upper (RAGE) and lower (NFkB). This indicates that AGE accumulation in prostate cancer tissue may increase inflammation and stress responses to promote a more aggressive cancer. Further experiments are ongoing to further support this hypothesis.

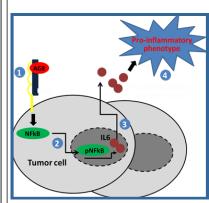


Fig 4 – Our model states that AGE's can bind to its receptor RAGE

(1) to alter cell signaling cascades and the activation of transcription factors such as s NFkB (2). This leads to the increased production of inflammatory factors such as IL6 (3) which leads to an increased inflammation in the tumor and a more aggressive phenotype (4).

| Normal prostate | Low grade | High grade |
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| Normal prostate | EA-HG | AA-HG |
| Normal prostate | EA-HG | AA-HG |

Fig 5: Above: Levels of the AGE receptor RAGE are increased in prostate cancer tissue from low grade and high grade prostate cancer patients as shown by higher red fluorescence. Below: Levels of NFkB are also increased in tumor tissue from the same patients as shown by higher red fluorescence.

This research is trying to further discern if the higher AGE levels observed in tumors correlate with higher AGE levels in the circulatory system. If so AGE levels may be a prognostic or diagnostic marker of tumor progression. Further experiments are underway to try and delineate the source of the increased AGE's observed in cancer patients and further isolate their pathogenic effects. In doing so we may identify novel approaches with which to target AGE function in cancer and identify a new treatment option in the fight against cancer.

Targeting AGE accumulation levels also offer several disease prevention strategies. It is becoming increasing clear that the accumulation of AGE's in our organs depends upon a balance between 1. Endogenous production during normal metabolism 2. Exogenous production through the foods we consume dietary and renal and enzymatic clearance. Imbalance in this "steady state" (as seen as we grow older) leads to increased AGE accumulation and disease phenotypes. While we can never prevent the accumulation of AGE's in our bodies we can adopt practical approaches in our everyday life to keep their accumulation at a minimum. Apart from watching what we eat and avoiding processed foods, altering our cooking habits can significantly reduce AGE intake. Over a lifetime even small changes in our dietary habits may make a significant contribution to our overall health as we grow older.

UPCOMING MEETINGS

PATHOLOGY SPRING SYMPOSIA

APRIL 28, 2014 - MAY 5, 2014

AT

KIAWAH ISLAND GOLF RESORT

ASCP 2013

BEYOND THE LAB

SEPTEMBER 18,-21, 2013

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THE PATHOLOGISTS MEET-ING

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