BioScience Research Collaborative
6500 Main Street
Houston, Texas

January 23-25, 2019

2nd Annual Texas Medical Center
Antimicrobial Resistance Research and
Stewardship Conference
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The Gulf Coast Consortia (GCC), located in Houston, Texas, is a dynamic, multi-institution collaboration of basic and translational scientists, researchers, clinicians and students in the quantitative biomedical sciences, who benefit from joint training programs, topic-focused research consortia, shared facilities and equipment, and exchange of scientific knowledge. Working together, GCC member institutions provide a cutting edge collaborative training environment and research infrastructure beyond the capability of any single institution. GCC training programs currently focus on Biomedical Informatics, Computational Cancer Biology, Molecular Biophysics, Neuroengineering and Pharmacological Sciences. GCC research consortia gather interested faculty around research foci within the quantitative biomedical sciences, and currently include Antimicrobial Resistance, Nanox, Mental Health, Chemical Genomics, Translational Pain Research, Theoretical and Computational Neuroscience, Alcohol and Addiction Research, Regenerative Medicine, Translational Imaging and Cellular and Molecular Biophysics. Current members include Baylor College of Medicine, Rice University, University of Houston, The University of Texas Health Science Center at Houston, The University of Texas Medical Branch at Galveston, The University of Texas M. D. Anderson Cancer Center, and the Institute of Biosciences and Technology of Texas A&M Health Science Center.

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Center for Antimicrobial Resistance and Microbial Genomics (CARMiG)

MISSION
The Mission of the Center for Antimicrobial Resistance and Microbial Genomics is to develop a comprehensive research and educational program to study the complex factors that contribute to the development of resistance and create an environment where specific actions can be taken to tackle this important public health problem. We seek to develop research in a variety of areas that involve, i) mechanisms of antimicrobial resistance, ii) epidemiology of antibiotic resistance organisms, iii) clinical impact of antibiotic resistance, iv) antibiotic stewardship and iv) the influence of antibiotic resistance in the environment (including in animals for human consumption). Primarily, we seek to develop innovative clinical and therapeutic approaches to deal with multi-drug resistant organisms.

CORE ACTIVITIES
Clinical and antibiotic stewardship cores – understanding the clinical aspects of antibiotic resistance and launching programs on surveillance, antibiotic stewardship, and interventional studies
Basic science – elucidation of the molecular mechanisms of antibiotic resistance and identification of potentially novel targets for drug discovery
Genomics – performing and analyzing bacterial genome sequencing to develop rapid diagnostic tools, evaluate antimicrobial resistance profiles, and study the molecular epidemiology of resistant pathogens
Pharmacology and animal models – development of animal models to study therapeutics and pharmacological aspects of antibiotics
Training and education – supporting the development of young investigators interested in antimicrobial resistance and launching educational training programs for clinicians and scientists at the graduate, postgraduate, and assistant professor level
Clinical trials – provide the ideal location and expertise to lead and conduct robust, large-scale clinical trials.

For additional information, please visit us at https://www.carmig.net/
Over 6 years ago under the direction of Dr. Raouf Arafat, the Houston Health Department (HHD) began discussions on hospital acquired infections (HAI) and antibiotic resistance (AR) with community experts. The results of these discussions lead to the following:

- Establishment of the Antimicrobial Stewardship Executive Committee (ASEC) – a committee of infectious disease experts who work collaboratively with HHD to strengthen the City’s collective efforts in preserving the use of antibiotics.
- Annual Antimicrobial Stewardship Symposiums which bring together approximately 200 healthcare professionals every year from across Houston and the surrounding communities to address the antibiotic resistance challenge.
- Successfully received a CDC grant in HAI/AR for over $700,000. Areas of focus includes strengthening local and state capacities to perform critical epidemiological and laboratory work by detecting, tracking and responding to known infectious disease threats and developing HAI/AR programs to increase antibiotic stewardship education.

Because of the impact to the community and public health, the Houston Health Department has made it a priority to address this patient safety issue working in a multifaceted approach that involves coordinated participation from leaders within the healthcare community, public health, and academia.
Agenda

Day 1 – Wednesday, January 23, 2019
Mechanisms of Antibiotic Resistance and Novel Antibiotic Approaches

8:00 – 8:30  
Registration

8:30 – 9:15  
Opening Key Note Lecture
Plasmids and Antibiotic Resistance: Novel Insights into Bacterial Sex
Alessandra Carattoli, BSc, PhD
Research Director – Istituto Superiore di Sanità

Session 1
Conveners: Barbara Murray, MD and Tim Palzkill, PhD

9:15 – 9:45  
Targeting Toxin Production and Toxin Activity as a Novel Non-Antibiotic Therapy for Clostridium difficile Infections
Charles Darkoh, PhD
Associate Professor – UTHealth School of Public Health

9:45 – 10:15  
CRISPR and Antibiotic Resistance
David Weiss, PhD
Associate Professor – Emory University

10-15- 10:30  
Break

Session 2
Conveners: Ashok Chopra, PhD and David Greenberg, MD

10:30 – 11:00  
Cell Membrane Adaptation Against Antimicrobial Peptides
Truc T. Tran, PharmD
Assistant Professor – UTHealth Science Center at Houston

11:00 – 11:30  
Evobiotics: Resistance to Resistance
Anthony Maresso, PhD
Associate Professor – Baylor College of Medicine

11:30 – 11:45  
Selected abstract: Parallel Dissemination of KPC-Producing Klebsiella pneumoniae ST258 and ST307 in Houston, TX
William Shropshire, University of Texas Health Science Center

11:45 – 12:00  
Selected abstract: Engineering β-lactamase Inhibitory Protein (BLIP) Variants with Altered Binding Specificity for β-lactamase Antibiotic Resistance Enzymes
Shuo Lu, Baylor College of Medicine

12:00 – 12:45  
Rapid Fire Session (Event Space)

12:45 – 2:00  
Poster session and lunch (Event Space) Posters 1-25, 75
Session 3
Conveners: Sam Shelburne, MP, PhD and Blake Hanson, PhD

2:00 – 2:30
*The Origin of Community-Associated MRSA*
Ashley Robinson, PhD
Professor – University of Mississippi

2:30 – 3:00
*Double Trouble: Tandem GES β-lactamases in ST309 Pseudomonas aeruginosa*
William R. Miller, MD
Assistant Professor – UTHealth Science Center at Houston

3:00 – 3:30
*Beta-Lactamase Inhibitors; A Renaissance*
Robert Bonomo, MD
Professor of Medicine – Case Western Reserve University

3:30 – 3:45
Break

Session 4
Conveners: Adriana Rosato, PhD and Eric Brown, PhD

3:45 – 4:15
*Antibiotic Adjuvants*
Gerry Wright, PhD
Professor – McMaster University

4:15 – 4:45
*Evolution of Antibiotic Resistance*
Yousif Shamoo, PhD
Vice Provost for Research – Rice University

4:45 - 5:00
Selected abstract: *The Fatty Acid Synthesis Protein Enoyl-ACP Reductase II (FabK) is a Target for Narrow-Spectrum Antibacterials for Clostridium difficile Infection*
Ravi Marreddy, Institute for Biosciences and Technology, TAMUHSC

5:00 – 5:15
Selected abstract: *Phage Loaded Magnetic Nanoparticles to Remove Multidrug Resistant Bacterial Biofilms: A Combined Experimental and Computational Study*
Pingfeng Yu, Rice University

Day 2 – Thursday, January 24, 2019
Pharmacology of Antimicrobials, Microbiome and Clinical Impact of Resistance

7:30 – 8:00: Registration

Session 5
Conveners: Tor Savidge, PhD and Samuel L. Aitken, PharmD

8:00 – 8:30
*Vancomycin – Current Practice Considerations*
Thomas Lodise, PharmD, PhD
Professor – Albany University
8:30 – 9:00  
**PK/PD Considerations for Optimizing Beta-lactam/beta-lactam Inhibitor Combinations**  
Vincent Tam, PharmD  
Professor – University of Houston

9:00 – 9:30  
**The Development of New Anti-infectives for Drug Resistant Infections**  
Omonike Olaleye, PhD  
Professor of Pharmacology – Texas Southern University

9:30 - 9:45  
Break

**Session 6**  
Conveners: Kevin Garey and Diana Panesso, PhD

9:45 – 10:15  
**Dysbiosis and Restoration of the Intestinal Microbiom**  
Herbert DuPont, MD  
Professor – UTHealth School of Public Health

10:15 – 10:45  
**The Microbiome Now and Beyond: A Functional Microbiome Perspective of Human Infectious Disease**  
Tor Savidge, PhD  
Associate Professor – Baylor College of Medicine

10:45 – 11:15  
**The Clinical Microbiology of the 21st Century**  
Robin Patel, MD  
Professor of Microbiology and Medicine – Mayo Clinic, Rochester

11:15 – 11:45  
**The Antibacterial Resistance: The Rationale for Strategy Trials**  
Vance Fowler, MD  
Professor of Medicine – Duke University

11:45 – 12:00  
**Selected abstract: LiaX Regulates Daptomycin (DAP) Resistance and is Responsible for the See-saw Effect by Altering Penicillin-binding Protein Localization in Multidrug-resistant Enterococcus faecalis**  
Ayesha Khan, University of Texas Health Science Center

12:00 – 12:15  
**Selected abstract: Transmission of Genetically Related, Multidrug Resistant, and Invasive Vancomycin-Resistant Enterococci (VRE) between Patients and Rooms on the Stem Cell Transplant (SCT) and Leukemia (LKM) Units**  
Lynn El Haddad, MD Anderson Cancer Center

12:15 – 1:00  
Rapid Fire Session (Event Space)

1:00 – 2:00  
Poster session and Lunch (Event Space) Posters 26-50, 59

**Session 7**  
Conveners: Julian Hurdle, PhD and Andrew Chou, MD

2:00 – 2:30  
**Bicarbonate Resensitization of MRSA to Beta-Lactams**
Arnold S. Bayer, MD
Distinguished Professor of Medicine – UCLA

2:30 – 3:00 Opening the DOOR: Novel Concepts in Clinical Trial Analyses for Resistance
Scott Evans, PhD
Sr. Research Scientist – George Washington University

3:00 – 3:30 Microbiome Predicts Clinical Outcomes in Cancer Patients
Jessica Galloway-Pena, PhD
Assistant Professor – UT MD Anderson Cancer Center

3:30 – 3:45 Break

Session 8
Conveners: Lynn Zechiedrich, PhD and Trish Perl-DeLisle, MD

3:45 – 4:15 Clinical Aspects of Enterococcal Bacteremia
Jose Munita, MD
Associate Professor – Clinica Alemana, Universidad del Desarrollo

4:15 – 4:45 Mold-resistant Infections
Dimitrios Kontoyiannis, MD
Professor – UT MD Anderson Cancer Center

4:45 – 5:15 NIH Funding for Antimicrobial Resistance
Clayton Huntley, PhD – NIH AMR program
Antimicrobial Resistance Program Officer – NIAID, Bethesda

5:15 – 6:15 A Futuristic View of the Microbiome
Lynn Bry, MD, PhD
Associate Professor – Harvard Medical School

Day 3 – Friday, January 25, 2019
Emerging Strategies for Antibiotic Stewardship to Prevent Resistance

7:30 – 8:00: Registration

Session 9
Conveners: Charlene Offiong, RPh, PharmD and Isaam Raad, MD

8:00 – 8:45: Keynote: A One Health Approach to Antibiotic Stewardship
Ruth Lynfield, MD
State Epidemiologist and Medical Director – Minnesota Department of Health

8:45 – 9:10: Public Health Policies and Activities for Antibiotic Stewardship
David Hyun, MD
Senior Officer – The Pew Charitable Trusts

9:10 – 9:35 The Joint Commission’s Antibiotic Stewardship Standards
David W. Baker, MD, MPH, FACP
Executive Vice President – The Joint Commission
9:35- 10:00  Statewide Antimicrobial Stewardship Collaborative
Timothy Jenkins, MD
Associate Professor – University of Colorado

10:00- 10:15  Break

Session 10
Conveners: Kristi Kuper, PharmD and Robert Atmar, MD

10:15-10:40  Quick Wins in Antibiotic Stewardship
Shivani Patel, PharmD, BCPS
Clinical Specialist - Memorial Hermann Southwest Hospital

10:40-11:05  Doing What’s Best for Our Patients
Antimicrobial Stewardship in the ED
Larissa May, MD
Professor of Emergency Medicine – UC Davis Health

11:05 – 11:30  Antibiotic Stewardship and the Quality Connection
Michael Hill, MD
Medical Director – St. Tammany Parish Hospital, Louisiana

11:30 – 12:00  The Social Determinants of Antibiotic Use: Towards a More “Human” Stewardship
Julia Szymczak, PhD
Assistant Professor – University of Pennsylvania

12:00 – 12:45  Rapid Fire Session (Event Space)

12:45 – 1:45  Poster session and Lunch (Event Space) Posters 51-74, 76

Session 11
Conveners: Barbara Trautner, MD and Will Musick, PharmD

1:45 – 2:10  Leveraging Microbiology in Antibiotic Stewardship
Katherine Perez, PhD
Professor and Chair – Houston Methodist

2:10 – 2:35  Antiviral Resistance Update
Roy F. Chemaly, MD, MPH, FACP, FIDSA
Professor – UT MD Anderson Cancer Center

2:35 – 3:00  Antifungal Resistance Update
Luis Ostrosky-Zeichner, MD
Professor – UTHealth Science Center at Houston

3:00 – 3:25  Antifungal Stewardship in Clinical Practice
Samuel L. Aitken, PharmD, BCPS (AQ-ID)
Clinical Pharmacy Specialist – UT MD Anderson Cancer Center

3:25  Closing remarks
**Samuel L. Aitken, Clinical Pharmacy Specialist**  
Infectious Diseases  
UT MD Anderson Cancer Center  

*Antifungal Stewardship in Clinical Practice*  

Samuel L. Aitken, PharmD received his pharmacy degree from the University at Buffalo School of Pharmacy and Pharmaceutical Sciences in 2011. He completed his pharmacy practice residency at Yale-New Haven Hospital and an infectious diseases pharmacotherapy fellowship at the University of Houston / Baylor St. Luke’s Medical Hospital. He is currently a clinical pharmacy specialist in infectious diseases and infectious diseases pharmacy residency program director at MD Anderson Cancer. His current research is focused on the epidemiology, molecular mechanisms, and treatment of multidrug-resistant bacterial infections and C. difficile in patients with hematologic malignancy. Additionally, Dr. Aitken is the founding chair of the Antimicrobial Stewardship in Cancer Consortium (ASCC), a group dedicated to advancing antimicrobial stewardship research and practice in cancer patients.

Abstract:
Invasive fungal infections are a common cause of morbidity and mortality in hospitalized patients. Despite the frequency of fungal infections and the widespread use of antifungal agents, provider knowledge of appropriate antifungal treatment is lacking. Inappropriate use of antifungals directly contributes to antifungal resistance and an emerging public health threat. Antifungal stewardship is a relatively easy-to-implement, impactful means of mitigating harm caused by inappropriate antifungal use.
Dr. David Baker is the Executive Vice President for Health Care Quality Evaluation at The Joint Commission. He leads the Department of Standards and Survey Methods, the Department of Quality Measurement, and the Department of Research. This position oversees the development of performance measures, standards, survey methods, and National Patient Safety Goals for all Joint Commission accreditation and certification programs. Dr. Baker is also Editor-in-Chief for the Joint Commission Journal on Quality and Patient Safety.

Dr. Baker is a graduate of the UCLA School of Medicine and the UCLA School of Public Health. He also completed a research and policy fellowship with the Robert Wood Johnson Clinical Scholars Program at UCLA. Before assuming his current position, Dr. Baker was the Michael A. Gertz Professor of Medicine, Chief of the Division of General Internal Medicine and Geriatrics, and Deputy Director of the Institute for Public Health and Medicine at the Feinberg School of Medicine at Northwestern University. He has published over 250 original research articles and book chapters and has won multiple national awards for his research.

Abstract:
In 2016, The Joint Commission developed Antimicrobial Stewardship (AS) requirements for hospitals and nursing care centers (NCCs) based on the Centers for Disease Prevention and Control’s (CDC) core elements, and these went into effect January 1, 2017. This first generation of standards required hospital and NCC leaders to prioritize AS, provide support for AS activities, establish multidisciplinary AS team, implement interventions to improve antimicrobial prescribing, monitor antimicrobial use, and undertake improvement activities. However, the standards were not overly prescriptive of what specific interventions and measurements AS programs needed to do because, at the time, there were limited scientific studies and experience to guide development of more specific recommendations. The Joint Commission’s early experience with surveying facilities’ compliance with these standards suggested that almost all hospitals and NCCs had basic AS infrastructure and practices in place. This led The Joint Commission, in partnership with Pew Foundation, CDC and other organizations, to hold a Leading Practices in Antimicrobial Stewardship Conference in May, 2018. The conference participants identified several recommendations that are being evaluated for possible incorporation into the CDC core elements and TJC standards. The Joint Commission is also about to release draft Antimicrobial Stewardship requirements for ambulatory care facilities, and these will be reviewed.
**Arnold Bayer**, Professor  
Medicine, David Geffen School of Medicine  
University of California of Los Angeles  

*Bicarbonate Resensitization of MRSA to Beta-Lactams*

Dr. Bayer is Distinguished Professor of Medicine, David Geffen School of Medicine at UCLA and a Faculty member in the Division of Infectious Diseases, Harbor-UCLA Medical Center. In a Faculty career spanning more than 30 years, Dr. Bayer has focused on deepening the understanding of infectious diseases, in the arena of bacterial pathogenesis. He earned his medical degree from Temple University School of Medicine and did his Internship and Residency at Thomas Jefferson University Hospital, both in Philadelphia, PA. He did his Fellowship in infectious diseases at Harbor-UCLA and post-doctoral research training at the West LA-VAMC. Funded through NIH/NIAID and pharmaceutical sources, Dr. Bayer focuses on the expression of key virulence factors and genes, which enhance the ability of *Staphylococcus aureus* (e.g. MRSA) to cause infection and resist antibiotics, including host defense peptides. In addition to his responsibilities in the Harbor-UCLA Medical Center community, Dr. Bayer serves an active role in the international medical and academic spheres. A co-author of over 300 scientific papers, book chapters and reviews, he frequently lectures on topics in infectious diseases. Dr. Bayer has served on the NIAID National Association for Research in *Staphylococcus aureus* (NARSA), is a charter committee member of the International Consortium for the Study of Infective Endocarditis (ICE), and Councilor in the International Society for Cardiovascular Infectious Diseases (ISCVID).

**Abstract:**

Methicillin-resistant *Staphylococcus aureus* (MRSA) are a leading cause of invasive infections in both community-acquired and hospital-associated contexts. MRSA strains are intrinsically resistant by standard *in vitro* susceptibility testing to β-lactam antibiotics. In contrast, methicillin-susceptible *S. aureus* (MSSA) strains remain highly susceptible to many standard-of-care β-lactams (e.g. oxacillin; nafcillin; cefazolin). β-lactams are not recommended for treating MRSA infections: i) MRSA β-lactam MICs are above current CLSI “breakpoints”; ii) they bind relatively poorly to penicillin-binding protein (PBP) 2a (predominant PBP in MRSA strains responsible for cell wall synthesis and division); iii) β-lactam levels required to saturate PBP 2a exceed human serum levels achieved with standard clinical dose-regimens; and iv) treatment of experimental MRSA infections (e.g., endocarditis) with β-lactams are generally ineffective. Several labs recently showed that bicarbonate supplementation of standard MIC testing media can “sensitize” some (but not all) MRSA strains *in vitro* to β-lactams and host defense peptides (e.g., LL-37 from neutrophils; skin). Further, MRSA strains exhibiting a “bicarbonate-responsive” phenotype in vitro (i.e., β-lactam-resistant in standard media, but susceptible in bicarbonate-containing media) were effectively eradicated in murine bacteremia models with selected β-lactams. We amplified these observations using four prototype MRSA strains (LAC-USA-300; COL [USA 100] ; MW-2 [USA 400]; BMC1001 [USA 300]) which demonstrated the following key outcomes: i) all strains were resistant *in vitro* in standard (MHB) to both oxacillin (OX) and cefazolin (CFZ); two strains exhibited a bicarbonate-responsive phenotype in bicarbonate-supplemented MHB, becoming highly susceptible to both β-lactams, while two did not; ii) two bicarbonate-responsive strains were heterotypic on population analyses, while the other two strains were homotypic (AUCs > 0.9); iii) both bicarbonate-responsive strains
were effectively cleared from all target organs by both OX and CFZ in experimental endocarditis (IE), while two bicarbonate- nonresponsive strains were refractory to therapy; and iv) bicarbonate impacted both the *mecA-pbp2a* and *sarA-sigB* genetic pathways. This presentation will highlight: i) the scope of the bicarbonate-responsive phenotype *in vitro* to β-lactams; ii) the translatability of such *in vitro* metrics to a relevant *in vivo* model of invasive MRSA infection (IE); and iii) the mechanism(s) underlying bicarbonate-responsiveness in MRSA.

**Robert Bonomo**, Chief of Medical Service and Director of Geriatric Research, Education, and Clinical Center
Louis Stokes Cleveland VA Medical Center
Vice Chair for Veteran Affairs, Department of Medicine
University Hospitals Cleveland Medical Center
Director, CWRU-Cleveland VAMC Center for Antimicrobial Resistance and Epidemiology
Professor of Medicine, Pharmacology, Molecular Biology and Microbiology, Biochemistry, and Proteomics and Bioinformatics
Case Western Reserve University School of Medicine
*Beta-Lactamase Inhibitors; A Renaissance*

Dr. Bonomo is Professor of Medicine, Pharmacology, Molecular Biology and Microbiology, Biochemistry, and Proteomics and Bioinformatics at Case Western Reserve University School of Medicine. He also serves as Chief of the Medical Service at the Louis Stokes Cleveland VA Medical Center, Director of the Cleveland Geriatric Research and Education Clinical Care Center, Vice Chair for Veterans Affairs in the University Hospitals Case Medical Center Department of Medicine and Director, CWRU-Cleveland VAMC Center for Antimicrobial Resistance and Epidemiology (Case VA CARES). His research interests include the mechanistic basis of resistance to β-lactam antibiotics and β-lactamase inhibitors, the molecular epidemiology of multidrug resistant Gram-negative bacteria, infections in the elderly, and the implementation of molecular diagnostics in clinical care of patients with infectious disease. He is an elected member of the American Academy of Microbiology, the Association of American Physicians, and has been appointed to PCORI as a representative of the Infectious Diseases Society of America. He is also Co-Director of the Laboratory Center of the NIH Sponsored Antibacterial Resistance Leadership Group (ARLG).

**Lynn Bry**, Director
Crimson Specimen Bank
Assistant Professor
Harvard Medical School, Harvard University
*A Futuristic View of the Microbiome*

Dr. Bry Directs the Massachusetts Host-Microbiome Center and is a Medical Director in the BWH Clinical Microbiology and Molecular Pathology labs. Her research group studies host-microbiota interactions in the gut, and has developed therapeutic and diagnostic clinical applications that leverage microbial species and metabolites. In addition, she oversees a multi-institutional pathogen genomic sequencing program that monitors genomic drivers of drug resistance in patient isolates. The program was the first CLIA lab to join the FDA GenomeTrakr program and has developed real-time and functional platforms to evaluate
highly drug-resistant organisms including carbapenem-resistant Enterobacteriaceae (CRE) and drug resistant Clostridium difficile.

Abstract:
Diverse vectors mobilize drug resistance in pathogens, including via conjugative plasmids and transposons. In addition to specifying capacity for mobilization of resistance within and among species, the complement of mobile vectors also provides a robust set of information to assist with outbreak and strain cluster analyses. However, the complex and repetitive nature of these vectors introduces complexities in genomic analyses, and often requires specific technical, experimental and computational approaches to resolve carrying vectors and those involving nested structures. We present clinical cases that used mobile vector information to support gene-, vector-, strain- and cluster-level analyses to provide actionable data to support infection control and clinical microbiology operations.

Alessandra Carattoli, Research Scientific Director
Department of Infectious Diseases, Istituto Superiore di Sanità
University of Rome

Plasmids and Antibiotic Resistance: Novel Insights into Bacterial Sex

Dr. Alessandra Carattoli is the Research Scientific Director at the Istituto Superiore di Sanita in Rome Italy. She graduated Laurea cum Laude in Biological Sciences from the University of Rome and has authored 151 papers with over 13000 citations. She was awarded the World's Most Influential Scientific Minds Award in 2015 by ISI Thompson Reuters and the Highly Cited Researcher Awards in 2016 and 2017 by Clarivate Analytics Web of Science. Dr. Carattoli is dedicated to innovation in the field of bacterial genomics, producing advanced diagnostic and bioinformatics tools useful for antimicrobial resistance identification, detection of novel mechanisms of resistance and identification of trafficking of antibiotic resistance among bacteria of clinical relevance for human health and food safety.

Abstract:
The ability to trace the circulation of resistance determinants located on plasmids within different bacterial populations helps clarifying all the routes by which antimicrobial resistant bacteria and their related genes can spread. Plasmids evolved acquiring multiple, critical resistance genes. Conventional plasmid typing has been widely used in the last decade. The impressive number of plasmids identified and assigned to homogeneous types and groups brought novel knowledge on the horizontal transfer of clinically relevant antimicrobial resistance genes among different bacterial lineages. Epidemiological information contributed to the identification of "epidemic" plasmids that spread worldwide in different bacterial species. The development of whole-genome sequencing helped to identify and describe the dynamics of plasmid-mediated transmission of antimicrobial resistance genes, within hospital environments but also between food-producing animals and humans, and to trace the spread of common plasmids circulating in different sources and reservoirs. Phylogenetic analysis of bacterial genomes, combined with plasmid typing and epidemiological data can clarify the circulation and spread of specific clinically relevant genetic determinants such as those conferring resistance to 3rd and 4th generation cephalosporins, colistin and carbapenems.
Dr Roy Chemaly is a Professor of Medicine in the Department of Infectious Diseases, Infection Control & Employee Health at The University of Texas MD Anderson Cancer Center in Houston, Texas. Dr. Chemaly completed his training in infectious diseases and medical microbiology at the Cleveland Clinic Foundation in Ohio. He is board-certified in internal medicine/infectious diseases, medical quality, and medical microbiology. During his fellowship, Dr. Chemaly completed a master’s degree in public health from Northeastern Ohio Universities in Rootstown, Ohio. In addition to Dr. Chemaly’s clinical research work in virology and multi-drug resistant organisms, he is the director of the Infection Control program at MD Anderson Cancer Center and the chair of the Infection Control Committee and the chair of the Conflict of Interest Committee. In addition, he was appointed as the chair of the Transplant Infectious Diseases Special Interest Group of the American Society of Bone Marrow Transplantation which is growing under his leadership.

Dr Chemaly devoted his career studying viral infections in immunocompromised patients, specifically those undergoing hematopoietic cell transplantation (HCT) for hematologic malignancies. He published extensively on mechanisms to treat and prevent viral infections in this population. During his tenure at MD Anderson Cancer Center, he established the clinical virology research program and assembled a research team that conducts phase II and phase III clinical trials for new antiviral drugs, desperately needed for our immunocompromised patients, in addition to PI-initiated trials and lab protocols. He led and successfully completed more than 15 international and national clinical trials and several studies focusing on diagnosis and management of viral infections; data from which have been presented nationally and internationally with subsequent publications in high-impact journals. Dr. Chemaly has distinguished himself as an outstanding research leader of infectious diseases. He is a very resourceful, innovative and productive expert research leader in infection prevention. He has produced high quality research which has had a great impact on preventing and appropriately managing infections (particularly viral infections) in immunocompromised patients. Dr. Chemaly’s work has been reported in numerous peer-reviewed journals including New England Journal of Medicine, Blood, Journal of Infectious Diseases, and Clinical Infectious Diseases.

Abstract:
Cytomegalovirus (CMV) infection is a significant complication in hematopoietic cell transplantation (HCT) recipients. With prolonged and repeated use of anti-CMV drugs, CMV can become resistant to standard therapy, resulting in increased morbidity and mortality, especially in HCT recipients. CMV resistance is diagnosed by detecting specific genetic mutations. Depending on the genotyping results, multiple strategies can be adopted to treat resistant CMV infections, albeit no randomized clinical trials exist so far except the newly published phase II trial for maribavir. Other strategies may include reducing immunosuppression (if possible), ganciclovir dose escalation, ganciclovir and foscarnet combination, leflunomide, and adjunct therapy such as CMV-specific cytotoxic T-lymphocyte infusions. Novel therapies such as maribavir, brincidofovir, and letermovir should be further studied for treatment of resistant CMV.
Charles Darkoh, Associate Professor  
Epidemiology, Human Genetics & Environmental Sciences  
UT School of Public Health  

Targeting Toxin Production and Toxin Activity as a Novel Non-Antibiotic Therapy for Clostridium difficile Infections

Dr. Charles Darkoh is a tenured Associate Professor at the University of Texas Health Science Center, School of Public Health-Center for Infectious Diseases and MD Anderson UTHealth Graduate School of Biomedical Sciences at Houston, Texas, USA. His laboratory studies pathogenesis and molecular basis of enteric infectious diseases with the goal to identify and understand the mechanisms of novel virulence factors, pathways, and unique microbial products that can be harnessed for diagnostics and therapeutics. Dr. Darkoh’s research in Clostridium difficile focuses on understanding how toxins A and B, which are directly responsible for disease, are regulated and produced as well as antibiotic resistance mechanisms in this pathogen. Recent discoveries in his laboratory have uncovered a new innovative approach to treat C. difficile infections, which is being developed as a novel non-antibiotic therapy for this multidrug-resistant pathogen.

Abstract:

Clostridium (Clostridioides) difficile infection (CDI) is the leading cause of hospital-acquired and antibiotic-associated diarrhea in the United States, with the total cost of treatment estimated to be up to 7 billion U.S. dollars annually. C. difficile, a multidrug-resistant Gram-positive anaerobic pathogen, overpopulates in the colon after the gut microbiota has been altered by antibiotic therapy. Thus, antibiotic therapy is a major risk factor for CDI. Treatment has become complicated due to the increasing virulence of the causative strains, sporulation, recurrence, tendency to resist multiple antibiotics, and antimicrobials used in treatment that further disrupt the composition and colonization resistance of the colonic microbiota. As a result, there is an urgent need for non-antibiotic treatments that preserve the colonic microbiota. During infection, C. difficile causes disease by releasing toxins A and B into the colon resulting in massive inflammation and diarrhea. To date, only strains that are able to produce toxins have been associated with disease. Therefore, inhibiting toxin production and/or toxin activity using non-antimicrobial drugs that spares the colonic microbiota is a promising approach to combat this multidrug-resistant pathogen. This talk will discuss our effort in developing non-antibiotic therapy for C. difficile infections using promising drugs that inhibit both toxin production and toxin activity.
Dr. DuPont is Professor and Director for the Center for Infectious Diseases at the University of Texas School of Public Health. Graduate of Emory University School of Medicine. Dr. DuPont’s major research goals are to define the epidemiology, immunology, genetic resistance, clinical features, control, prevention and therapy of enteric infectious diseases. Laboratory techniques and procedures are typically developed in Houston and taken to the field in an international setting.

Four lines of research are currently being conducted by Dr. DuPont and his colleagues. The first is a study of the pathogenesis and epidemiology of diarrhea caused by definable enteric pathogens including enterotoxigenic Escherichia coli (ETEC), enteroaggregative E. coli and norovirus. Molecular typing for epidemiologic studies and novel detection methods including Real-time PCR laboratory methods are being performed in Houston to define microbial virulence genes. Secondly, this group is looking at the host genetics and susceptibility to travelers’ diarrhea and post-infectious irritable bowel syndrome (PI-IBS) with a focus on inflammatory cytokine polymorphisms. Thirdly, clinical trials with novel compounds to prevent and treat travelers’ diarrhea are being carried out. Currently, the group is evaluating a nonabsorbed rifamycin derivative, rifaximin in the prevention and treatment of acute bacterial diarrhea. Finally, this group is working with new vaccines to prevent bacterial and viral diarrhea. The group just completed an international field trial demonstrating a high degree of effectiveness in preventing travelers’ diarrhea and ETEC diarrhea through administration of a transcutaneously applied patch ETEC vaccine developed by IOMAI Corporation. Future clinical studies of safety, immunogenicity and efficacy with this patch vaccine are being planned.

Abstract:
Healthy people show dramatically different intestinal microbiomes. While showing different patterns, healthy people all have a rich and diverse microbiomes, with strict anaerobic bacteria responsible for more than 90% of the total gut microbiota. Microbiota disturbances associated with reduced microbiome diversity and an overgrowth of facultative anaerobes are referred to as dysbiosis. Dysbiosis is seen in central nervous system disease, cancer, intestinal infections and disease, allergic disorders, metabolic syndrome with type 2 diabetes and obesity and carriage of antibiotic resistant bacteria. Fecal microbiota transplantation is a powerful way to reverse dysbiosis. The value of this approach has been proven for recurrent Clostridium difficile infection. The Kelsey Research Foundation and the University of Texas have established a cooperative program of research aimed at product development and research discovery through microbiome reversal as a means of controlling or preventing complications of microbiota-associated disease states.
Dr. Scott Evans is a tenured Professor of Epidemiology and Biostatistics and the Director of the George Washington Biostatistics Center.

Professor Evans interests include the design, monitoring, analyses, and reporting of and education in clinical trials and diagnostic studies. He is the author of more than 100 peer-reviewed publications and three textbooks on clinical trials including Fundamentals for New Clinical Trialists. He is the Director of the Statistical and Data Management Center (SDMC) for the Antibacterial Resistance Leadership Group (ARLG), a collaborative clinical research network that prioritizes, designs, and executes clinical research to reduce the public health threat of antibacterial resistance.

Professor Evans is a member of the Board of Directors for the American Statistical Association (ASA) and the Society for Clinical Trials (SCT) and is a former member of the Board for the Mu Sigma Rho (the National Honorary Society for Statistics). He is a member of an FDA Advisory Committee, the Steering Committee of the Clinical Trials Transformation Initiative (CTTI), and serves as the Chair of the Trial of the Year Committee of the SCT.

Professor Evans is the Editor-in-Chief of CHANCE and Statistical Communications in Infectious Diseases (SCID), and the Co-Editor of a Special Section of Clinical Infectious Diseases (CID) entitled Innovations in Design, Education, and Analysis (IDEA).

Dr. Evans is a recipient of the Mosteller Statistician of the Year Award, the Robert Zackin Distinguished Collaborative Statistician Award, and is a Fellow of the ASA, SCT, and IDSA.

Abstract:
Randomized clinical trials are the gold standard for evaluating the benefits and risks of interventions. However, these studies often fail to provide the necessary evidence to inform practical medical decision-making. The important implications of these deficiencies are largely absent from discourse in medical research communities. Typical benefit:risk assessment involves separate data summaries for each efficacy and safety outcome. Outcome-specific effects are estimated and potentially systematically or unsystematically combined in B-R assessment with the belief that such analyses inform the totality of effects on patients. However, this approach is suboptimal for informing medical decision-making, as it fails to: (i) evaluate the association between component outcomes (i.e. efficacy, toxicity, quality of life); (ii) evaluate the cumulative nature of outcomes; (iii) systematically incorporate the relative importance of the component outcomes; (iv) address competing risks, and since efficacy and safety analyses are conducted on different analysis populations, the population to which these B-R analyses apply, is unclear.

We discuss methodologies to address these challenges. The Desirability of Outcome Ranking (DOOR) is an approach to pragmatic benefit:risk evaluation to better inform medical decision-making. When applying these methods, the probability of more desirable overall result from a benefit:risk perspective when using a treatment relative to a control, is estimated. This probability may be attractive for clinicians and patients.
when making treatment decision in practice. Partial credit analyses can be used to incorporate patient/clinician preferences associated with global patient outcome. Visual displays can be used to display treatment contrast as the partial credit preferences vary. The methods are illustrated using examples from studies conducted by the Antibacterial Resistance Leadership Group (ARLG).

Vance Fowler, Professor  
Medicine and Molecular Genetics & Microbiology  
Duke University School of Medicine  

_The Antibacterial Resistance: The Rationale for Strategy Trials_

Vance Fowler, MD, MHS, Professor, Departments of Medicine and Molecular Genetics & Microbiology, Duke University Medical Center. Dr. Fowler has extensive expertise in clinical and translational research in bacterial infections. Dr. Fowler created the S. aureus Bacteremia Group and co-founded the International Collaboration on Endocarditis. He is a project PI in the Clinical Trials Transformation Initiative (CTTI; total value 37 million), the Communicating PI of the Antibacterial Resistance Leadership Group (ARLG; total value >100 million); and PI of a multinational NIH-funded trial of staphylococcal bacteremia (total value 20 million). He has over 225 publications, has been cited over 13,000 times, and has an h-index of 58.

Abstract:
The Antibacterial Resistance Leadership Group (ARLG) was formed in June 2013. To date, over 35 studies have been initiated in the 4 priority areas of Gram negative infections, Gram positive infections, Stewardship and Diagnostics. This presentation highlights some of the critical studies and findings, as well as ground-breaking statistical innovations and novel resources, that have been developed by the ARLG.

Jessica Galloway-Peña, Assistant Professor  
Medical Genetics  
UT MD Anderson Cancer Center  

_Microbiome Predicts Clinical Outcomes in Cancer Patients_

Dr. Jessica Galloway-Peña received her undergraduate degree in Biology and Chemistry from Our Lady of the Lake University in San Antonio, TX. She received her PhD in Microbiology and Molecular Genetics at The University of Texas Graduate School of Biomedical Sciences performing her dissertation work in Barbara E. Murray’s lab. She executed her postdoctoral fellowship in Samuel Shelburne’s lab at MD Anderson cancer Center to include being a prestigious Odyssey Fellow for three of those years. She then transitioned to research faculty in the Department of Genomic Medicine at MDACC. The majority of her studies have incorporated the genetic basis of pathogenesis as well as the molecular epidemiology of clinically relevant gram-positive pathogens, focusing on those with multi-drug resistance. She has more recently shifted her focus to microbiome dynamics during cancer treatment and the intense antibiotic
therapy seen in the hematological malignancy setting to determine the microbiome’s impact on cancer treatment outcomes, toxicities, and colonization/infection by antibiotic resistant organisms. Clinical applications of her research include determining genetic and chemical markers for microbial diversity that can be used in the clinical setting, designing predictive risk models for antibiotic resistant infectious risk during chemotherapy, and promoting antimicrobial stewardship and microbial conscious treatments during cancer therapy.

Abstract:
The microbiome is all of the microscopic organisms and their genomes present in association with the human body. Many microorganisms live in harmony with their human hosts, as well as living in equilibrium with other microorganisms in order to provide functions essential for human health and survival. However, when this precarious balance is disrupted by illness, or administration of therapeutics, this can often lead to detrimental effects in a patient, particularly in immunocompromised oncology patients. This talk mostly focuses on recent work elucidating that a patient’s microbial diversity, even before they start cancer treatment, can be linked to risk of infection during leukemia induction chemotherapy. Many of these patients demonstrate decreases in microbial diversity, microbial temporal instability, and increases in the presence of taxa known to cause infection over the course of chemotherapy. Patients that are able to maintain their microbiome, however, are more likely to remain infection free post neutrophil recovery. Our studies show that antibiotic exposure is driving the instability of the microbiome during induction chemotherapy and has long-term infectious consequences. The negative effects of systemic antibiotics on cancer treatment outcomes shown to be wide spread across other cancer types and treatment regimens will also be briefly discussed. Thus, baseline and serial monitoring of the microbiome may allow for risk-stratification for a number of cancer therapy toxicities and outcomes, to include pre-emptive therapy in patients at high-risk for infections and more judicious use of antimicrobials in in low-risk patients.

Michael Hill, Vice President Quality & Utilization Management
Medical Director, St. Tammany Quality Network
St. Tammany Parish Hospital

Antibiotic Stewardship and the Quality Connection

Dr. Michael Hill provides leadership and direction for planning, organizing and coordinating the mission of creating a successful St. Tammany Quality Network.

He also is responsible for assisting in the development of physician relationships across this same network, for stimulating the growth of the organization and providing leadership in medical management by working through and with network physicians. He is seen as technical and mentoring resource to physicians.

Dr. Hill has been a physician in Southeastern Louisiana for close to 30 years and cared for patients in over ten hospitals during that time. He has held several committee and administrative roles along the way and he has been elected to the Medical Executive Committee of three different hospitals and was the board chairman for IMG Healthcare for more than seven years.
Dr. Hill received his bachelor's degree from the University of New Orleans and his medical degree from Louisiana State University Medical Center. After an internship and residency in internal medicine at Charity Hospital of New Orleans, where he was named Chief Resident, he went on to complete a fellowship in infectious disease in the LSU Department of Medicine.

Abstract:
Antibiotic Stewardship is now well established and entangled in the DNA of St. Tammany Parish Hospital—it has been a journey. In 2006 the antibiotic stewardship program, Bug Club, began informally including several other hospitals in the area to standardize antimicrobial use. Over the next several years, several other initiatives were launched to reduce inpatient antibiotic use. As a result of previous efforts, it became apparent that a formal antibiotic stewardship program was needed. In 2013 a formal antibiotic stewardship program was introduced at STPH following the traditional stewardship activities. Continued efforts showed significant impact: decreasing C. difficile rates, antibiotic usage and costs. The program expanded to include: penicillin skin allergy testing, rapid diagnostic testing, negative predictive value for MRSA PCR, antibiotic timeout, and outpatient collaboration. In 2018, STPH developed an outpatient antibiotic stewardship program in order to bridge the gap between referring facilities and STPH.

It is imperative to link antibiotic stewardship programs to quality and improved patient outcomes. Once that connection was made the STPH administration fully endorsed the program and provided the necessary resources for success.

Clayton Huntley, Program Officer
Division of Microbiology and Infectious Diseases, Bacteriology and Mycology Branch
National Institute of Allergy and Infectious Diseases

NIH Funding for Antimicrobial Resistance

Clayton Huntley, PhD is the antibiotic resistance Program Officer at the National Institute of Allergy and Infectious Disease. He earned his doctorate at Texas A&M University studying RNA plant viruses, and did postdoctoral research at the Cleveland Clinic working on Respiratory Syncytial Virus (RSV). He then worked on discovering and developing new small molecule antivirals at Wyeth Pharmaceuticals. At the NIH, Dr. Huntley currently oversees a large portfolio of grants and clinical trials focused on Staphylococcus and Enterococcus.
David Hyun, M.D., researches and develops strategies for Pew’s antibiotic resistance project, which supports policies that remove regulatory, economic, and scientific obstacles to the discovery of new antibiotics and ensures that antibiotics are prescribed only when necessary. Hyun’s work focuses on stewardship of antibiotics used in human medicine. Before joining Pew, he practiced medicine at Children’s National Medical Center, where he developed and co-chaired the antibiotic stewardship program. He also was an assistant professor of pediatrics at George Washington University School of Medicine. Hyun received a bachelor’s degree in chemistry from the University of Minnesota and a Doctor of Medicine from Ohio State University’s College of Medicine. He completed his residency in pediatrics at Indiana University and a fellowship program in pediatric infectious disease at Baylor College of Medicine.

Abstract:
National and state policies for inpatient antibiotic stewardship programs have significantly increased the number of hospitals that have implemented such programs. Further refinement to existing policies or implementation of new policies will be needed to support implementation of meaningful stewardship activities across all hospitals, especially facilities with limited resources. Efforts are also needed to develop policies to expand stewardship efforts into outpatient settings by developing financial incentives for clinicians, providing data-driven feedback on their prescribing patterns, and supporting their stewardship efforts with educational tools and resources for both physicians and patients.

Tim Jenkins, MD is an Infectious Diseases physician, the Director of the Antibiotic Stewardship Program at Denver Health, and an Associate Professor of Medicine at the University of Colorado. For the last 10 years he has been involved in the development and execution of quality improvement initiatives, research, and policies to improve antibiotic use at the local, state, and national levels. He was a co-author of the Infectious Diseases Society of America guideline for implementing antibiotic stewardship programs and he is also member of the NIH Antibacterial Resistance Leadership Group.

Abstract:
Twenty-six Colorado hospitals participated in a statewide antimicrobial stewardship collaborative. The initial focus of the collaborative was to improve the diagnosis and antibiotic treatment of patients
hospitalized with urinary tract and skin and soft tissue infections. This talk will review the organization, objectives, and methodology of the collaborative, the results of the initial intervention, and lessons learned.

**Dimitrios Kontoyiannis**, Distinguished Endowed Professor
Cancer Research
UT MD Anderson Cancer Center

*Mold-resistant Infections*

Dr. Dimitrios P. Kontoyiannis received his medical degree Summa Cum Laude from the National and Kapodistrian University of Athens in Greece. He then completed a postdoctoral clinical research fellowship in Mycology at the Section of Infectious Diseases at the University of Texas MD Anderson Cancer Center in Houston, Texas, followed by training in Internal Medicine at Baylor College of Medicine in Houston, where he served as a Chief Medical Resident. He was subsequently trained as a clinical fellow in Infectious Diseases at Massachusetts General Hospital and obtained a Master in Clinical Sciences from Harvard Medical School in Boston. He spent 3 years at the Whitehead Institute for Biomedical Sciences/Massachusetts Institute of Technology as a fellow in the Harvard MIT Clinical Investigators Training Program. He is currently the Director of the Mycology Research Program and Professor in the Department of Infectious Diseases Infection Control and Employee Health at the University of Texas MD Anderson Cancer Center. He is also an adjunct professor at the University of Houston College of Pharmacy. He serves as an associate editor for Medical Mycology and sits on the editorial boards of Antimicrobial Agents & Chemotherapy, Medical Mycology, and Transplant Infectious Disease. He is a reviewer in several peer-reviewed journals in infectious diseases, oncology, and hematology. Dr Kontoyiannis has authored more than 170 peer-reviewed manuscripts and over 30 invited articles and book chapters.

Abstract:
Dr. Kontoyiannis will provide an overview of antifungal resistance in molds, using the emerging molds such as Mucorales and Fusarium as an examples. We will discuss the artificialities of in vitro testing and contrast them to the pathophysiology of in vivo invasive growth of molds. We will further elaborate on the fact that the clinical significance of in-vitro susceptibility remains unclear, as outcome in highly immunosuppressed patients is multifactorial such as net state of immunosuppression and co-morbidities, site of infection, offending fungus, timing to diagnosis and start of treatment, TDM, and drug-drug interactions. A low or a high MIC do not predict outcome; however, in-vitro resistance may help to predict a treatment scenario where the antifungal is less likely to be effective. Furthermore, the absence of clinical breakpoints against molds, the lack of automatized methods and limited number of laboratories performing susceptibility testing for molds in the US remain significant barriers. Finally, I will be discussing some interesting investigational diagnostic and therapeutic approaches that are promising in improving outcomes of opportunistic infections caused by Aspergillus and other resistant molds.
Dr. Lodise is a Clinical Pharmacist at the Stratton VA Medical Center in Albany, NY. He received his Ph.D. in Epidemiology at the University at Albany and his Pharm.D from Temple University School of Pharmacy. Integrating his dual interests in scholarship and patient care, Dr. Lodise’s overall research goal is to quantitatively enhance our current understanding of antimicrobial exposure-response relationships in patients with invasive bacterial infections. His research encompasses three interrelated domains: pharmacokinetics (PK)/pharmacodynamics (PD), epidemiology, and outcomes. His specific research objectives are 4-fold: develop “personalized” patient care strategies that improve outcomes; reduce the likelihood of drug-induced toxicities; minimize the emergence of antibiotic resistant infections; and reduce healthcare costs. To date, he has published over 100 peer-reviewed articles in reputable scientific journals, including Clinical Infectious Diseases, Antimicrobial Agents and Chemotherapy, Lancet Infectious Diseases, Chest, and Journal of Antimicrobial Chemotherapy. He has also secured over $1.5 million in grant funding from various sources.

Abstract:
Vancomycin is the most commonly administered antibiotic in United States. Despite its introduction over a half century ago, the optimal dosing strategy for vancomycin remains undefined. The current vancomycin consensus statement indicates that the pharmacokinetic/pharmacodynamic (PK/PD) target for vancomycin is an area under the curve to minimum inhibitory concentration (AUC/MIC) ratio ≥ 400. Due to perceived difficulties in calculating AUCs in current practice, the guidelines recommend maintaining trough concentrations between 15-20 mg/L for serious methicillin resistant Staphylococcus aureus (MRSA) infections as a surrogate for achievement of an AUC/MIC ratio ≥ 400.

In this lecture, the speaker will discussed the data used to support the current approaches to dosing vancomycin for patients with serious S. aureus infections. As part of the review, the speaker will discuss the augmented risk of acute kidney injury associated with the currently recommended “15-20 mg/L” trough monitoring approach. He will discuss alternative approaches, and their scientific basis, to dosing and monitoring vancomycin in clinical practice. In particular, he will discuss the rationale for shifting away from the current recommended practice of trough-only monitoring to area under the curve (AUC)-guided vancomycin dosing. He will also review ways to estimate the AUC in real-time in clinical practice.
Ruth Lynfield, State Epidemiologist and Medical Director
Minnesota Department of Health

A One Health Approach to Antibiotic Stewardship

Ruth Lynfield, M.D. received her medical degree from Cornell University Medical College and did postgraduate training in pediatrics and in pediatric infectious diseases at Massachusetts General Hospital (MGH). She attended in pediatric infectious disease at MGH from 1992-1997, and was Assistant Director of the New England Regional Newborn Screening Program at the Massachusetts State Laboratory from 1992-1997. Dr. Lynfield then joined the Minnesota Department of Health as a medical epidemiologist, and was appointed State Epidemiologist in 2007 and Medical Director of the department in 2010. She is the Co-Principal investigator of the Minnesota Emerging Infections Program. She has conducted numerous infectious disease investigation and responses to outbreaks; she has also developed surveillance systems, and conducted public health research, evaluation and planning. Her research focuses on emerging infections, antimicrobial resistance and prevention of infectious diseases. Dr. Lynfield has also worked on antimicrobial stewardship tools and guidance across the continuum of care, and across disciplines (a One Health Approach).

Dr. Lynfield chairs the CDC Board of Scientific Counselors- Office of Infectious Diseases, and is a past member of the FDA Vaccines and Related Biological Products Advisory Committee, and the National Vaccine Advisory Committee. She is also a member of the American Academy of Pediatrics’ Committee on Infectious Disease and an Associate Editor of the “Red Book”. She has co-chaired the CDC Emerging Infections Program Steering Committee since 2008. Dr. Lynfield has chaired the Infectious Disease Society of America (IDSA) Public Health Committee, the IDSA Antibiotic Resistance Working Group, and has served on multiple other public health workgroups and committees. She is an Adjunct Professor of Medicine and Epidemiology at the University of Minnesota.

Abstract:
One health is the recognition that human, animal and environmental health are interconnected. One Health encourages the collaboration of multiple disciplines to achieve optimal health for people, animals and the environment. Antibiotic resistance is a One Health issue. All antibiotic use contributes to resistance. Exposure to resistant bacteria or genes is not limited only to the sector from which they emerged and they can persist in varied settings. People share their bacteria with each other and with their pets. Pets share their bacteria with humans. People are exposed to bacteria from agricultural sources. Resistance determinants and antibiotics have been found in soil, surface and groundwater. Methods to address antibiotic resistance and tools to promote antibiotic stewardship can be shared across disciplines.
Anthony Maresso is Associate Professor in Molecular Virology and Microbiology at Baylor College of Medicine. The focus of his laboratory is the molecular basis of disease caused by pathogenic bacteria. He runs an active research program at BCM that includes investigating the mechanism by which bacteria eat during host infection, the development of organotypic models to study host-pathogen interactions, the development vaccines against anthrax bacillus and E. coli, and the use of evolved viral predators as antibacterial agents.

Abstract:
A great strength of pathogenic bacteria is their inherent ability to rapidly change in the face of extreme selective pressures. The foundation of these changes lie in their ability to undergo de novo mutation and/or exchange DNA via horizontal transfer mechanisms. The current medical paradigm for drug development is to generate fixed small molecule chemical platforms, subject these platforms to rigorous safety and efficacy studies, and market these products to patient populations diagnosed with a disease the drug is intended to address. This process can take a decade, and at great costs. Whereas such an approach is a valid solution to diseases whose pathology is also fixed, it struggles to fill the void when the mechanistic underpinnings of the disease also changes. Perhaps there is no better example than multi-drug resistance to chemical antibiotics. In this presentation, the speaker will introduce the concept of the evolvable medicine, using as an example an approach that focuses on the discovery and/or directed evolution of efficient bacterial viruses (phage) to increase the pool of useable and safe antimicrobials on a time frame consistent with the evolution of resistance itself. Specific examples of the utility of this approach, as well as some limitations, will be discussed, in the context of modeled, but common, human infections.

Larissa May is Professor of Emergency Medicine and Director of Emergency Department Antibiotic Stewardship at the University of California-Davis. She is a national expert in antibiotic stewardship in the emergency department (ED). Dr. May received her M.D., her MSPH in Public Health Microbiology and Emerging Infectious Diseases, and her MSHS in Clinical and Translational Research from The George Washington University. Dr. May’s research interests center on infectious diseases in the emergency department, quality improvement and patient safety with a specific focus on antibiotic stewardship in the
ED. Dr. May has served as an investigator on multiple federally-funded and industry-sponsored research studies and has published over 50 peer-reviewed articles. She has served on committees and task forces for the Centers for Disease Control and Prevention including the outpatient antimicrobial stewardship working group, the National Institutes of Health ARLG diagnostics subgroup and professional organizations including the Infectious Diseases Society of America and the Society for Healthcare Epidemiology of America.

Abstract:
The emergency department is a critical setting for antimicrobial stewardship given its nexus between the hospital and community setting. While there are numerous barriers to antimicrobial stewardship in this fast-paced setting, we will discuss facilitators of stewardship and several examples of successful stewardship implementations using behavioral economics, diagnostics, and other methods for improving patient safety around antibiotic use in the ED.

William Miller, Assistant Professor
Division of Infectious Diseases
UT Health Science Center at Houston

Double Trouble: Tandem GES β-lactamases in ST309 Pseudomonas aeruginosa

William R. Miller, M.D. is an assistant professor with the Division of Infectious Diseases and a member of the Center for Antimicrobial Resistance and Microbial Genomics (CARMiG). He received his medical degree and completed his residency training in Internal Medicine/Pediatrics at McGovern Medical School at The University of Texas Health Science Center at Houston (UTHealth). He completed a fellowship in adult Infectious Diseases at McGovern Medical School at UTHealth and the UT MD Anderson Cancer Center program, where he served as Chief Fellow and completed a third year in basic science research as a part of the Advanced Academic Track. He joined the division as a faculty member in July 2017.

Dr. Miller’s current research interests involve the clinical impact and mechanistic bases of antimicrobial resistance. Active projects include studying the multilayered cell membrane defense networks of Gram-positive pathogens using enterococci as model organisms, understanding the inoculum effect in severe methicillin-sensitive Staphylococcus aureus infections and characterizing the molecular mechanisms of resistance of multidrug resistant Gram-negative bacteria. He was awarded the 2017 IDSA Education and Research Foundation Young Investigator Award in Infectious Diseases to study the evolving landscape of carbapenem-resistance across clinical isolates of Pseudomonas aeruginosa.

In addition to research, Dr. Miller is active in education and clinical service. He enjoys teaching medical students, residents, fellows and graduate students, both on the wards and in the laboratory, and serves as co-director of the Infectious Diseases Seminar in the Graduate School of Biomedical Sciences.

Abstract:
Infections due to Multidrug-resistant Pseudomonas aeruginosa are a challenging clinical problem. The novel β-lactam/β-lactamase inhibitor combination ceftolozane/tazobactam (C/T) is an antibiotic of last resort for these MDR-infections, but the emergence of resistance is concerning. We recently identified
C/T resistance in association with two acquired GES extended spectrum β-lactamases in clinical P. aeruginosa isolates from Houston, Texas, and show that ST309 has the potential to become an emerging high-risk clone.

Jose Munita, Associate Professor
Division of Infectious Diseases
Universidad del Desarrollo, Chile
Director, Millennium Initiative for Collaborative Research on Bacterial Resistance (MICROB-R)

Clinical Aspects of Enterococcal Bacteremia

Jose M. Munita is a physician scientist currently working at Clinica Alemana – Universidad del Desarrollo in Santiago, Chile, where he holds a position as an Associate Professor. Dr. Munita received his medical degree from Universidad de los Andes, Santiago, Chile in 2004 and trained as an Internal Medicine specialist in Clinica Alemana, also in Santiago, Chile. Jose then moved to the US where he completed a three-year Infectious Diseases fellowship (Advanced Academic track) at the University of Texas McGovern Medical School and the MD Anderson Cancer Center, Houston, TX. After his training, he moved back to Chile where he is currently working. In 2018, Jose received the Young Investigator award by the International Society for Infectious Diseases and is a member of the Editorial Board for Antimicrobial Agents and Chemotherapy. Dr. Munita’s group is studying the genomic evolution of the Chilean-Cordobes clone of methicillin-resistant S. aureus and he is currently leading a country wide effort to improve the knowledge about antimicrobial resistant organisms in Chile.

Abstract:
The knowledge of antimicrobial resistant organisms in developing regions like South America is far from ideal, forcing local experts to rely on international data to help shape public policy and to guide clinical therapy. In order to improve our understanding of antimicrobial resistance (AMR) in Chile, we assembled a group of scientists with expertise studying AMR from different perspectives, including clinician scientists, veterinarians, evolutionary biology, mathematical modelling, epidemiology and experts in AMR and the environment. Through this group we expect to study AMR using a One Health approach in a developing country. In this talk, I will be presenting our main objectives, results so far and future perspectives.

Omonike Olaleye, Professor
Pharmacology
Texas Southern University

The Development of New Anti-infectives for Drug Resistant Infections

Omonike Olaleye, Ph.D. MPH. is a Professor of Pharmacology and Co-Program Director of the Maternal and Child Health Program at Texas Southern University. Her formal training in pharmacology and epidemiology, were at Johns Hopkins University School of Medicine and Harvard University respectively. Omonike has 18 years of research experience in infectious diseases drug discovery in the pharmaceutical
industry and academia. Her research studies are focused on targeting Methionine Aminopeptidase (MetAP), an essential metalloprotease that removes the initiating N-terminal methionine from proteins and peptides after protein translation. The essential role of MetAP, makes this enzyme a prospective target for the development of new therapeutic agents targeting clinically relevant pathogens. The emergence of multi- and extensively drug-resistant pathogens has increased the need for novel anti-infective agents. Using a high-throughput screening assay, she screened over 175,000 structurally diverse small molecules and identified potent and selective inhibitors of MetAP with in vitro and in vivo activity against clinically relevant pathogens. Omonike and her team have successfully progressed to pre-clinical development of the lead compounds. These inhibitors will be used as probes to understand the physiological role of MetAP in pathogenesis of infectious diseases, and help facilitate the design of novel therapeutics. Presently, she serves as the Core leader for Collaborations and Partnerships, at the Texas Southern University Research Center for Minority Institutions. To date, she has established 12 productive infectious diseases drug discovery programs at TSU. By the grace of God, our work has led to the discovery and development of novel therapeutic agents for potential treatment of drug resistant infections such as: HIV, Tuberculosis, Leishmaniasis and Hospital Acquired Infections. In collaboration with Dr. Janice Endsley at UTMB Galveston, they developed a novel drug screening assay of co-infection. Omonike’s strong background and expertise in infectious diseases pharmacotherapy, particularly in diseases that threaten both maternal and child health, strengthened her interest to further expand her research scope to pharmacoepidemiology of anti-infectives at Harvard University. There, she had the opportunity to study the effect of preventative antimalarial treatment on birth outcomes, a study involving over 60,000 pregnant women from Bostwana. Recently, she established the prestigious Federal Maternal and Child Health Fellowship Program at TSU in partnership with Baylor College of Medicine. Altogether, she has over 10 years of experience working with promising diverse students/professionals; having mentored over 100 individuals, who have progressed successfully in their academics and/or professional career. Previously as Assistant Dean of Student Services at TSU, Omonike had administrative oversight of the Office for Student Services in COPHS serving over 1,490 racially, ethnically and culturally diverse students in 6 undergraduate and 3 graduate degree programs. She provided leadership for all COPHS student admissions, orientation, advising, registration, recruitment, retention, and graduation.

Abstract:
Multidrug-resistant infections have changed the global therapeutic management of infections such as: Human immunodeficiency virus (HIV) and Mycobacterium tuberculosis (Mtb), two leading infectious killers of mankind worldwide. The emergence of multidrug-resistant Mtb strains (MDR-TB) and lethal synergy of Mtb with HIV has further complicated the therapeutic management of patients co-infected with TB-HIV. Moreover, the efficacies of certain TB/HIV drugs have been rendered futile in this setting. Hence, novel ways engineered to treat HIV patients co-infected with TB/MDR-TB are imperative. Our research efforts have been focused on the development of drug candidates for TB/MDR-HIV infections using an unconventional and strategic model. Our hypothesis is that engineering the simultaneous therapeutic targeting of N-terminal processing of essential Mtb proteins will treat and/or limit the progression and dissemination of Mtb in the HIV co-infection setting. We used an innovative approach to simultaneously inhibit N-terminal processing of proteins in Mtb. We have identified structurally diverse small molecules that target N-terminal processing enzymes in Mtb. We have proceeded to testing our inhibitors in assays of MDR-TB. Furthermore, we determined the potency of these inhibitors on HIV infectivity. Thereafter, we developed a novel assay for the high-throughput screening (HTS) of inhibitors that have both anti-HIV and anti-mycobacterial activity. To date, we have discovered potential drug
candidates that have dual anti-TB/MDR-TB and anti-HIV activity with efficacy in the low micromolar range. The engineering of simultaneous therapeutic targeting of TB-HIV activity could accelerate the future development of new agents to treat TB-HIV co-infections. Our results provide an unconventional model, potential drug candidates, and targets for efforts to control the global HIV and TB pandemics. In addition, our new unconventional approach provides an effective strategic model for the discovery of new drug targets and therapeutic candidates for other drug resistant infections.

Luis Ostrosky-Zeichner, Professor
Medicine and Epidemiology
UTHealth

*Antifungal Resistance Update*

Luis Ostrosky-Zeichner, MD is Professor of Medicine and Epidemiology and Vice Chair for Healthcare Quality for the Department of Medicine; he is also the director of the Laboratory of Mycology Research at the Division of Infectious Diseases and serves as the associate program director for the Infectious Diseases fellowship and as Medical Director for Epidemiology for Memorial Hermann Texas Medical Center. Dr. Ostrosky-Zeichner obtained his medical degree from Universidad Nacional Autonoma de Mexico. He completed his internal medicine residency at Instituto Nacional de Ciencias Medicas y Nutricion Salvador Zubiran, and his infectious diseases fellowship at the UTHealth McGovern Medical School and MD Anderson Cancer Center combined fellowship program. He has advanced training and experience in medical mycology, hospital epidemiology, transplant infectious diseases and healthcare quality.

Dr. Ostrosky-Zeichner is a fellow of the American College of Physicians, the Infectious Diseases Society of America, and the Society of Healthcare Epidemiology of America. He is an editor for Journal of Antimicrobial Chemotherapy and an editorial board member of Antimicrobial Agents and Chemotherapy and Critical Care Medicine. He has served as a consultant to the FDA and CDC. Dr Ostrosky-Zeichner is a board member of the Mycoses Study Group and the Immunocompromised Host Society, and previously served on and chaired the IDSA Guidelines Committee.

Dr. Ostrosky-Zeichner has published over 130 peer-reviewed articles and actively pursues translational and clinical research in medical mycology, transplant infectious diseases, hospital epidemiology, antimicrobial stewardship, and healthcare quality. His areas of interest include antifungal susceptibility testing, fungal diagnostics, PK/PD of antifungals, in vivo models of human mycoses, immunology, clinical hospital epidemiology, infection control, antimicrobial stewardship, transplant infectious diseases, and healthcare quality.

**Abstract:**

While still relatively uncommon, antifungal resistance is starting to become a real issue for clinicians. Intrinsic azole resistance has been traditionally described for certain Candida Species, such as C. krusei and C. glabrata, but acquired azole resistance in C. albicans is increasingly being reported. More worrisome, is the increase of echinocandin resistance in multiple Candida species, since these drug class
is currently the cornerstone for the management of invasive candidiasis. The world-wide emergence of Candida auris, constitutes an epidemiological emergency as not only can some strains be multi-drug resistant, but they have a high rate of nosocomial transmission. Resistance in cryptococcosis is rare, as is in endemic mycoses. Azole resistance in aspergillosis is being increasingly reported in geographically-defined areas and appears to be related to environmental strains and the use of azole-based pesticides.

Robin Patel, Chair
Clinical Microbiology
Consultant, Divisions of Clinical Microbiology and Infectious Diseases
Director, Infectious Diseases Research Laboratory
Elizabeth P. and Robert E. Allen Professor of Individualized Medicine
College of Medicine and Science
Mayo Clinic

The Clinical Microbiology Laboratory of the 21st Century

Dr. Robin Patel graduated from Princeton University with a BA in Chemistry and from McGill University in Montreal, Canada with an MD. She then moved to Rochester, Minnesota, where she completed a residency in Internal Medicine and fellowships in Infectious Diseases and Clinical Microbiology at the Mayo Clinic. Upon completion of post-graduate training, she joined the staff of the Mayo Clinic. She is currently Professor of Medicine and Professor of Microbiology, the Elizabeth P. and Robert E. Allen Professor of Individualized Medicine, Co-Director of the Clinical Bacteriology Laboratory, Director of the Infectious Diseases Research Laboratory, and Chair of the Division of Clinical Microbiology, at Mayo Clinic. Doctor Patel is board certified in Infectious Diseases (American Board of Internal Medicine), Medical Microbiology/Public Health Microbiology (American Board of Medical Microbiology), Clinical Pathology/Medical Microbiology (American Board of Pathology), and Internal Medicine, Medical Microbiology and Infectious Diseases (Collège des Médecins du Québec and Royal College of Physicians and Surgeons of Canada). Doctor Patel’s research focuses on clinical bacteriology diagnostic testing, antimicrobial resistance, and microbial biofilms. She has published over 350 peer reviewed manuscripts and has delivered numerous national and international presentations. She is a Fellow of American Academy of Microbiology, past chair of the United States Medical Licensing Examination Microbiology and Immunology Test Material Development Committee, co-chair of the ASM Microbe 2016-2018 Program Planning Committee, member of NIAID Council, advisor for the CLSI Subcommittee on Antimicrobial Susceptibility Testing, member of the American Board of Pathology Medical Microbiology and Clinical Pathology for ABPath CertLinkTM Test Development and Advisory Committees, member of the ASM Board of Directors and ASM President Elect (Presidency - July 2019-June 2020). She is an associate editor for the Journal of Clinical Microbiology and Clinical Infectious Diseases and course director for the Mayo Medical School Microbiology course.

Abstract:
Diagnostic testing for infectious diseases is undergoing a rapid revolution. In this presentation, the specific technologies and applications that are part of this change will be overviewed. Those applications that specifically address antibacterial resistance and appropriate use of antibacterial agents will be highlighted. Rapid nucleic acid amplification technologies, panel-based molecular diagnostics, proteomics,
metagenomics and advances in susceptibility testing will be presented. The ‘science’ needed to define appropriate utilization of microbiology diagnostics will be highlighted.

**Shivani Patel**, Clinical Specialist
Infectious Diseases
Memorial Hermann Southwest

*Quick Wins in Antibiotic Stewardship*

Shivani Patel, PharmD, BCPS is graduate of the University of North Carolina at Chapel Hill, School of Pharmacy and completed a PGY1 Residency at Vanderbilt University Medical Center. Dr. Patel currently is Clinical Specialist in Infectious Diseases at Memorial Hermann Southwest Hospital. At this location she consults with multiple infectious disease physician groups on appropriate antibiotic use for the facilities diverse patient population. In addition Dr. Patel actively participates in hospital wide quality improvement programs, cost savings initiatives and supports other medical service lines.

**Abstract:**
The road to creating a successful antimicrobial stewardship program is long and challenging. National organizations have provided robust guidelines for successful programs and minimum requirements for running a successful program. These recommendations center around improving patient outcomes through improved antimicrobial utilization. Even with these detailed guidelines, antimicrobial stewardship programs are challenged with the task of finding effective ways to create initiatives to drive change. The purpose of this presentation is to review the selection of obtainable targets for antibiotic stewardship versus more complicated projects, recognize stewardship activities that are less resource intensive and provide a positive impact on patient care, and develop programs that focus on areas of need and provide early successes in order to optimize clinical outcomes. Antimicrobial stewardship efforts are multi-disciplinary and should be based on local data in order to drive maximal change.

**Katherine Perez**
Infectious Diseases Clinical Specialist
Houston Methodist Hospital System

*Leveraging Microbiology in Antimicrobial Stewardship*

Katherine Perez, PharmD is an Infectious Diseases Clinical Specialist at Houston Methodist Hospital System in Houston, Texas, an Assistant Professor of Health Sciences at the Institute of Academic Medicine and Assistant Clinical Member at Houston Methodist Research Institute. In her current position, she is responsible for coordinating antimicrobial stewardship efforts for Houston Methodist Hospital System. Dr. Perez is a contributing member and holds leadership roles in several professional associations and holds a national appointment to the Clinical Laboratory Improvement Advisory Committee of the US
Centers for Diseases Control and Prevention. Her areas of research interest include antimicrobial resistance, rapid diagnostics, and clinical outcomes research.

Abstract:
Microbiology laboratories and clinical microbiologists can make significant contributions to antimicrobial stewardship programs (ASPs). The clinical microbiology laboratory plays a critical role in the timely identification of microbial pathogens and the performance of susceptibility testing. The advance of molecular diagnostics allows for rapid identification and susceptibility testing avoiding the need for extended courses of empiric antibiotic therapy when paired with an ASP. More novel research has focused on utility of rapid whole genome sequencing to predict antibiotic susceptibility testing results and to identify emerging mechanisms of resistance. As drug-resistant infection rates continue to rise, the need for rapid and accurate methods to detect antibiotic resistance is becoming increasingly important to optimize antibiotic therapy.

Ashley Robinson, Professor
Microbiology and Immunology
University of Mississippi

The Origin of Community-Associated MRSA

Dr. D. Ashley Robinson is a Professor of Microbiology and Immunology and Associate Director of Microbial Genomics at the University of Mississippi Medical Center. His research combines methods and concepts from genomics, evolution, and epidemiology, to study the natural history of pathogenic staphylococci and streptococci. His research has applications for outbreak investigations, infectious disease surveillance, and the development of new diagnostics. Dr. Robinson's Ph.D. training was at the University of Alabama at Birmingham and focused on the population genetics of Streptococcus pneumoniae. His postdoctoral training was at the University of Bath and focused on the population biology and epidemiology of Staphylococcus aureus. He was lead editor of a 2010 book published by Wiley-Blackwell, Bacterial Population Genetics in Infectious Disease, that synthesized knowledge about the natural history of major bacterial pathogens at the dawn of the next-generation sequencing era.

Abstract:
The USA300 North American Epidemic (USA300-NAE) clone of methicillin-resistant Staphylococcus aureus emerged in the early 2000s and became a leading cause of severe bacterial infections in the United States. Whole genome sequencing combined with phylogenetic analysis has not resolved the place of origin of this clone. To uncover the USA300-NAE origin, we recently applied a population genetics approach that relies on patterns of genetic variation that are expected to occur when an origin population expands its geographic range by a series of smaller populations. Under the so-called serial founder model of range expansion, new geographic regions are colonized by a small portion of the clone's genetic variation that is present in previously colonized regions. This neutral demographic process causes distinctive population genetic signatures: genetic diversity decreases and newly generated alleles increase in frequency, on average, during the clone's spread to increasingly distant regions. We detect both of these signatures
among USA300-NAE populations. Our results consistently point to the northeastern US, specifically the Pennsylvania region, as the place of origin of this clone. We also find that a fluoroquinolone resistance allele is one of only two non-fixed, derived alleles to have persisted throughout the range expansion and that resistance associates with significantly increased recombination rate. Our study shows that the process of geographic range expansion of an epidemic bacterial clone can leave population genetic signatures that are informative of place of origin even when phylogenetic analysis is inconclusive. Moreover, our study implies that random genetic drift can have a major influence on the early genetic history of an epidemic bacterial clone even when the influence of natural selection is also detected.

**Tor Savidge**, Associate Professor  
Pathology & Immunology  
Baylor College of Medicine

*The Microbiome Now and Beyond: A Functional Microbiome Perspective of Human Infectious Disease*

Dr. Savidge is Associate Professor in the Department of Pathology & Immunology at Baylor College of Medicine and is the Associate Director of the Texas Children’s Microbiome Center. His research interests include studying microbial-neuroimmune interactions in the gastrointestinal tract and nervous systems. This work has established new disease susceptibility biomarkers to Clostridium difficile infection, as well as identifying novel innate host defense mechanisms which are being pioneered as “allosteric therapeutics”.

**Abstract:**
The Human Microbiome Project (HMP) consortium established a unique population-scale framework which characterized the relationship between the human host and its microbial communities. These data provide strong initial evidence for host influences on microbial community structure and underscores the capacity for metagenomics and metabolomics to explore host-pathogen interactions in disease states. We used a systems-based approach to investigate the disease pathogenesis of Clostridioides (Clostridium) difficile infection (CDI) since this has increased dramatically over the last two decades. Despite a known correlation between antimicrobial disruption of protective gut bacteria and development of symptoms, it is not clearly understood why certain individuals are susceptible to CDI. To investigate stool biomarkers of CDI disease susceptibility we used global unbiased microbiome, metabolome, metaproteomics and miRNA profiling with supervised learning of omics data to identify disease-susceptibility signals that correlated strongly with clinical risk and treatment outcomes. Notably, population-scale microbiome analysis defined a new precision-infection control algorithm that accurately predicts CDI treatment outcomes and identifies susceptible patients in the general hospitalized population.
Dr. Shamoo’s research lab studies the dangerous rise of multi-drug resistant bacteria. With multi-drug resistant bacteria becoming increasingly common in hospitals, antibiotic resistance has threatened to return us to a pre-antibiotic era that would completely undermine modern medicine. His work seeks to elucidate the underlying biophysical principles of adaptation within bacterial populations during protein evolution. His group uses a combination of experimental evolution and biophysical approaches including X-ray crystallography, enzyme kinetics, protein folding, calorimetry and genomics to link changes in protein structure and function to their resulting phenotypes within evolving populations. His lab extends these physicochemical principles to predict the success or failure of specific adaptive alleles undergoing selection. By combining approaches from biophysics, genomics and experimental evolution, his group is able to identify and characterize successful evolutionary trajectories and then link those intermediates to the overall evolutionary trajectory of the bacterial populations.

Abstract:
Antibiotic resistance among bacterial pathogens remains one of the great challenges confronting public health in the world today. The widespread use of antibiotics has facilitated the rise of multi-drug resistant pathogens that threaten to undermine the remarkable success of modern medicine. Daptomycin is a frontline antibiotic with efficacy against Gram positive organisms and is used with increasing frequency against multi-drug resistant enterococci such as vancomycin-resistant enterococci (VREs). The goal of our work is to comprehensively map the evolutionary trajectories leading to DAP resistance in enterococci and elucidate how the identified changes in protein structure-function establish the physicochemical basis for the observed resistance phenotypes. We use quantitative experimental evolution in a novel, continuous culture bioreactor system as well as other experimental evolution approaches to identify and rank the most important evolutionary trajectories leading to resistance. Based upon these results, we then characterize the most relevant proteins and pathways to daptomycin resistance using a combination of biochemical and structural approaches that link the change in biophysical properties to resistance. We use a variety of techniques including X-ray crystallography, enzyme activity, ligand affinity, protein stability studies, RNAseq, qPCR, and others. This approach seeks to determine, not only the biochemical basis for resistance, but also those candidate proteins and pathways that would be well suited for the development of a new class of co-drugs that would target and delay the development of resistance. We also provide valuable molecular indicators of emerging resistance. Taken together, we take a multi-pronged approach to uncovering the mechanisms and physicochemical basis for the evolution of antibiotic resistance.
Dr. Julia E. Szymczak, PhD is an Assistant Professor of Epidemiology in the Department of Biostatistics, Epidemiology and Informatics at the University of Pennsylvania’s Perelman School of Medicine, where she also holds a secondary appointment in the Division of Infectious Diseases and is a faculty affiliate in the Center for Health Incentives and Behavioral Economics (CHIBE). Dr. Szymczak received her PhD in Sociology from the University of Pennsylvania and completed a postdoctoral research fellowship in the Division of Infectious Diseases at the Children’s Hospital of Philadelphia. Her research focuses on understanding how the social organization of medical work shapes the implementation of efforts to improve the safety and quality of healthcare. Dr. Szymczak seeks to uncover the social, cultural, and contextual factors that shape how healthcare workers and patients behave in complex clinical settings. She has examined various case studies of efforts to improve healthcare quality including resident duty hour restrictions, healthcare associated infection prevention and the integration of palliative care into pediatric oncology. Her current research focuses on understanding the social determinants of antibiotic prescribing and the sociobehavioral factors that shape clinician acceptance of antibiotic stewardship interventions.

Abstract:
Efforts to improve the use of antimicrobials require changing the behavior of prescribers and patients. This lecture will introduce the audience to a framework for approaching antimicrobial stewardship informed by sociobehavioral theory. The social determinants of antimicrobial use will be defined and applications to the design and implementation of antimicrobial stewardship will be presented. Practical strategies to uncover and overcome social barriers to implementing antimicrobial stewardship will be reviewed.

Vincent H. Tam received a B.S. from the National University of Singapore, Singapore and his Pharm.D. from Albany College of Pharmacy in Albany, New York. He completed an infectious diseases pharmacy residency at Detroit Receiving Hospital in Detroit, Michigan and a clinical pharmacology / infectious diseases fellowship at Albany Medical College. He is currently a (tenured) Professor at the University of Houston.

PK/PD Considerations: New β-lactam/β-lactamase Inhibitors
Houston College of Pharmacy in Houston, Texas. He is board certified in pharmacotherapy with added qualifications in infectious diseases. Dr. Tam is the (co-) author of over 150 peer-reviewed publications in antimicrobial pharmacokinetics / pharmacodynamics and infectious disease therapeutics. He is on the Editorial Boards of *Antimicrobial Agents and Chemotherapy,* and *Journal of Global Antimicrobial Resistance.* He has also served as a grant proposal reviewer for the National Institutes of Health (NIH) and reviewer for 25 journals such as the *Clinical Infectious Diseases, International Journal of Infectious Diseases, Journal of Antimicrobial Chemotherapy, Journal of Clinical Pharmacology,* and *Lancet Infectious Diseases.*

**Abstract:**
Dosing regimen design of antimicrobial agents involves a complex interplay of drug potency, pathogen susceptibility and toxicity constraints. Different possible combinations of dose, dosing interval and duration of therapy could be considered by clinicians for optimal patient outcomes. Our research group has a primary focus of examining the pharmacokinetics (PK) and pharmacodynamics (PD) of antimicrobial agents, as they relate to the optimal treatment of infections and suppression of resistance development.

Beta-lactams are the most commonly used agents for serious nosocomial infections. Despite a lack of intrinsic antimicrobial properties, beta-lactamase inhibitors are frequently used in combination of a beta-lactam to restore susceptibility of beta-lactam resistant pathogens. However, conventional PK/PD approach using time above MIC (%T>MIC) may not be directly applicable under these circumstances. Various investigators have proposed different approaches to better characterize the PK/PD exposures of beta-lactam / beta-lactamase inhibitor combination therapy. If validated, the clinical use of these agents could be optimized for specific pathogens. In this presentation, basic PK/PD concepts will be briefly reviewed. We will examine contemporary literature with respect to the experimental / clinical evidence supporting beta-lactam / beta-lactamase inhibitor dosing based on PK/PD. Finally, the potential to improve susceptibility testing of beta-lactam / beta-lactamase inhibitor combinations will also be discussed.

**Truc Tran, Assistant Professor**
Division of Infectious Diseases
UT Health Science Center at Houston

*Daptomycin Resistance in Enterococci: Beyond liaFSR*

Truc is an Assistant Professor in the Division of Infectious Diseases and a member of the Center for Antimicrobial Resistance and Microbial Genomics (CARMiG) at the University of Texas McGovern Medical School. Truc obtained her PharmD degree at the University of Houston College of Pharmacy and is also currently serving as the infectious diseases clinical pharmacist at the Memorial Hermann Hospital Texas Medical Center. Her areas of interest include antibiotic resistance and pharmacology. She hopes to dissect antibiotic-associated pathways to provide insights into bacterial physiology that could be exploited with novel pharmacological strategies and be translated into patient care.
Abstract:
Daptomycin (DAP) has been used clinically for multidrug-resistant enterococci, especially vancomycin-resistant enterococci Enterococcus faecium (VRE). Unfortunately, emergence of DAP resistance has become a major concern in recent years. Previous studies have identified the LiaFSR three-component regulatory system as a major mediator of DAP resistance in enterococci. In enterococci, LiaFSR has been shown to mediate cell membrane adaptive response and DAP resistance phenotype is associated with remodeling of cell membrane architecture especially in E. faecalis. Besides LiaFSR, the YycFG two-component regulatory system has also been linked with DAP resistance in E. faecium, although their contribution remains unclear. E. faecium isolates harboring mutations in yycFG were shown to be tolerant to DAP (lack of DAP bactericidal activity) and were not killed by combination of DAP and the β-lactam ampicillin. In this work, we aimed to define the contribution of a YycG substitution (S333L), encode the putative histidine kinase, in the development of DAP resistance.

David Weiss, Director, Emory Antibiotic Resistance Center
Associate Professor, Infectious Diseases
Emory University

Undetected Resistance as a Cause of Antibiotic Treatment Failure

Dr. Weiss received his PhD degree in Microbiology from New York University in 2004. Working under Dr. Arturo Zychlinsky, he studied how Toll-like Receptors work together to fight bacterial infections. He completed his postdoctoral training at Stanford University under Drs. Stanley Falkow and Denise Monack, studying virulence mechanisms of Francisella and the role of the inflammasome in host defense. Dr. Weiss started his laboratory at Emory University in 2008 and now studies the ways bacteria resist antibiotics focusing on the highly antibiotic-resistant Gram-negative bacteria Acinetobacter baumannii, Klebsiella pneumoniae, and Enterobacter cloacae. He is Director of the Emory Antibiotic Resistance Center and a Burroughs Wellcome Fund Investigator in the Pathogenesis of Infectious Disease.

Abstract:
Antibiotic resistance threatens the delivery of safe and effective healthcare and is projected to lead to 10 million annual deaths worldwide by 2050. Further complicating this epidemic are unexplained antibiotic treatment failures caused by bacteria that appear susceptible to an antibiotic. We describe an Enterobacter cloacae isolate harboring a minor subpopulation of cells that are highly resistant to the last-line antibiotic colistin. This subpopulation is genetically identical to the majority susceptible subpopulation and distinct from persisters. The resistant cells became predominant in the presence of colistin, yet returned to baseline after removal of the drug. Presence of the resistant subpopulation was dependent on the histidine kinase PhoQ, which was required for modification of the outer membrane component lipid A. In a murine infection model, this isolate mediated colistin treatment failure, in contrast to a susceptible strain. A genetically distinct Enterobacter clinical isolate displayed an even lower frequency colistin-resistant subpopulation which was undetectable by current diagnostic methods, yet similarly led to in vivo colistin treatment failure. These data demonstrate the ability of low frequency bacterial
subpopulations to contribute to clinically relevant antibiotic resistance, elucidating an enigmatic cause of antibiotic treatment failure and highlighting the critical need for more sensitive diagnostics.

Gerry Wright, Professor
Biochemistry and Biomedical Sciences
McMaster University, Canada

*Antibiotic Adjuvants*

Gerry Wright is the Director of the Michael G. DeGroote Institute for Infectious Disease Research and a Professor in the Department of Biochemistry and Biomedical Sciences at McMaster University. He holds the Michael G. DeGroote Chair in Infection and Anti-Infective Research and a Tier 1 Canada Research Chair in Antibiotic Biochemistry. He is a Fellow of the Royal Society of Canada (2012) and a fellow of the American Academy of Microbiology (2013) and is the co-founder of the Canadian Anti-Infective Innovation Network ([www.cain-amr.ca](http://www.cain-amr.ca)). He is the author of over 260 manuscripts and is a member of the editorial boards of several peer-reviewed. He has filed a number of patents and is the co-founder of Symbal Therapeutics. In 2016 he was named a McMaster Distinguished University Professor, the highest academic honor at the university.

Abstract:

For over six decades, the combination of antibiotics to improve efficacy and suppress resistance has been an effective therapeutic strategy. The combination of antibiotics and non-antibiotic compounds - antibiotic adjuvants - offers a complimentary approach that can directly overcome resistance and expand the potency of antibiotics. The search for such combinations is powered by our increasing knowledge of the antibiotic resistomes that are both intrinsic to bacteria and that can be shared through microbial populations. Targeting the resistome therefore becomes an orthogonal strategy in developing combination drugs that can contribute to solving the antibiotic crisis.
Dr. Lynn El Haddad is a Postdoctoral Fellow working under the mentorship of Dr. Roy F Chemaly at the Department of Infectious Disease at the University of Texas MD Anderson Cancer Center, Houston, TX. Dr. El Haddad earned her Master's and Doctoral degrees in Microbiology from the Laval University, Canada. Her current research focuses on the design and evaluation of a bacteriophage mixture that can aid in the fight against multi-drug resistant organisms in cancer patients. She is particularly interested in preventing the emergence of Vancomycin-resistant enterococci colonization in hematopoietic cell transplant recipients and leukemia patients. Dr. El Haddad’s work is funded by the American Cancer Society Postdoctoral award.

Abstract:
Background: VRE are a major cause of morbidity and mortality in immunocompromised patients. We adopted whole genome sequencing to track the emergence and spread of VRE strains in the hospital setting.

Methods: Whole Genome Sequencing (WGS) and phylogenetic analyses were performed to identify the different clusters of VRE strains and the potential transmission networks between patients and their rooms on the leukemia (LKM) and the stem cell transplant (SCT) units, located on two consecutive floors. The genomes of 89 VRE strains were analysed, including 35 VRE strains from rectal swabs from SCT and LKM patients, and 54 environmental VRE swabs from the patients’ main rooms and bathrooms. The 89 sequences were compared to each other using the maximum likelihood estimation. Sequence types, drug resistance genes, virulence genes, and patients’ outcomes were also determined.

Results: We identified two major clusters responsible of bacteremia events. The first cluster grouping 19 Daptomycin-resistant VRE strains, belonging to sequence types (ST) 736 and 664, were responsible of 2 out of the 5 bacteremia events (40%). The other cluster grouped 11 VRE strains that were lacking the pili operon fms14-17-13 (ST203, ST17) were responsible of 3 out of the 5 bacteremia events (60%). Interestingly, of 10 patients harboring daptomycin-resistant strains, only 3 (30%) were exposed to daptomycin within 18 months before strain recovery. Newly identified STs were isolated on both floors such as ST494, and ST772. Of note, the VRE genetic lineage belonging to ST494 was the only VanB-type strain (only previously isolated in Peru) whereas all other strains harbored the vanA gene. We observed highly genetically related strains transmitted between distinct rooms, floors, and time periods within the hospital in a period of 1 month (Figure).
Conclusion: Our findings confirmed horizontal transfer of highly related genetic lineages of multidrug resistant and invasive VRE strains between SCT and LKM patients and their room environment. New STs were identified and some correlated with bacteremia events. The use of a routine real-time WGS can characterize VRE strains and identify potential reservoirs of transmission in the healthcare setting in order to design interventions to prevent and control the spread of opportunistic and highly resistant organisms. Nanopore sequencing is underway to identify accessory genes of VRE strains of interest (Daptomycin-resistant and causing bacteremia events) that could explain the virulent behavior of VRE.

Ayesha Khan, PhD Student
Infectious Diseases and Microbiology
UT Health Science Center Houston

Antibiotic and Innate Immune Resistance Mediated by a Single Protein through a Novel Mechanism of Extracellular Signaling and Membrane Protection

Ayesha is a Ph.D. student in Infectious Diseases and Microbiology at UT Health in the lab of Dr. Cesar Arias and a member of the Center for Antimicrobial Resistance and Microbial Genomics (CARMiG). She currently serves as the president of the Texas Medical Center chapter of the American Society of Microbiology. Ayesha obtained her Bachelors in Microbiology, Immunology, Molecular Genetics and Global Studies with Public Policy at the University of California, Los Angeles where she worked on antibiotic synergy networks under Dr. Jeffrey Miller. Her Ph.D. focuses on elucidating mechanisms of daptomycin and antimicrobial peptide resistance in multi-drug resistant enterococci; and finding the molecular bridge between bacterial membrane homeostasis and cell wall synthesis. Ayesha hopes to continue translational research in clinical microbiology with an emphasis on the dissection of mechanisms of antibiotic resistance for the development of innovative therapeutics and on the advancement of molecular, genetic, and genomic diagnostic platforms to improve detection of resistant organisms.

Abstract:

Background: DAP is a front-line, lipopeptide, cell membrane (CM) disrupting antibiotic used for the treatment of MDR enterococcal infections. DAP is structurally and functionally similar to antimicrobial peptides (AMPs) secreted by the innate immune system. The LiaFSR stress response system regulates DAP resistance (DAP-R) through CM remodeling where anionic phospholipid microdomains are redistributed away from the division septum. LiaX, an effector of the LiaFSR system, is a novel 533 amino acid protein with distinct N and C-terminal domains. LiaX regulates DAP-R through its C-terminal domain. LiaX and/or the N-terminal (Nt) alone bind DAP with high affinity and are surface exposed and secreted in DAP resistant (DAP-Rt) clinical and lab strains. Here, we aim to dissect the extracellular role of LiaX in DAP and AMP resistance.

Methods: We assessed LiaX production dynamics under stress by incubating DAP susceptible (DAP-S) and DAP-Rt pairs of clinical and lab strains with 0.25x, 0.5x or 0.75x the DAP MIC for 1 hour followed by immunoblotting lysates and supernatants. DAP MICs by macrobroth dilution were determined on the DAP susceptible (DAP-S) strains using media (“spent” media) of DAP-Rt derivatives. The pair of strains were as follows: i) S613 (MIC=1µg/ml) and R712 (MIC=12 µg/ml), clinical strains isolated from a patient with fatal
bacteremia before and after DAP therapy, respectively; ii) OG1RF (MIC=2 µg/ml) and OG1RF\textit{liaX}^{289} (a DAP-Rt strain harboring a deletion of C-terminal domain of LiaX, MIC=12 µg/ml), iii) OG1RF\textit{ΔliaR} (derivative of OG1RF with deletion in \textit{liaR} encoding the LiaFSR response regulator, MIC=0.047 µg/ml) and OG1RF\textit{liaX}^{289}, iv) \textit{S. aureus ATCC29213} (MIC=1 µg/ml) and R712 or OG1RF\textit{liaX}^{289}. OG1RF\textit{ΔliaX} media was used as control.

qRT-PCR was performed on OG1RF to measure \textit{liaFSR} and \textit{liaXYZ} transcription in the presence or absence of DAP, LiaX or in the presence of both. Virulence of DAP-Rt and DAP-S strain pairs was evaluated by infecting wild-type (WT) \textit{C. elegans} or Δ\textit{pmk-1} worms that lack an innate immune system actively secreting AMPs.

Results: DAP-S strains produced LiaX in the presence of DAP in a concentration manner. DAP-Rt strains constitutively secreted high amounts of LiaX in the absence and presence of DAP. DAP MIC of susceptible enterococcal strains markedly increased in the presence of spent media from DAP-Rt strains, but not with spent media from OG1RF\textit{ΔliaX}. Spent media from DAP-Rt enterococcal strains did not have any effect in the DAP MIC of OG1RF\textit{ΔliaR} and \textit{S. aureus}. qRT-PCR experiments showed upregulation and activation of the \textit{liaFSR} system upon addition of LiaX (more so N-t) but only in the presence of DAP. All DAP-Rt strains were significantly more virulent \textit{in vivo} than their DAP-S counterparts in WT worms but showed no differences in virulence were observed in the Δ\textit{pmk-1} worms.

Conclusion: LiaX regulates DAP and AMP resistance in enterococci by modulating the remodeling of the CM and serving as a sentinel protein that likely senses the presence of antibiotics and AMP, triggering the cell envelope stress response. DAP-Rt strains are more virulent \textit{in vivo} due to enhanced innate immune resistance. LiaX is a novel protein that plays a major role in antibiotic resistance and virulence \textit{in vivo}.

**Shuo Lu**, Postdoctoral Scholar
Pharmacology and Chemical Biology
Baylor College of Medicine

*Engineering β-lactamase Inhibitory Protein (BLIP) Variants with Altered Binding Specificity for β-lactamase Antibiotic Resistance Enzymes*

Dr. Shuo Lu received her PhD degree of Biochemistry from the University of Oklahoma under the supervision of Dr. Helen I. Zgurskaya. She started as a postdoctoral fellow in Dr. Lei Zheng’s lab at the University of Texas McGovern Medical School, where she studied the functional roles of bacterial and mammalian Ca\textsuperscript{2+} transporters in intercellular Ca\textsuperscript{2+} homeostasis. After that, she joined Dr. Timothy Palzkill’s lab as a postdoctoral scholar in the Department of Pharmacology and Chemical Biology at Baylor College of Medicine. She is now working on multiple projects to study the structure and function of β-lactamases and β-lactamase inhibitory proteins.

Abstract:
CTX-M β-lactamases belong to the class A family of serine hydrolases and are one of the most widespread plasmid mediated sources of resistance to oxyimino-cephalosporins in Gram-negative bacteria. The development of novel inhibitors and diagnostic tools is essential for restoring the effectiveness of existing antibiotics. β-lactamase inhibitory protein (BLIP) is a potent proteinaceous inhibitor that binds a wide
range class A β-lactamases. However, BLIP binds weakly to CTX-M β-lactamases, with sub-micromolar affinity. Therefore, the goal of this study is to engineer and identify BLIP variants which bind to CTX-M enzymes with higher affinity and specificity. In initial selection process we were able to identify a BLIPE73W mutant by an in vivo genetic screen and an in vitro inhibition assay, which exhibited a low nanomolar binding affinity towards CTX-M-14, but also to other β-lactamases, such as KPC-2 and TEM-1. To further increase the binding specificity of BLIPE73W to CTX-M-14, we introduced mutations into thirteen different positions of BLIP, which were previously identified as hot spots for BLIP interactions with β-lactamases. In order to establish a fast screening method we fused BLIP mutant genes into phage display plasmids and performed a two-step phage display assay to select CTX-M-14-specific BLIP displayed phages for each of the thirteen phage libraries. For each round of selection BLIP displayed phage libraries were first incubated in KPC-2 immobilized immune tubes to eliminate KPC-2 binding and then transferred into CTX-M-14 immobilized immune tubes. CTX-M-14 specific binding phages were enriched after three rounds of selection and tight binding BLIP phages were identified by single point ELISA in 96-well immulon microtiter plates. The presence of BLIP mutations was reviewed by DNA sequencing. To gain more detailed biochemical information, the BLIPE73W double mutants were purified and Ki values were determined for inhibition of CTX-M-14 β-lactamase by monitoring hydrolysis of a colorimetric β-lactam substrate using a spectrophotometric assay. Several BLIP mutants have been identified with decreased Ki values to KPC-2 and TEM-1 but similar Ki values to CTX-M-14 compared to wild-type BLIP. We also performed binding assays to characterize the binding affinity of BLIP mutants to β-lactamases using surface plasmon resonance (SPR). To further expand the differences of Ki values among β-lactamases, we combined single point mutations in BLIPE73W and were able to create a quadruple mutation BLIPE73WK74RW112KH148V, which exhibits Ki values of 28 nM, 660 nM and 440 nM to CTX-M-14, KPC-2 and TEM-1, respectively. Interestingly, despite the fact that BLIPE73WK74RW112KH148V showed low inhibition towards KPC-2 enzyme activity, its binding to KPC-2 was retained. In order to understand the molecular basis for the loss of inhibition but not binding affinity, we determined the crystal structure of BLIPE73WK74RW112KH148V in complex with KPC-2. The structure revealed that BLIPE73WK74RW112KH148V varied significantly from BLIPwt and the residues involved in stabilizing the complex interface also changed.

Ravi Marreddy, Postdoctoral Research Associate
Infectious and Inflammatory Diseases
Institute of Biosciences and Technology, Texas A&M University

The Fatty Acid Synthesis Protein Enoyl-ACP Reductase II (FabK) is a Target for Narrow-Spectrum Antibacterials for Clostridium difficile Infection

Dr. Marreddy received his PhD from University of Groningen, Netherlands. He was a postdoctoral fellow at Goethe University, Frankfurt from 2011-2015. He has been a Postdoctoral Research Associate in the Center for Infectious and Inflammatory Diseases at Institute of Biosciences and Technology, Texas A&M University since 2016.

Abstract:
Clostridium difficile infection (CDI) is an antibiotic-induced microbiota shift disease of the large bowel. While there is a need for narrow-spectrum CDI antibiotics, it is unclear which cellular proteins are appropriate drug targets to specifically inhibit C. difficile. We evaluated the enoyl-acetyl carrier protein reductase II (FabK) that catalyzes the final step of bacterial fatty acid biosynthesis. Bioinformatics showed C. difficile uses FabK as its sole enoyl-ACP reductase, unlike several major microbiota species. The essentiality of fabK for C. difficile growth was confirmed by failure to delete this gene using Clostron
mutagenesis and by growth inhibition upon gene silencing with CRISPR-interference antisense to fabK transcription or by blocking protein translation. Inhibition of C. difficile’s FASII pathway could not be circumvented by supply of exogenous fatty acids, either during fabK’s gene silencing or upon inhibition of the enzyme with a phenylimidazole-derived inhibitor 1. The inability for fatty acids to bypass FASII inhibition is likely due to the function of transcriptional repressor FapR. Inhibition of FabK also inhibited spore formation, reflecting the enzyme’s role in de novo fatty acid biosynthesis for the formation of spore membrane lipids. Compound 1 did not inhibit growth of key microbiota species. These findings suggest that C. difficile FabK is a druggable target for discovering narrow-spectrum anti-C. difficile drugs to treat CDI but avoid collateral damage to the gut microbiota.

William Shropshire, PhD Student
Epidemiology
UT School of Public Health

Parallel Dissemination of KPC-Producing Klebsiella pneumoniae ST258 and ST307 in Houston, TX

Mr. Shropshire is a fourth year PhD in Epidemiology student within the Center for Infectious Disease at UTHealth’s School of Public Health. After graduating from the University of Texas at Austin (BA Biochemistry; 2010), he began an NIH post-baccalaureate intramural research training award (IRTA) fellowship with Dr. Benjamin White within the National Institutes of Mental Health. The White lab is where he began to grow a strong interest in genetics and basic science applications; nevertheless, rather than just work on parsing out the central nervous system of Drosophila melanogaster, he also wanted to begin working on more translational research. After taking a class on public health research on the campus of NIH with Dr. Chris Hafner-Eaton, he decided to enroll in the masters in public health (MPH) program at the UTHealth regional campus in Dallas. There he gained a very holistic perspective on community and global public health. I worked with Dr. Arnold Schecter with pesticide, e-waste, and industrial contaminant monitoring and surveillance as well as with the Montana Department of Public Health and Human Services and Dr. Katherine Froehlich-Grobe researching how populations with disabilities utilize public health programs, e.g. cancer screening and smoking cessation programs.

He moved to Houston, TX in 2015 where he decided that as a PhD student, he wanted to integrate both of his past lives as a basic science and public health researcher. His introduction to microbiology and infectious disease began with my first lab rotation working with Dr. Charles Darkoh. he became interested in the interface of how basic clinical practices, e.g. antimicrobial stewardship, begot the evolutionary pressures that resulted in bacterial species developing resistance to antimicrobials. This ultimately led him to join the Center for Antimicrobial Resistance and Microbial Genomics (CARMiG) at the McGovern Medical School in June of 2017 where he currently now works under the guidance of Dr. Cesar Arias and Dr. Blake Hanson. Over the past year, he has had an “ultimate infectious disease genomics boot camp” where he has been involved in essentially the entire workflow of bacterial genomic culturing, sequencing, processing, and analysis. His current interest is utilizing the advantages of both short-read and long-read sequencing platforms to resolve mobile genetic elements (MGEs) which is a primary means for bacteria to adapt and evolve to evolutionary pressures. The goal is to resolve these complicated structures and then determine their spatial distribution, how they are temporally disseminated between and within
bacterial taxons, and what significant correlations may exist between specific groups and individual MGEs
and clinical outcomes using genome wide association study (GWAS) and machine learning approaches.

Abstract:
Background: Carbapenem-resistant *Klebsiella pneumoniae* (CR-Kp) cause a diverse range of healthcare-
associated infections (HAIs). *Klebsiella pneumoniae* carbapenemase (KPC) is one of the most clinically
significant beta-lactamases carried by CR-Kpn and clonal group 258 (CG258) is the most common genetic
lineage of CR-Kp causing pandemic outbreaks. Recently, a new emerging lineage of *K. pneumoniae*,
sequence type 307 (ST307), carrying the CTX-M-15 extended spectrum β-lactamase (ESBL) was identified
in a single hospital in Houston, TX. There is current evidence to suggest ST307 has the potential to clonally
expand and co-circulate with the ST258 lineage.

Methods: We conducted a prospective observational study in 13 hospitals in the Houston metropolitan
area as part of the Consortium on Resistance Against Carbapenems in *Klebsiella pneumoniae* and other
Enterobacteriaceae (CRACKLE II) study. We identified patients infected or colonized with CR-Kpn. Whole
genome sequencing (WGS) was accomplished using the Illumina MiSeq, HiSeq4000, and Oxford Nanopore
Technology (ONT) MinION platforms. A custom bioinformatics pipeline was used to assemble genomes,
perform quality checks, and identify antibiotic resistance (AMR) genes. A maximum likelihood
phylogenetic tree was generated from single nucleotide polymorphism alignment.

Results: A total of 81 patients carrying CR-Kp and harboring KPC were identified between July 2016 and
December 2017 with urine as the most common site of isolation. A total of 95 isolates were sequenced
with phylogenetic analyses revealing three CR-Kp clades. Sequence types ST307 (37%) and ST258 (40%)
were the dominant lineages, predominantly carrying KPC-2. Both ST258 and ST307 represented
independent monophyletic lineages. CTX-M-15 and SHV106 were present in all isolates belonging to
ST307 lineage and generally absent in ST258.

Conclusion: An emerging strain of KPC-harboring CR-Kp ST307 is prevalent in hospitals in Houston, TX,
equaling ST258 as the most frequent CR-Kpn genetic lineage. The ST307 lineage is a single clade that also
carries CTXM-15 and appears to be as prevalent as isolates belonging to ST258. KPC-carrying CR-Kpn ST307
has the potential to replace CG258 isolates in the United States.

Pingfeng Yu, Postdoctoral Fellow
Epidemiology
Rice University

*Phage Loaded Magnetic Nanoparticles to Remove Multidrug Resistant Bacterial Biofilms: A Combined Experimental and Computational Study*

Pingfeng Yu is a postdoctoral fellow at Rice University. He received his B. Eng.
and Ph.D. degrees from Tsinghua University and Rice University, respectively. His research interests
include bacteriophage, antibiotic resistance, and nanotechnology. Current projects are supported by NSF
research centers of Nanotechnology-Enabled Water Treatment (NEWT) and Halting Environmental
Antimicrobial Resistance Dissemination (HEARD).

Abstract:
Multidrug-resistant bacterial biofilms can shield pathogenic bacteria and their existence in both medical
and environmental settings poses a significant health threat. Biofilms are refractory to antimicrobial treatment because of the physical hindrance and bacterial heterogeneity. Therefore, there is an urgent need for effective biofilm eradication while avoiding undesirable health risks. Here, natural existing phages and magnetic nanoparticles were conjugated to achieve effective biofilm removal regardless of bacterial antibiotic resistance. Polyvalent phages infecting *Escherichia coli* NDM-1 and *Pseudomonas aeruginosa* PA01 were isolated from environmental samples by the sequential multiple-host approach. The broad host ranges of polyvalent phages allowed them to reproduce in a variety of biofilm bacteria enhanced their penetration in biofilm conditions. In addition, the ability to produce depolymerase enzymes further facilitated biofilm matrix disruption. To accelerate biofilm removal, a “Trojan Horse” strategy was proposed to eradicate well-established biofilms. Magnetic phage clusters (MPCs) could penetrate the biofilms with a weak magnetic field, which significantly improved biofilm removal efficiency relative to free phages. The nanoparticles were modified with amine functional group to mitigate NPs aggregation and to optimize phage orientation after conjugation. After six-hour treatment, the smaller MPCs (more infectious centers but weaker physical disruption) eradicated the 2-day mixed-species biofilms to a greater extent compared to the larger MPCs (fewer infectious centers but stronger physical disruption). Accordingly, a mathematical model was developed to simulate the phage-biofilm interactions under different treatments (e.g., free phage, larger MPCs, and smaller MPCs). The model demonstrated that larger MPCs worked better in intact and thick biofilms while smaller MPCs were preferred in scattered and thin biofilms. Overall, MPCs enhanced phage infection in biofilms and hold the promise as an alternative or supplement in biofilm removal.
Rapid Fire Talk Presenters
(In order of appearance)

Wednesday

Ian Furey, Baylor College of Medicine
*Investigating the Molecular Basis of KPC-2 Carbapenem Hydrolysis by Differentiating Penicillin from Carbapenem Hydrolysis*
Poster #3

Amy Prater, Rice University
*The Environment Influences How Enterococcus faecium Evolves Daptomycin Resistance*
Poster #6

Blake Hanson, UT Health Science Center Houston
*Resistance and Diversity of Staphylococcus aureus Causing Bacteremia in Latin America*
Poster #4

Jourdan Andersson, Baylor College of Medicine
*Use of host-directed therapeutics to combat Clostridium difficile infection*
Poster #1

Travis Carlson, University of Houston
*Epidemiologic Surveillance of Carbapenem-resistant Enterobacteriaceae in a Large Academic Teaching Hospital in Houston, Texas*
Poster #2

Patrick Hornak, UT Medical Branch at Galveston
*Adjunctive Ceftaroline in Combination with Daptomycin or Vancomycin for Complicated Methicillin-Resistant Staphylococcus aureus Bacteremia after Monotherapy Therapy: A Single-Center Experience*
Poster #5
Thursday

**Mindy Engevik**, Baylor College of Medicine  
*Fusobacterium nucleatum Bolsters Clostridium difficile Biofilms in Intestinal Mucus*  
Poster #27

**Ramya Prabhakar**, Rice University  
*Microfluidic Droplet Platform for Directed Evolution of Synthetic E. coli Populations*  
Poster #30

**Kevin Chen**, Texas A&M University  
*Ibuprofen Displays Synergistic Antibacterial Activity with FDA-Approved Antibiotics against Pseudomonas aeruginosa*  
Poster #26

**April Nguyen**, UT Health Science Center Houston  
*Cardiolipin Synthase Mediates Membrane Remodeling in Daptomycin Resistant Enterococcus faecalis*  
Poster #29

**Austen Terwilliger**, Baylor College of Medicine  
*Developing a Field-To-Practice Phage Therapy Pipeline*  
Poster #31

**Julia Messina**, Duke University  
*Enterococcal Bloodstream Infection during First Induction Chemotherapy Cycle Is Associated With Treatment-Related Mortality in Patients with Acute Leukemia*  
Poster #28
Friday

**Paul Kilgore**, UT Medical Branch at Galveston
*Non-antibiotic Therapies to Treat Gram-negative Antibiotic Resistant Bacterial Pathogens*
Poster #53

**Olga Macias**, UT Health Science Center Houston
*Unexpected Relationships Between Frequency of Antibiotic Resistance, Disease Phenotype, and emm Type in Group A Streptococcus*
Poster #54

**Aditi Deshpande**, Texas A&M University
*Metronidazole Resistance Mechanisms In Clostridium difficile*
Poster #51

**Sabrina Green**, Baylor College of Medicine
*Purging Invasive Species from the Gut Ecosystem Using Viral Predators*
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**Heer Mehta**, Rice University
*Complex and Unexpected Mechanisms of Antimicrobial Resistance in Pseudomonas aeruginosa and Nocardia*
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