Strandwitz, Philip

Philip Strandwitz, PhD
CEO and Co-founder, Holobiome, Inc.
Visiting Scholar, Antimicrobial Discovery Center
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https://holobiome.org/

**The Gut-Brain-Axis — Potential Therapeutic Targets**

Born in Wisconsin but brought to Boston by the allure of biotechnology, Philip is a microbial ecologist with expertise in the human microbiome, bacterial cultivation, and the gut-brain-axis. He received his Bachelor’s in Microbiology from the University of La Crosse – Wisconsin, and his PhD from Northeastern University in Boston, MA, where he focused on cultivating unique bacteria from the human gut microbiota, as well as studying their ability to modulate neurotransmitters. Philip’s work has been published in top journals like Nature Microbiology, and he has presented at numerous scientific and industrial conferences, including those sponsored by the New York Academy of Science and BIO. He is now CEO at Holobiome, a gut-brain-axis microbiome company he co-founded, located in Kendall Square in Cambridge, MA. Here he has assembled and manages a team of leaders in neuroscience, microbiology, and drug development, with the goal to translate microbiome science into interventions to treat diseases of the nervous system, with planned expansion into other markets.

**Conference Lecture Summary**
Dr. Joanna Lyon is an advanced clinical pharmacist at the University Of Maryland School of Pharmacy. As part of her responsibilities for the University of Maryland, she leads a transitions of care team at the MedStar National Rehabilitation Hospital in DC. This team includes doctorate level pharmacy students and seeks to better educate patients about their medications during their discharge from the hospital. Some of Dr. Lyon’s recent accomplishments consist of several publications including an analysis of the ABCB1 gene and Post Treatment Lyme disease in patients, and opioid usage patterns at the MedStar National Rehabilitation Hospital. In addition, this year Dr. Lyon has presented her work at the American Pharmacist’s Association national meeting, the Maryland Pharmacist’s Association annual meeting, and the Exchanged Quality Data for Rehabilitation national meeting. Dr. Lyon is motivated to use her education and skills to bring the best healthcare treatment to all patients. Compelled by the suffering that Lyme disease patients endure, she is particularly committed to helping find the underlying cause and treatment for Post Treatment Lyme Disease Syndrome.
Conference Lecture Summary

The high incidence of patients that have been treated for a known Lyme disease infection and yet have not returned to baseline health is a concerning healthcare issue in the United States. Sources cite the incidence of this Post Treatment Lyme Disease Syndrome (PTLDS) from 5% to 17% of individuals treated for an initial Lyme disease infection. There has been an increasing body of research to suggest that individual human genetic differences may be part of the explanation for these residual complications in some patients that experience PTLDS. The ABCB1 gene encodes for a series of efflux pumps in the human body called permeability glycoproteins (P-gp). These P-gps are found in high concentration in many key drug transport regions of the body: epithelial cells of the blood-brain barrier, epithelial cells lining the colon and small intestine, and cells in the kidney proximal tubules. The role of P-gps is to pump or transport a variety of toxins and medications away from vulnerable body organs. It has now been determined that variations in regions of the ABCB1 gene may influence not only how many of these P-gps are found in an individual’s tissue, but how well these pumps move toxins and medications through various tissue membranes. In addition, there are a number of medications that can increase and decrease how well these P-gps work in the body. Currently, how P-gp number and function correlates with PTLDS is still poorly understood, but a randomized control trial of 142 patients positively associates several single nucleotide polymorphism variations in ABCB1 gene with the PTLDS disease state. This finding strengthens the view that human genetic variation in the number and/or function of P-gp efflux pumps may be part of the association between PTLDS patients and the chronic, persistent symptoms they experience.
Liotta, Lance A.

Lance A. Liotta, MD PhD
University Professor
Co-Director Center for Applied Proteomics and Molecular Medicine
Medical Director Clinical Proteomics Lab
College of Science
George Mason University
Manassas, Virginia

 Shedding of urinary tick pathogen-specific proteins in patients with tick borne diseases

Dr. Liotta is a University Professor in the College of Science, George Mason University. He received the MD and PhD (Bioengineering) from Case Western Reserve University, and fulfilled his residency at the National Institutes of Health (NIH), where he initiated a research program that, to date, has yielded more than 700 publications (Highly Cited Investigator), and more than 100 issued or allowed patents. At NIH Dr. Liotta was Chief, Laboratory of Pathology, Chief, Section of Tumor Invasion and Metastasis, and Deputy Director of NIH under NIH Director Bernadine Healy. He and Dr. Emanuel Petricoin of the FDA set up the first NIH/FDA Clinical Proteomics Program. In 2005 Mason recruited Dr. Liotta, and Dr. Petricoin (co-Directors), and their distinguished scientific team, to create the Center for Applied Proteomics and Molecular Medicine (CAPMM). The Mission of CAPMM is to discover disease mechanisms, invent new technologies, educate the scientists of the future, and translate knowledge to help patients through prevention, early detection, and treatment. Dr. Liotta has invented and patented, along with his laboratory co-inventors, high-impact technologies in the
fields of diagnostics; microdissection (Laser Capture Microdissection), and proteomics (Reverse Phase Protein Microarrays, Biomarker Harvesting Nanoparticles, Preservation chemistries tissue, and Protein Painting to discover drug targets), that have been used to make broad discoveries. The Laser Capture Microdissection prototype is in the Smithsonian Collection. The CAPMM team applies these technologies, for example, to markers for early stage disease, accurate diagnosis of Tuberculosis and Lyme disease (Dr. Alessandra Luchini), individualized therapy for primary and metastatic cancer (Dr. Mariaelena Pierobon, Dr. Julia Wulfkuhle), therapy of premalignant breast cancer as a strategy for prevention (Dr. Virginia Espina), and anaccredited CAP/CLIA diagnostic lab for patient testing. Dr. Liotta has received numerous scientific awards, including election to AIBME Fellows status, the Arthur S. Flemming Award, the NIH Award of Merit, the Surgeon General’s Medallion, and the 2015 Virginia SHEV award for Research and Scholarship.

Conference Lecture Summary

Post-treatment Lyme disease syndrome (PTLDS) defines a subset of patients who experience persistent symptoms following antibiotic therapy. The cause of PTLDS, and the appropriate treatment, is highly controversial because direct molecular evidence of pathogen persistence has not previously existed. We utilized mass spectrometry enhanced by nanotechnology to study pathogen-specific proteins shed in the urine of acute Lyme and PTLDS patients (analytical sensitivity = 2.5 pg/mL in urine). We analyzed 415 urine samples comprising 1) acute Lyme disease (LD, Centers for Disease Control and Prevention definition), 2) PTLDS patients defined according to the Infectious Disease Society of America guidelines, 3) diseased negatives (tuberculosis and HIV), and 4) healthy controls. Our target pathogens were Borrelia, Babesia, Anaplasma, Rickettsia, Ehrlichia, Bartonella, Francisella, Powassan virus,
encephalitis virus, and Colorado tick fever virus. Specificity was ensured by a 3-tier authentication algorithm requiring 100% amino acid sequence identity with tick-pathogen proteins, evolutionary taxonomic verification for related pathogens, and lack of overlap with human or other organisms.

Hodzic, Emir

Emir Hodzic, DVM, MSc, PhD
Director, Real-Time PCR Research & Diagnostics Core Facility
School of Veterinary Medicine
Department of Medicine and Epidemiology
University of California, Davis, CA

Post-treatment persistence of antimicrobial tolerant replicatively-attenuated Borrelia burgdorferi in a mouse model

Dr. Hodzic graduated in 1978 from the School of Veterinary Medicine at University of Sarajevo, Bosnia and Herzegovina, then received Master (1984) and PhD (1990) degrees from the same university in the area of Microbiology and Molecular biology. In 1992, his career was abruptly stopped for two years as a result of the war in his home country of Bosnia and Herzegovina. After resettlement to the U.S., Dr. Hodzic earned a position at Yale University as a postdoctoral fellow. In 1997, he was awarded a faculty position at the Center for Comparative Medicine, at UC Davis. Currently, Dr. Hodzic is the Director of the Real-time PCR Research and Diagnostics Core Facility at UC Davis. After relocating to the U.S., his research has focused on using animal models to investigate the interaction of Borrelia burgdorferi, the agent of Lyme borreliosis, and Anaplasma phagocytophilum, the agent of human
granulocytic anaplasmosis, with tick vectors and mammalian hosts. He developed a mouse model of Anaplasma infection that mimics many of the same aspects as seen in humans. While working on B. burgdorferi persistence, Dr. Hodzic revealed groundbreaking evidence of B. burgdorferi in collagenous tissues after antimicrobial therapy. Most recent findings confirmed previous studies, in which B. burgdorferi could not be cultured from tissues, but B. burgdorferi DNA (BbDNA) was detectable in tissues months, and even a year after completion of treatment. In addition, there was a resurgence of spirochete BbDNA in multiple tissues at 12 months, with BbDNA copy levels nearly equivalent to those found in saline-treated mice. Despite the continued non-cultivable state, RNA transcription of multiple B. burgdorferi genes was detected in host tissues, BbDNA was acquired by xenodiagnostic ticks, and spirochetal forms could be visualized within ticks and mouse tissues by. During his career, Dr. Hodzic has collaborated with other researchers and produced numerous peer-reviewed publications from each project.

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**Conference Lecture Summary**

A basic feature of infection caused by Borrelia burgdorferi, the etiological agent of Lyme borreliosis, is that persistent infection is the rule in its many hosts. The ability to persist and evade host immune clearance poses a challenge to effective antimicrobial treatment. A link between therapy failure and the presence of persister cells has started to emerge. There is growing experimental evidence that viable, but non-cultivable spirochetes persist following treatment with several different antimicrobial agents. In earlier studies, we have detected a population of B. burgdorferi in tissues after antimicrobial therapy in mice treated with ceftriaxone, doxycycline, or tigecycline at various intervals of infection, and tissues were tested at intervals after treatment. Specific BbDNA was consistently detected in tissues.
of mice at late at 12 months after treatment, but culture was consistently negative. Spirochetes could be visualized by immunohistochemistry in tissues rich in collagen of mice after treatment. Antimicrobial-tolerant persistent spirochetes could be acquired by larval ticks; after blood meal ticks molt into next life stage, nymps and adults, which remained BbDNA-positive; nymps transmitted BbDNA to recipient immunocompromised mice with multiple tissues PCR-positive, with no obvious inflammation being observed; and allografts from treated mice transplanted into recipient immunocompromised mice transferred BbDNA to recipient mice. Transcriptional activity of BbDNA-positive tissues was detected for several target genes, suggesting their viability. In the next study we detected the subpopulation of viable, antimicrobial-tolerant, but slow dividing and persistent spirochetes of B. burgdorferi resurged in mice 12 months after treatment and re-disseminated into multiple tissues. Isolation of these spirochetes has been unsuccessful. In the most current study, we utilized a disease-susceptible (C3H/HeN) and disease-resistant (C57BL/6) mouse strain infected with B. burgdorferi strains N40 and B31, to confirm the generality of this phenomena as well as to assess the persisters’ clinical relevance. The status of infection was evaluated at 12 and 18-months after treatment. The results demonstrated that persistent spirochetes remain viable for up to 18 months following treatment, as well as being non-cultivable. The clinical relevance of persistent spirochetes and their resurgence beyond 18 months following antimicrobial treatment compels further studies utilizing other animal models.
Armstrong, Paige

Paige Armstrong, MD, MHS, LCDR
LCDR, US Public Health Service
Medical Epidemiologist
Rickettsial Zoonoses Branch
Centers for Disease Control & Prevention
Atlanta, GA

*Rickettsial diseases: Epidemiologic trends, clinical diagnosis and management*

Dr. Paige Armstrong is a Medical Epidemiologist in the Rickettsial Zoonoses Branch at the Centers for Disease Control and Prevention (CDC). She leads the branch’s response to ongoing outbreaks of Rocky Mountain spotted fever (RMSF) in multiple states in Mexico. LCDR Armstrong also serves as clinical advisor for research, coordinates outreach to states, and oversees healthcare provider education initiatives for rickettsial diseases, such as RMSF, Ehrlichiosis, Anaplasmosis, and Q fever. She is board certified in Emergency Medicine and completed her residency and chief year at the George Washington University in Washington, DC. Paige holds an MD from the University of Connecticut, and received a Master of Health Sciences and undergraduate degree from Johns Hopkins University in Baltimore, MD. Paige has led outbreak investigations in the Dominican Republic, Colombia and Panama. She also assisted in the Emergency Operations Center at the CDC on the Zika response, and worked alongside the Ministries of Health in El Salvador and Nicaragua to evaluate their national surveillance systems. Paige received the Alpha Omega Alpha Post-Graduate Research Award for her work identifying barriers to care in non-English speaking patients.
Dr. Armstrong’s talk on Spotted Fever Rickettsioses (SFR) will discuss the range of diseases that fall within this group, as well as national trends. Rocky Mountain spotted fever (RMSF) is the most severe and well known of the SFR, but there are other emerging species, such as R. parkeri and 364D. The talk will address clinical aspects, diagnostics, treatment, and prevention methods.

Baranchuk, Adrian

Adrian Baranchuk, MD, FACC, FRCPC, FCCS
Professor of Medicine
Queen’s University
Kingston, Ontario, Canada

Systematic approach for the diagnosis and treatment of Lyme carditis

Dr. Adrian Baranchuk, a native of Buenos Aires, Argentina, obtained his MD from the University of Buenos Aires in 1990. After qualifying in Internal Medicine and Cardiology (1995), he completed a Clinical Fellowship in Cardiac Electrophysiology (1997). In 2002 he immigrated to Spain for a Research Fellow. Dr. Baranchuk was appointed as a Clinical Fellow in Electrophysiology at McMaster University in September 2003. Dr. Baranchuk was appointed as an Assistant Professor of Medicine at Queen’s University (2006), promoted to Associate Professor in 2010 and to Full Professor in 2016 (with Tenure). He has founded the EP Training Program in 2007. He is a member of numerous editorial boards (Europace, Annals of Noninvasive Electrocardiology, JACC en Español, etc) and
Conference Lecture Summary

Lyme carditis represents about 2-3% of all Lyme disease presentations. Usually, patients with Lyme carditis are under-recognized in many ER departments. Patients need to visit the ER 2-3 times in average until the diagnosis is suspected. During this presentation, the “Suspicious Index in Lyme carditis (SILC)” will be discussed and its acronym “COSTAR” will be presented. This may help physicians to become more familiarized with this type of Lyme disease presentation. A full algorithm on how to treat this condition will be presented. High-degree AV block represents about 90% of all Lyme carditis cases. Failure to recognize this entity may result in preventable fatalities.

Chaconas, George

George Chaconas, PhD
Professor of Biochemistry and Microbiology & Microbiology, Immunology and Infectious Diseases
Snyder Institute for Chronic Diseases
University of Calgary,
Calgary, Alberta, Canada
An inside look at the life of a pathogen: Intravital imaging to study hematogenous dissemination of the Lyme disease spirochete

George Chaconas obtained his Ph.D. in the Division of Medical Biochemistry at the University of Calgary in Alberta, Canada and did postdoctoral work Cold Spring Harbor Laboratory in New York. He subsequently took a faculty position in the Department of Biochemistry at the University of Western Ontario. His laboratory focused on the molecular mechanism of DNA transposition or “how jumping genes jump”. In 1999-2000, with the help of a Guggenheim Fellowship, Dr. Chaconas’ research interests took a turn through a sabbatical at the NIH Rocky Mountain Labs in Montana where he began working on the Lyme disease pathogen *Borrelia burgdorferi*. In 2002 he took a position in the Department of Biochemistry & Molecular Biology and the Department of Microbiology, Immunology & Infectious Diseases at the University of Calgary in Alberta, Canada where he is currently Professor in the Snyder Institute for Chronic Diseases. He held the Canada Research Chair in the Molecular Biology of Lyme Borreliosis from 2003-2017. Work in his laboratory is focused on *Borrelia burgdorferi*, the spirochete causing Lyme disease. His research includes studies on a variety of processes in this organism including, telomere resolution, antigenic variation, genome dynamics, regulation of global gene expression, intravital microscopy to study *B. burgdorferi* dissemination in a living host and metabolomics approaches for Lyme disease diagnosis.

Conference Lecture Summary

One of the salient features of Lyme borreliosis that makes diagnosis difficult is the multitude of symptoms that can result from the disease. The wide variety of disease manifestations is a consequence of the ability of the spirochete (*Borrelia burgdorferi* in North America) to
disseminate to many locations within the body and to promote pathogenic processes at multiple sites. The disease process starts by inoculation in the skin through the bite of an infected hard-shelled tick (Ixodes species). The first stage of the disease is a localized infection in the skin. The next stage, early disseminated disease, results from invasion of the vasculature by the spirochetes. During this stage B. burgdorferi gets a free ride throughout the body. Finally, when spirochetes exit the bloodstream at an assortment of locations, they can cause a wide variety of physical problems and symptoms. The process by which B. burgdorferi departs from the vasculature into surrounding tissue remains largely unknown and is a focus of studies in my laboratory. This is a complex, multistage procedure that we study using intravital microscopy, a methodology that allows us to see live spirochetes at work in a living mouse, in real time, using spinning disk laser confocal microscopy. The early stages of escape require that a spirochete slows its wild ride in the bloodstream, which is like an ant being swept in a stream of water in a garden hose. This occurs through physical interactions with the endothelium that are mediated by adhesins on the spirochete, proteins that function like Velcro. The spirochetes also promote changes in the blood vessels which allow them to escape to the other side through an as yet uncharacterized mechanism. Intravital videos and data will be presented describing processes involved in both vascular interactions and vascular escape.
Naviaux, Robert K.

Robert K. Naviaux, MD, PhD
Professor of Medicine, Pediatrics, Pathology, and Genetics
University of California, San Diego School of Medicine
San Diego, CA

naviaux.ucsd.edu

Metabolomic Features of Acute and Chronic Lyme Disease—Early Results from the UCSD Lyme-ME/CFS Comparison Study

Dr. Naviaux is the founder and co-director of the Mitochondrial and Metabolic Disease Center (MMDC), and Professor of Medicine, Pediatrics, Pathology, and Genetics at UCSD, where he directs a core laboratory for metabolomics. He is the co-founder and a former president of the Mitochondrial Medicine Society (MMS), and a founding associate editor of the journal Mitochondrion. He is an internationally known expert in human genetics, inborn errors of metabolism, metabolomics, and mitochondrial medicine. Dr. Naviaux is the discoverer of the cause of Alpers syndrome—the oldest Mendelian form of mitochondrial disease—and the developer of the first DNA test to diagnose it. His lab also discovered the first mitochondrial DNA (mtDNA) mutations that cause genetic forms of autism and the metabolic features of the cell danger response (CDR). He directed the first FDA-approved clinical trial to study the safety and efficacy of the antipurinergic drug suramin as a new treatment for autism spectrum disorder (ASD). His lab has developed new methods in metabolomics and environmental toxicology that have shown that many complex chronic disorders like ASD and chronic fatigue syndrome (ME/CFS) have a metabolic signature that can be used in diagnosis and lead to fresh insights to treatment. Information
about Naviaux Lab research can be found on the web at: naviauxlab.ucsd.edu.

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**Conference Lecture Summary**

The symptoms of post-treatment Lyme disease syndrome (PTLDS) and myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) are very similar. Without knowledge of the triggering event, it is not possible to distinguish the two disorders on purely clinical grounds. Both are chronic fatiguing illnesses that lead to long-term pain and disability. Both disorders can display significant post-exertional malaise, disrupted and unrefreshing sleep, headaches, brain fog, autonomic dysfunction with or without postural orthostatic tachycardia syndrome (POTS) and small fiber polyneuropathy (SFPN), GI abnormalities, and joint and muscle pain, with or without fibromyalgia. In this study, we will compare the metabolomic signature of PTLDS to ME/CFS, and to Borreliosis associated with acute Lyme disease. In addition, we will present the first evidence for and against the role of environmental pesticide and other toxicant exposures in regulating innate immune and cell danger responses in patients with PTLDS and controls.

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**Storch, Eric**

Eric Storch, PhD  
Professor and McIngvale Presidential Endowed Chair  
Vice Chair & Head, Psychology  
Menninger Department of Psychiatry and Behavioral Sciences
Baylor College of Medicine, Texas

https://www.bcm.edu/healthcare/care-centers/psychiatry/services/obsessive-compulsive-disorder

**Obsessive Compulsive Disorder**

Dr. Eric Storch is Professor and McIngvale Presidential Endowed Chair in the Menninger Department of Psychiatry and Behavioral Sciences at Baylor College of Medicine (BCM). He serves as Vice Chair and Head of Psychology, and oversees the CBT for OCD program at BCM. Dr. Storch has received multiple grants from federal agencies for his research (i.e., NIH, CDC), is a Fulbright Scholar, and has published over 14 books and over 550 articles and chapters. He specializes in the nature and treatment of childhood and adult obsessive-compulsive disorder and related conditions, anxiety disorders, and anxiety among youth with autism.

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**Conference Lecture Summary**

There is a well-documented link between obsessive-compulsive symptoms and various immunological conditions, where the latter confers additional burden above and beyond the illness alone. This talk will focus on what is known about the link between immune disorders including Lyme and obsessive-compulsive symptoms. Particular attention will be given to discussing pediatric autoimmune neuropsychiatric disorder associated with streptococcus as a potential model for understanding this linkage. Discussion will be provided regarding the nature of standard therapies as well as more immune-modulating approaches.
Soloski, Mark J.

Mark J. Soloski, PhD
Professor of Medicine
Co-Director for Basic Research, Lyme Disease Research Center
Johns Hopkins School of Medicine
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Host Immune Response in Lyme Borreliosis

Mark J. Soloski, Ph.D., is currently a Professor of Medicine in the Division of Rheumatology at the Johns Hopkins University School of Medicine and he holds joint appointments in the School of Medicine’s Departments of Pathology and Molecular Biology and Genetics as well as the Department of Molecular Microbiology and Immunology in the School of Public Health. He is the Co-Director for Basic Research for the Johns Hopkins Lyme Disease Research Center. Dr. Soloski received his Ph. D. in Microbiology from Rutgers, the State University of New Jersey and then completed post-doctoral training in Immunology at the University of Texas Health Science Center at Dallas, Southwestern Medical School prior to joining the faculty at the Johns Hopkins School of Medicine in 1983. The overarching theme of his research is understanding how infection can lead to long-term persistent symptoms. At this time, working with John Aucott, M.D. the Director of the Lyme Center at Hopkins, he is focused on understanding how the immune system contributes to the symptoms and severity of Human Lyme disease. He is very active and excited about the teaching of students, at all levels, about how the immune system evolved, how it protects us from infection and how it can contribute to disease.
Lyme disease is an inflammatory illness initiated by infection with Borrelia burgdorferi following a bite from an infected tick. Over the last four decades, the number of Lyme disease cases has risen sharply and it is now the most common vector-borne disease in the United States with over 300,000 cases each year. Symptoms of early Lyme disease can range from erythema migrans (EM) alone to systemic toxicity with signs of disseminated infection. Further, a number of patients with undetected and untreated early Lyme disease will develop late-onset musculoskeletal or neurological symptoms. While the acute infection and late-onset disease can be controlled by antibiotic therapy, in a subset of patients, arthritis with inflammation can be antibiotic-refractory. In addition, 10-20% of patients treated for early Lyme disease develop Post-Treatment Lyme Disease Syndrome (PTLDS), a condition with an unknown pathophysiological etiology that may have an immune component. The rising incidence of Lyme disease as well as the complexity of disease outcomes demands a deeper understanding of the immune-mediated process triggered by infection with B. burgdorferi.

In this presentation the current knowledge of the host immune response to B. burgdorferi in human Lyme disease will be reviewed with particular attention to how variation in host immunity may play a role in driving persistent symptoms versus return to health. Data from the study of the Johns Hopkins Slice cohort will also be presented that will address our hypothesis that the nature of the immune response plays a key role in the range of pathophysiological outcomes in human Lyme disease.