Accreditation Statement 2016 Columbia/LDA CME Lyme Conference

AMA Credit Designation Statement: The College of Physicians and Surgeons designates this live activity for a maximum 14 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Accreditation: This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of the College of Physicians and Surgeons of Columbia University and the Lyme Disease Association. The College of Physicians and Surgeons of Columbia University is accredited by the ACCME to provide continuing medical education for physicians.

Disclosure: The College of Physicians and Surgeons must ensure balance, independence, objectivity, and scientific rigor in its educational activities. All faculty participating in this activity are required to disclose to the audience any significant financial interest and/or other relationship with the manufacturer(s) of any commercial product(s) and/or provider(s) of commercial services discussed in his/her presentation and/or the commercial contributor(s) of this activity. When unlabeled uses are discussed, these will also be indicated.

Target Audience: The target population is physicians from all specialties, nurses, psychologists, scientists, public health workers. It is also open to the public, and Lyme disease educators generally attend. The geographic area being reached is nationwide. No special background is required for effective participation, although those whose practices contain a high proportion of Lyme disease patients and those whose research concentrates on Borrelia burgdorferi will receive the most benefit.

Learning Objectives: Practitioner should be better able to identify the multiple manifestations of Lyme disease. Practitioner should be better informed about new diagnostic advances. Practitioner should be able to conduct a broader differential diagnosis when faced with a patient with illness after tick bite. Awareness that there is a new mouse vaccine that helps diminish tick infection rates.

GIVE
The LDA received educational gift support for this conference from the Steven & Alexandra Cohen Foundation
SCHOLARSHIPS OFFERED
Lyme Disease Association, Inc. (LDA) is offering scholarships to the conference to eligible medical students, residents, post-doctoral candidates, fellows, veterinarians with equivalent status to the above, and nurse practitioner candidates.

REGISTRATION, HOTEL & CONFERENCE DETAILS go to the LDA website www.LymeDiseaseAssociation.org
Lyme & Tick-Borne Diseases: New Strategies to Tackle an Expanding Epidemic

AGENDA - SATURDAY October 15, 2016

Registration/Exhibits/Continental Breakfast (7:15 a.m.-8:00 a.m.)
Patri西亚 V. Smith, BA (8:00 a.m.-8:20 a.m.)
President, Lyme Disease Association, Inc.; Jackson, NJ
Conference Planner, Conference Organizing Committee
"Welcome, Remarks / Brief Overview of LDA, Lyme & TBD"

Introductions:
Brian A. Fallon, MD Course Director, Organizing Committee;
Saturday Afternoon Facilitator
Elizabeth Maloney, MD; Conference Organizing Committee;
Morning Session Facilitator
Robert D. Moir, PhD Keynote (8:20 a.m.-8:40 a.m.)
Assistant Professor Neurology, Harvard Medical School, MA
Assistant Professor, Laboratory Medicine & Pathology
"Amyloid-Beta, Alzheimer’s and Lyme Disease"

Judith Miklosy, MD, PhD (8:40 a.m.-8:55 a.m.)
Director, International Alzheimer Research Center, Switzerland
"Historic and Recent Evidence that Spirochetes Are Able to Reproduce the Clinical, Pathological and Biological Hallmarks of Alzheimer's Disease."

Brian A. Fallon, MD, MPH (8:55 a.m.-10:10 a.m.)
Professor of Psychiatry, Columbia University College of Physician & Surgeons
Director, Columbia Lyme and Tick-Borne Diseases Research Center
Director, Center for Study of Neuroinflammatory Disorders & Biobehavioral Medicine, NYUPI
"Acute & Chronic Neuropsychiatric Lyme Disease"

Morning Discussion Panel (10:10 a.m.-10:25 a.m.)
Kevin Eweitz, PhD (10:05 a.m.-10:15 a.m.)
Chief Operating Officer
US BIOLIGIC, Memphis, TN
"Community-Guided Ecological Immunization to Prevent Tick-Borne Disease"

Steve Zateckla, PhD, MBA (10:15 a.m.-10:25 a.m.)
"A One-Health Path to Prevent Lyme and Other Tick-Borne Diseases"

Rafal Tokarz, PhD (10:25 a.m.-12:05 p.m.)
Associate Research Scientist, Center for Infection & Immunity
Columbia University, Mailman School of Public Health, NY
"Viroxme Analysis in Ticks"

Lunch (12:05 p.m.-1:45 p.m.)
Brian A. Fallon, MD, MPH, Conference Director, Organizing Committee
Saturday Afternoon Facilitator

Kerry Clark, PhD, MPH (1:45 p.m.-2:20 p.m.)
Professor, Epidemiology & Environmental Health
University North Florida, Jacksonville, FL
"Update on Evidence of Lyme Borreliiosis in Ticks, Dogs and People in the Southeastern US"

Bobbi S. Pritt, MD (2:20 p.m.-2:55 p.m.)
Associate Professor, Laboratory Medicine & Pathology
Director, Clinical Parasitology Lab,Mayo Clinic, Rochester, MN
"Borrelia Mayonii: A New Cause of Lyme Disease in the Upper Midwestern US"

Timothy Lepore, MD, FACS (2:55 p.m.-3:30 p.m.)
General Surgery, Family Medicine
Surgeon at Nantucket Cottage Hospital, MA
"Tularemia"

Mid-Afternoon Discussion Panel (3:30 p.m.-4:45 p.m.)

Afternoon Coffee Break (3:45 p.m.-4:00 p.m.)

Evan M. Bloch, MBChB, MD, MS (4:00 p.m.-4:35 p.m.)
Assistant Professor of Biology, Dept. of Pathology
Johns Hopkins University, MD
"Babesia & Blood Supply: Lessons Learned"

Alessandra Luchini, PhD (4:35 p.m.-5:10 p.m.)
Assistant Professor, Applied Proteomics & Molecular Medicine
George Mason University, VA
"Nanotechnology & Proteomics: Improved Diagnostics & Therapeutics in the Era of Personalized Medicine"

Afternoon Discussion Panel (5:10 p.m.-5:20 p.m.)
Wrap up comments / questions (5:20 p.m.-6:00 p.m.)

NETWORKING RECEPTION (6:00 p.m.-7:00 p.m.)

AGENDA - SUNDAY October 16, 2016

(Breakfast on your own)

Registration/Exhibits/Registration/Exhibits (7:30 a.m.-8:00 a.m.)

Kenneth Liggner, MD Sunday Morning Facilitator
Brian A. Fallon, MD Sunday Afternoon Facilitator

Ying Zhang, MD, PhD (8:00 a.m.-8:35 a.m.)
Professor of Molecular Microbiology & Immunology
Johns Hopkins University, MD
"Eradication of Borrelia Persists for More Effective Treatment of Lyme Disease"

Daniel Cameron, MD MPH (8:35 a.m.-9:10 a.m.)
Internal Medicine
Private Practice, Mt. Kisco, NY
"Studies in Practice"

Lise Nigrovic, MD, MPH (9:10 a.m.-9:45 a.m.)
Associate Professor of Pediatrics & Emergency Medicine
Director, Education, Clinical Research Center; Senior Associate Physician in Medicine
Boston Children’s Hospital, MA
"Initial Management of a Child with Potential Lyme Disease"

Elizabeth I. Maloney, MD (9:45 a.m.-10:20 a.m.)
President, Partnership for Tick-Borne Diseases Education, a Lyme CME Provider
Family Physician, Wyoming, MN
"Lyme Carditis: A Front-Line Perspective"

Morning Discussion Panel (10:20 a.m.-10:40 a.m.)

Food Break (10:40 a.m.-11:15 a.m.)

Tim Sellati, PhD (11:15 a.m.-11:50 a.m.)
Distinguished Fellow & Chair Department of Infectious Diseases Drug Discovery Division
Southern Research Institute, AL
"Controlling the Inflammatory Response in Lyme Arthritis--What Mouse Model teaches Us about Human Disease"

Patricia K. Cogley MD, FAAN, FANA (11:50 a.m.-12:10 p.m.)
Assistant Professor Neurology, Mass General Hosp. Neurology Research, MA
"Diagnosing Neurologic Lyme Disease"

Mid-Morning Discussion (12:10 p.m.-12:35 p.m.)

John Aucott, MD (12:35 p.m.-1:00 p.m.)
Director, Johns Hopkins Lyme Disease Clinical Research Center
Assistant Professor of Medicine, Johns Hopkins University School of Medicine, MD
"Measuring the Human Immune Response in Lyme Disease"

C. Ben Beard, MS, PhD (1:10 p.m.-1:45 p.m.)
Chief, Bacterial Diseases Branch, Division of Vector-Borne Diseases
Associate Director for Climate and Health
National Center for Emerging and Zoonotic Infectious Diseases,
Centers for Disease Control & Prevention, Ft. Collins, CO
"The Expansion in Distribution of Ixodes scapularis & Ixodes pacificus and Reported Cases of Lyme Disease in the U.S."

Marna E. Ericson, PhD (1:45 p.m.-2:20 p.m.)
Director, Cutaneous Imaging Center
Department of Dermatology, University of Minnesota, MN
"Mechanisms of Persistence of Bartonella Species"

Afternoon Discussion Panel (2:20 p.m.-3:20 p.m.)

THANK YOUR FOR JOINING US!
Robert D. Moir, PhD, Keynote
Recent confirmation of a protective antimicrobial role for the amyloid-β (Aβ) peptide of Alzheimer’s disease (AD) has fueled debate on the role of infection in AD etiology. Aβ appears to belong to the antimicrobial peptide (AMP) family of immune proteins. AMPs are natural antibiotics and immunomodulators that act as the foot soldiers of innate immunity. Infection in transgenic animal models of AD leads to protective entrapment of microbes within β-amyloid plaques. Deposition of β-amyloid is a histological hallmark of AD. Here we discuss pathways leading to β-amyloid mediated microbial entrapment and the implications of Aβ’s emerging innate immune role for an AD-like disease link.

Judith Miklossy, MD, PhD
That pathogens suppress, subvert or evade host defences and establish chronic or latent infection had received little attention in the past. Various spirochetes, including Treponema pallidium (T. pallidum), Borrelia burgdorferi (B. burgdorferi) and several peripheral pathogen spirochetes have the ability to escape host defences and establish chronic infection. Various spirochetes, in an analogous way to T. pallidum, are involved in the pathogenesis of several chronic disorders including cerebrovascular disorders and in slowly progressive cognitive decline with dementia. T. pallidum, B. burgdorferi, and periodontal pathogen Treponemes (T. denticola, T. periodontium, T. vincentii, T. intermedius) persisting in the brain cause dementia and beta amyloid deposition. The two major histological forms of chronic neuroinvasions, namely the meningovascular form with cerebral infarcts and cognitive decline resulting in dementia have been clinically and pathologically confirmed more than 20 years ago, indicating that Borrelia burgdorferi can cause chronic Lyme disease and chronic neuroinvasions. Spirochetes, including Borrelia burgdorferi are able to reproduce in vitro and in vivo the pathological and biological hallmarks defining AD dementia. A strong statistically significant association between spirochetes and AD fulfills Hill’s criteria and confirms a causal relationship between spirochetes and dementia. Validation of these observations by historic and recent reports further confirm that senile plaques are made up by spirochetes and correspond to birosins. That host pathogen interactions in chronic spirochetal infection are identical to those occurring in AD indicates that escaping host immune reactions, spirochetes, including Borrelia burgdorferi, sustain chronic infection and cause, in addition to cerebral infarcts, slowly progressive dementia associated with amyloid deposition in the brain. Association of co-infecting pathogens and formation of the bacterial birosin further aggravates the degenerative process and the outcome of dementia. Importantly, these observations indicate that Alzheimer’s dementia can be prevented.

Brian A. Fallon, MD, MPH
Dr. Fallon will describe the discovery of a novel pathogen causing Lyme disease in the upper Midwestern United States. This new bacterium, preliminarily called Borelia mayonii, causes higher levels of spirocheteemia than what is seen with Borrelia burgdorferi, and has been associated with potential neurologic involvement and severe disease. Dr. Prült will discuss the tests that lead to the detection of B. mayonii, the clinical features observed so far, and the preferred diagnostic methods.

Steve Zatechka, PhD, MBA
A One-Health Path to Prevent Zoonotic Disease*
With 75% of all emerging infectious diseases being zoonotic in nature, the need of safe, effective, and cost-efficient prevention methods becomes a necessary endeavor. Recognizing the complexities of addressing a range of species (human, animal, insect), diseases, and ecologies, a One Health approach is best suited to cause an effective change. This talk will focus on one example of a One Health program – effective oral delivery of vaccines and therapeutics to wildlife and food animals. Data will be presented from successful approaches, focusing on a successful orally delivered vaccine targeting the wildlife disease reservoir for Lyme disease, the white-footed mouse.

Rafal Tokarz, PhD
Tick-borne diseases are the most common vector-borne illnesses in the United States. In a proportion of presumed tick-borne-associated infections, the etiologic agent is never identified, and the full range of tick-borne pathogens has not yet been explored. In contrast to bacterial pathogens, there is a limited understanding of the diversity of tick-borne viruses and their role in human infection. We are performing a virisome analysis of the three main human-biting ticks endemic to the New York metropolitan area, with the goal of uncovering novel viral agents and determining their geographic distribution and prevalence. For novel viruses with homology to known pathogens, we will design serological assays to examine sera from subjects with history of tick bites for evidence of spillover of these viruses into the human population.

Kerry Clark, PhD, MPH
Lyme disease continues to be a controversial subject in the southern United States. Based solely on 2-tiered serological laboratory confirmation test results, the disease continues to appear to be relatively rare in this region compared to highly endemic areas of the Northeast and Upper Midwest. However, some more recently obtained evidence challenges this belief. Borrelia burgdorferi sensu lato (Bbll) DNA has been detected in scores of human patients and dogs from southern states, many of which have no travel history to other regions. Bbll has also been isolated in culture from several human patients from Florida and Georgia, most of which have not been described in the scientific literature. DNA evidence of Bbll continues to be detected in lone star ticks (Amblyomma americanum), which may serve as a bridge vector of transmission to humans under certain circumstances. This report summarizes some of the published and unpublished evidence of Lyme Borelia infection in humans, dogs, and ticks from several southern states, attempts to help explain the disparity between surveillance case numbers and observed Lyme-like illness in the southern U.S., and provides insights into better understanding the ecology and epidemiology of Lyme disease in the South.

Bobbi S. Pritt, MD
Dr. Bobbi Pritt will discuss the discovery of a novel pathogen causing Lyme disease in the upper Midwestern United States. This new bacterium, preliminarily called Borrelia mayonii, causes higher levels of spirocheteemia than what is seen with Borrelia burgdorferi, and has been associated with potential neurologic involvement and severe disease. Dr. Pritt will discuss the tests that lead to the detection of B. mayonii, the clinical features observed so far, and the preferred diagnostic methods.

Timothy Lepore, MD, FACS
Tularemia is a tick-borne pathogen traditionally associated with rabbit hunting. Tularemia in Massachusetts has a very definite history, only appearing after 1935 with the introduction ofMidwestern rabbits. The islands of Nantucket and Martha’s Vineyard have a very different history with Tularemia involving a pneumonic presentation. The discussion will be about the ecology of this atypical presentation.

Evan M. Bloch, MBChB, MD, MS
Babesia and the Blood Supply: Lessons Learned
Babesiosis is the clinical illness named for infection by Babesia, a genus of tick-borne, intraerythrocytic protozoan parasites that are endemic to parts of the United States (US). Babesia is readily transfusion transmissible: following an increase in naturally acquired- (i.e. tick-borne) and transfusion transmitted babesiosis (TTB) in the US, TTB, the overwhelming majority of which is caused by Babesia microti, has been recognized as the foremost infectious risk to the US blood supply for which licensed donor screening is still not available. Change is underway whereby new tools have been developed, including novel assays and pathogen reduction technology. The purpose of this talk is to discuss Babesia as a model for understanding the response to an emerging infectious disease in the context of blood safety. The talk will include lessons that were learned during the evolution from recognition of risk, to selection of a mitigation strategy, research and development through to implementation and policy. While nuanced challenges may be specific to Babesia, the same principles apply to other pathogens, both known as well as emerging.

SYNOPSES OF LECTURES
Bartonella spp., are vector-borne stealth pathogens. Our goal is to understand the role of biofilms in manifestation of prolonged bacteremia and resulting pathologies caused by persistent or persister bacteria that are tolerant to current Lyme antibiotics in vitro and in vivo. The implications of these findings for more effective treatment of persistent Lyme disease will be discussed.

Borrelia persisters. In addition, I will discuss various drug combinations and their effectiveness to eradicate more resistant Borrelia persister including round bodies and biofilm-like microcolonies in vitro systems. In stark contrast, arthritis-susceptible C3H mice lack basal levels of cAMP production and dampen the release of pro-inflammatory mediators elicited by CD14-dependent p38 MAPK activity increases binding of STAT3 and SP1 to their cognate sites on the now accessible IL-10 promoter, facilitating increased IL-10 production. Thus, cAMP and CD14-p38-MAPK signaling; which, in combination, is responsible for increased production of the anti-inflammatory cytokine IL-10 and decreased production of potent pro-inflammatory and arthritogenic cytokines, including TNF. CD14 regulates chromatin structure through modification of histones while CD14-dependent p38 MAPK activity increases binding of STAT3 and SP1 to their cognate sites on the now accessible IL-10 promoter, facilitating increased IL-10 production. Thus, CD14 dependent IL-10 production and dampen the release of pro-inflammatory mediators elicited by Borrelia burgdorferi by changing the epigenetic 'landscape'. In stark contrast, arthritis-susceptible C3H mice lack basal levels of CD14 compared to those of their disease-resistant B6 counterparts and thus are ill-equipped to mitigate the damaging consequences of B. burgdorferi-induced TNF through production of IL-10. Intriguingly, reciprocal regulation of IL-10 and TNF by CD14 and CD14-dependent mechanisms are operable in human primary peripheral blood monocytes and CD14-enhancing drugs show therapeutic efficacy in our mouse model of Lyme arthritis.

John Aucott, MD
The host immune response plays a critical role in determining the outcome of infectious diseases. In addition, ongoing host immune and inflammatory responses can perpetuate symptoms that persist and drive illness. Studies of the human immune response in Lyme disease are showing the complexity of the interaction between Borrelia burgdorferi and the innate and adaptive immune responses. Data will be presented to show the unique immune responses among patients with early Lyme disease before and after treatment.

C. Ben Beard, MS, PhD
Ixodes scapularis or deer ticks are now found in over 41% of counties in 43 states across the United States. This marks an increase of 44.7% in the number of positive counties since 1996. The number of counties where I. scapularis has now established has more than doubled over the last 20 years. Over a similar period of time, the number of high incidence counties for Lyme disease in the northeastern U.S. increased by greater than 320% and in the north-central U.S. by over 250%. The numbers of Lyme disease cases in the U.S. continue to increase, and the geographic distribution of both Lyme disease and its tick vectors is greater than ever before. These findings highlight the critical importance of safe and effective prevention and control tools and methods.

Marna E. Ericson, PhD
Lyme disease is our major human spirochetal infection. The nervous system is a favored target organ. This talk will discuss the diagnosis of neurologic Lyme disease in practical terms. It will cover when to suspect the infection, characteristic as well as unusual syndromes, the main differential diagnoses, and appropriate workup including how to interpret the variety of testing. The goal is not to miss the diagnosis of neurologic Lyme disease, so that appropriate and timely treatment may be provided.

Ying Zhang, MD, PhD
In this presentation, I will discuss the problem of post-treatment Lyme disease syndrome (PTLDS) and its possible causes, the phenomenon of Borrelia persistence despite antibiotic treatment in animal models, the demonstration of Borrelia persister bacteria that are tolerant to current Lyme antibiotics in vitro. I will then discuss our recent work on identification of FDA-approved drugs that are more active than the current Lyme antibiotics against Borrelia burgdorferi persister. In addition, I will discuss various drug combinations and their effectiveness to eradicate more resistant Borrelia persister, including FDA-approved FDA-approved drugs and new mouse models to understand disease persistence and stealth mechanism.

Elizabeth L. Maloney, MD
This presentation begins with a case presentation before moving to a broader discussion of Lyme carditis. It traces the evolution of the patient's clinical picture from multiple erythema migrans lesions to third degree heart block. In addition to reviewing the literature on Lyme carditis, the presentation highlights some unique features of the case-patient's course that may inform the care of other patients.

Tim Sellati, PhD
Genetically profoundly influences disease severity in the murine model of Lyme borreliosis, caused by the spirochetal bacterium Borrelia burgdorferi. Infected C57BL/6 (B6) and C3H/HeN (C3H) mice develop very mild and severe Lyme arthritis, respectively. Expression of the immunosuppressive cytokine interleukin-10 (IL-10) by B6, but not C3H mice has long been associated with these strain differences in disease presentation. However, the underlying mechanism(s) of genotype-specific IL-10 regulation remained elusive. Herein, we reveal a cyclic AMP (cAMP)-mediated mechanism of IL-10 regulation in B6 mice that is absent in C3H mice, which provides insight into the clinical spectrum of human Lyme disease, particularly those suffering from treatment-refractory arthritis. We show that bone marrow-derived monocytes (BMDMs) from B6 mice mount a more tempered, protective immune response to borreliial infection by virtue of the action of CD14 and CD14-p38-MAPK signaling; which, in combination, is responsible for increased production of the anti-inflammatory cytokine IL-10 and decreased production of potent pro-inflammatory and arthritogenic cytokines, including TNF. CD14 regulates chromatin structure through modification of histones while CD14-dependent p38 MAPK activity increases binding of STAT3 and SP1 to their cognate sites on the now accessible IL-10 promoter, facilitating increased IL-10 production. Thus, CD14 and CD14 regulate IL-10 production and dampen the release of pro-inflammatory mediators elicited by B. burgdorferi by changing the epigenetic 'landscape'. In stark contrast, arthritis-susceptible C3H mice lack basal levels of CD14 compared to those of their disease-resistant B6 counterparts and thus are ill-equipped to mitigate the damaging consequences of B. burgdorferi-induced TNF through production of IL-10. Intriguingly, reciprocal regulation of IL-10 and TNF by CD14 and CD14-dependent mechanisms are operable in human primary peripheral blood monocytes and CD14-enhancing drugs show therapeutic efficacy in our mouse model of Lyme arthritis.

Lise Nigrovic, MD, MPH
Children commonly get Lyme disease. Initial diagnostic decisions must be made before Lyme disease test results are available. I will present three common clinical case scenarios of children with potential Lyme disease. I will then review the best available evidence to guide clinical decision-making.

Patricia K. Coyle MD, FAAN, FANA
Lyme disease and other tick-borne diseases. He is the author of Lyme disease and related disorders since 1988. He has published articles on Lyme disease in peer-reviewed scientific journals and has presented poster abstracts and talks at national and international conferences on Lyme disease and other tick-borne diseases.