International Symposium 2-2

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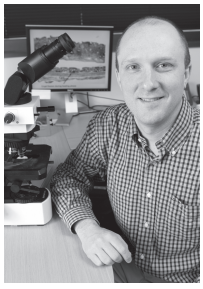

Education

1987 B.A., Integrated Science Program Physiology, Northwestern University, Evanston, IL
1995 M.D., Ph.D., Medical Scientist Training Program, Northwestern University, Evanston, IL
1995–1999 Resident, Anatomic Pathology, Washington University Medical Center, St. Louis, MO
1999–2000 Clinical Fellow, Pathology, Washington University Medical Center, St. Louis, MO

Academic positions/employment

2002–2003 Instructor, Department of Developmental Biology, Washington University Medical School, St. Louis, MO
2003–2009 Assistant Professor, Department of Pathology & Immunology, Department of Molecular Biology & Pharmacology, Washington University School of Medicine, St. Louis, MO

University and hospital appointment & committes

2002– Director, Histology Core, Department of Molecular Biology and Pharmacology, Washington University Medical School, St. Louis, MO
2007 Liaison Committee on Medical Education, ad hoc
2008– Strategic Planning Committee for the Office of Post Graduate Affairs
2008– Co-Organizer of Washington University Immunology retreat
2009– Graduate School admissions committee

Honors and awards

1987 Phi Beta Kappa
1992 Sigma Xi Research Award
1993 Journal of Cell Science Travel Fellowship to visit laboratory of Dr. Birgit Lane
1993 International Union of Pure and Applied and Biophysicists Travel Fellowship
2001 Laser Capture Microdissection and Macromolecular Analysis of Normal Development and Pathology Travel Award
2005 Pew Scholar
2009 American Society for Clinical Investigation
2010 Kavli Fellow
2010 Pluto Society
2012 American Gastroenterological Association (AGA) Fellowship
2013 Organizer, Gastrointestinal Tract XV: Epithelia, Microbes, Inflammation and Cancer
August 11-15, 2013 Steamboat Colorado (FASEB conference)

Model of host microbial interactions in inflammatory bowel disease

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The normally peaceful coexistence of intestinal microbes and the host is broken in inflammatory bowel disease (IBD) which occurs in genetically susceptible individuals. Meta-analyses of genome wide association studies have discovered many new susceptibility loci associated with interesting genes. In European ancestral populations IBD, these studies have found ~163 susceptibility loci and many overlap between the two major forms of this disease, Crohn's disease and ulcerative colitis. However, this is not solely a genetic disease. Many of the susceptibility loci are present in high frequency in target populations and the incidence of this disease has been increasing in recent times. Thus, it has long been recognized that environmental factors, potentially including pathogenic infections as well as shifts in commensal microbiota, are also required to trigger disease. Thus a key area for the advancement in our understanding of IBD pathogenesis is the better understanding host susceptibility in the context of interaction with the microbial environment. Improved methods to analyze the commensal microbiota and functionally test its components as well as better methods to define dysbiosis and its potential role in disease have been crucial to improve our understanding of host-microbial interactions that affect disease. Lastly, identification of microbial triggers including viruses in experimental systems of IBD suggests a potential role in IBD. Understanding the precise microbial and immune triggers of IBD in a genetic context will hopefully lead to a better understanding of the pathogenesis of this disease and the discovery of novel therapeutic approaches.