BIOGRAPHICAL SKETCH

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NAME: BEAMER, GILLIAN

eRA COMMONS USERNAME: GBEAMER

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date	FIELD OF STUDY
The University of Pennsylvania, Phila, PA	BA	05/1996	Paleobiology
The University of Pennsylvania, Phila, PA	VMD	05/2000	Veterinary Medicine
The Manhattan Veterinary Group, NYC, NY	Internship	06/2001	Medicine/Surgery
The Ohio State University, Columbus, OH	PhD	06/2009	Tuberculosis
The Ohio State University, Columbus, OH	Residency DACVP	09/2008	Pathology
The Ohio State University, Columbus, OH	Research Scientist	11/2011	Tuberculosis

A. Personal Statement

I am an assistant professor and am awaiting promotion to associate professor (anticipated by year-end 2021), having already received departmental and school support of my promotion. I am a scientist, veterinarian, and board-certified pathologist with >15 years of tuberculosis research. My goal is to translate knowledge from animal models of tuberculosis (TB) to improve human lives. My research focuses on host genetic control of differential susceptibility to *Mycobacterium tuberculosis* (*M.tb*). Because humans are neither inbred, nor gene-knockouts, we now use the Diversity Outbred (DO) mouse population to model and understand complex phenotype-genotype interactions. Like the human population, the DO mouse population contains individuals who develop pulmonary TB rapidly and others who establish long-term asymptomatic control. As a board-certified pathologist, I have a special interest in the granuloma and its structure-function relationships as well as passion, training, and expertise to lead this project along with Dr Metin Gurcan (who has complementary expertise in computer science, machine learning, and image analysis). For RFA-AI-20-57 “Understanding the Role of the *M. tuberculosis* Granuloma in Tuberculosis (TB) Disease and Treatment Outcomes,” we propose to couple whole lung gene expression data with data from hematoxylin & eosin (H&E) stained lung sections. Using AI algorithms that we developed (named image2gene), we will predict the intra-granuloma locations of differentially expressed genes, producing two-dimensional (2D) models that map gene expression of susceptible and resistant hosts to regions within granulomas. These developments will improve visualization and quantification of granuloma function jointly with structure, allowing a more precise inference of cell function and granuloma evolution over time. Below are publications showing joint authorship, and application of image analysis to TB granulomas:

a. Tavolara TE, Niazi MKK, Ginese M, Piedra-Mora C, Gatti DM, **Beamer G**, Gurcan MN. Automatic discovery of clinically interpretable imaging biomarkers for *Mycobacterium tuberculosis* supersusceptibility using deep learning. EBioMedicine. 2020 Nov 6;62:103094. doi: 10.1016/j.ebiom.2020.103094. Epub ahead of print. PMID: 33166789; PMCID: PMC7658666.

b. Koyuncu D, Niazi MKK, Tavolara T, Abeijon C, Ginese M, Liao Y, Mark C, Gower A, Gatti D, Kramnik I, Gurcan M, Yener B**, Beamer G**. Tuberculosis biomarkers discovered using Diversity Outbred mice. The Lancet Infectious Diseases MS #S-20-08067 In Review Dec 2020

c. Kus P, Gurcan MN, **Beamer G**. Automatic Detection of Granuloma Necrosis in Pulmonary Tuberculosis Using a Two-Phase Algorithm: 2D-TB. Microorganisms. 2019 Dec 7;7(12):661. doi: 10.3390/microorganisms7120661. PMID: 31817882; PMCID: PMC6956251.

d. Niazi MK, Dhulekar N, Schmidt D, Major S, Cooper R, Abeijon C, Gatti DM, Kramnik I, Yener B, Gurcan M, **Beamer G**. Lung necrosis and neutrophils reflect common pathways of susceptibility to *Mycobacterium tuberculosis* in genetically diverse, immune-competent mice. Dis Model Mech. 2015 Sep;8(9):1141-53. doi: 10.1242/dmm.020867. Epub 2015 Jul 23. PMID: 26204894; PMCID: PMC4582107.

B. Positions and Honors

Employment

2000 - 2001	Veterinary Intern, VCA Manhattan Veterinary Group, Manhattan, NY
2000 - 2003	Veterinarian (Staff), North Shore Animal League, Port Washington, NY
2002 - 2003	Veterinarian (Relief), Bide-A-Wee Animal Shelter, Manhattan, NY
2003 - 2004	Graduate Research Associate, The Ohio State University, Columbus, OH
2004 - 2007	Post-Doctoral Fellow, The Ohio State University, Columbus, OH
2007 - 2011	Senior Research Associate, The Ohio State University, Columbus, OH
2009 - 2011	Clinical Instructor, The Ohio State University, Columbus, OH
2012 - 2020	Assistant Professor, Tufts University, Department of Biomedical Sciences, North Grafton, MA
2015 - current	Assistant Professor, Tufts University, Clinical and Translational Science Institute, Boston, MA
2012 - current	Assistant Professor, Tufts University, Dept of Infectious Disease and Global Health

Other Professional

2000 - 2017	# VT1042, Veterinary Licensure ME
2000 - current	Member, American Veterinary Medical Association
2000 - 2003	# 008497, Veterinary Licensure NY
2000 - 2003	Member, NY State Veterinary Medical Society
2008 - current	Member & Diplomate, American College of Veterinary Pathologists
2010 - 2011	Member, Center for Microbial Interface Biology, The Ohio State University

Honors

1994	Summer Research Student, The Jackson Laboratory
1995	Thuron Undergraduate Research Award, University of Pennsylvania
1998	Summer Pathology Research Fellow, Armed Forces Institute of Pathology
2006	Travel Award Veterinary Research Day, The Ohio State University
2006	Travel Award, American College of Veterinary Pathologists
2007	Travel Award OSUMC Research Day, The Ohio State University
2007	P.E.O. Scholar Award, P.E.O. International
2007	Travel Award Veterinary Research Day, The Ohio State University
2008	Young Investigator Award, American College of Veterinary Pathologists
2008	Travel Award, American College of Veterinary Pathologists
2008	Harold W Casey Scholarship, American College of Veterinary Pathologists
2008	NIH Health Disparities Loan Repayment Award, National Institutes of Health
2008	Roche Applied Sciences Seminar Award, The Ohio State University
2009	Roche Applied Sciences Seminar Award, The Ohio State University
2011	Becoming Faculty Workshop, Burroughs Wellcome Fund
2012	Tufts University CSVM nominee, Ellison Medical Foundation
2012	Tufts University CSVM nominee, Burroughs Wellcome Fund
2013	Tufts University Nominee, Smith Family Foundation
2015	Junior Faculty Research Award, Cummings School of Veterinary Medicine
2018	Tufts University Academic Leadership Development Program
2021	Tufts CSVM Zoetis Award for Veterinary Research Excellence

C. Contributions to Science

1. Modeling TB in complex mouse populations. About six years ago, we began using the Diversity Outbred (DO) mouse population because its genetic diversity, heterozygosity, and phenotypic range better model humans than inbred strains. When infected with *M.tb*, DO mice show a huge response range (like humans); key disease features: neutrophil-associated inflammatory signatures, granuloma necrosis, and cavitation (like humans); and we detect novel phenotypes that do not occur in inbred strains (i.e. "supersusceptibility" or early morbidity within 8 weeks of infection). This provides a validated model of human pulmonary TB. We have also identified multiple new loci associated with primary *M.tb* infection and pursue candidate genes in mechanistic studies elsewhere. Our publications, ongoing studies, and preliminary data establish a successful baseline to determine the effects of host genetics on ID93+GLA-SE as a candidate therapeutic vaccine.

    a. Kurtz SL, Rossi AP, **Beamer GL**, Gatti DM, Kramnik I, Elkins KL. The Diversity Outbred mouse population is an improved animal model of vaccination against tuberculosis that reflects heterogeneity of protection. mSphere. 2020 Apr 15;5(2). pii: e00097-20.
    b. Smith CM, Proulx MK, Olive AJ, Laddy D, Mishra BB, Moss C, Gutierrez NM, Bellerose MM, Barreira-Silva P, Phuah JY, Baker RE, Behar SM, Kornfeld H, Evans TG, **Beamer G**, Sassetti CM. Tuberculosis susceptibility and vaccine protection are independently controlled by host genotype. MBio. 2016 Sep 20;7(5).
    c. Niazi MK, Dhulekar N, Schmidt D, Major S, Cooper R, Abeijon C, Gatti DM, Kramnik I, Yener B, Gurcan M, **Beamer G**. Lung necrosis and neutrophils reflect common pathways of susceptibility to *Mycobacterium tuberculosis* in genetically diverse, immune-competent mice. Dis Model Mech. 2015 Sep;8(9):1141-53.
    d. Harrison DE, Astle CM, Niazi MK, Major S, **Beamer GL**. Genetically diverse mice are novel and valuable models of age-associated susceptibility to *Mycobacterium tuberculosis*. Immun Ageing. 2014 Dec 16;11(1):24.

2. Artificial intelligence methods to extract diagnostic and prognostic features from *M.tb*-infection. Our current R01 with Dr Metin Gurcan at Wake Forest and Dr Bulent Yener at RPI applies innovative and cross-disciplinary methods to TB research. This includes producing algorithms to automatically detect and quantify microscopic features of granulomas; using machine learning, data mining, and network analysis to develop signatures that classify forms of TB; and generating models to predict outcomes of *M.tb* infection using genotypic and biomarker signatures from DO mice.

    a. Tavolara TE, Niazi MKK, Ginese M, Piedra-Mora C, Gatti DM, **Beamer G**, Gurcan MN. Automatic discovery of clinically interpretable imaging biomarkers for *Mycobacterium tuberculosis* supersusceptibility using deep learning. EBioMedicine. 2020 Nov 6
    b. Kus P, Gurcan MN, **Beamer G**. Automatic detection of granuloma necrosis in pulmonary tuberculosis Using a two-phase algorithm: 2D-TB. Microorganisms. 2019 Dec 7;7(12). pii: E661. doi: 10.3390/microorganisms7120661.
    c. Niazi MKK, **Beamer G**, Gurcan MG, "An application of transfer learning to neutrophil cluster detection for tuberculosis: efficient implementation with nonmetric multidimensional scaling and sampling," Proc. SPIE 10581, Medical Imaging 2018: Digital Pathology, 1058108 (6 March 2018).
    d. Niazi MKK, **Beamer G**, Gurcan MG, "A computational framework to detect normal and tuberculosis infected lung from H and E-stained whole slide images," Proc. SPIE 10140, Medical Imaging 2017: Digital Pathology, 101400J (1 March 2017).

3. Defining mechanisms of host responses to *M.tb* with and without vaccination. In addition to working as an independent PI, I happily participate in team science. We have recently shown that manipulating *M. bovis* BCG improves its ability to protect inbred mice from *M.tb*-challenge under multiple conditions. We have also investigated interleukin (IL)-21, IL-10, and MHCII haplotype all shapes immune responses in inbred mice, although each contributes, none alone dictate final TB disease outcomes.

a. Moliva JI, Duncan MA, Olmo-Fontánez A, Akhter A, Arnett E, Scordo JM, Ault R, Sasindran SJ, Azad AK, Montoya MJ, Reinhold-Larsson N, Rajaram MVS, Merrit RE, Lafuse WP, Zhang L, Wang SH, **Beamer G**, Wang Y, Proud K, Maselli DJ, Peters J, Weintraub ST, Turner J, Schlesinger LS, Torrelles JB. The Lung Mucosa Environment in the Elderly Increases Host Susceptibility to Mycobacterium tuberculosis Infection. J Infect Dis. 2019 Jul 2;220(3):514-523. doi: 10.1093/infdis/jiz138.
b. Moliva JI, Hossfeld AP, Sidiki S, Canan CH, Dwivedi V, **Beamer G**, Turner J, Torrelles JB. Selective delipidation of *Mycobacterium bovis* BCG enables direct pulmonary vaccination and enhances protection against *Mycobacterium tuberculosis*. Mucosal Immunol. 2019 May;12(3):805-815. doi: 10.1038/s41385-019-0148-2.
c. Moliva JI, Hossfeld AP, Canan CH, Dwivedi V, Wewers MD, **Beamer G**, Turner J, Torrelles JB. Exposure to human alveolar lining fluid enhances *Mycobacterium bovis* BCG vaccine efficacy against *Mycobacterium tuberculosis* infection in a CD8+ T-cell-dependent manner. Mucosal Immunol. 2018 May;11(3):968-978. doi: 10.1038/mi.2017.80.
d. Booty MG, Barreira-Silva P, Carpenter SM, Nunes-Alves C, Jacques MK, Stowell BL, Jayaraman P, **Beamer G**, Behar SM. IL-21 signaling is essential for optimal host resistance against *Mycobacterium tuberculosis* infection. Sci Rep. 2016 Nov 7;6:36720. doi: 10.1038/srep36720.

4. Identify how *M.tb* bacilli persist and contribute to reactivation TB. By using the Cornell model of latent *M.tb* infection, we showed that CD271+ bone marrow mesenchymal stem cells protected living *M.tb* bacilli from the same antibiotic therapy that sterilizes the lung and spleen. This has important ramifications for treatment failure and reactivation TB, because stem cells allow bacilli to evade immunity and antibiotics.

   a. **Beamer G**, Major S, Das B, Campos-Neto A. Bone marrow mesenchymal stem cells provide an antibiotic-protective niche for persistent viable *Mycobacterium tuberculosis* that survive antibiotic treatment. Am J Pathol. 2014 Dec;184(12):3170-5.

D. Research Support and/or Scholastic Performance

Ongoing

R01HL145411 BEAMER, GILLIAN (PI) 01/15/2019-12/31/2023
NIH Predicting tuberculosis outcomes using genotypic and biomarker signatures
Role PI

R21AI155003 MARTINOT (PI) 09/01/2020-06/30/2022
NIH Myeloid-Derived Suppressor Cells in Tuberculosis Granuloma Structure and Function
Role Co-I

R01AI137424 TELFORD (PI) 05/01/2018-04/30/2022
NIH Emergence of tick-borne encephalitis in North America
Role Pathologist

Completed

PR0093 BEAMER, GILLIAN (PI) 08/07/2018-08/06/2019
Vaxil Bio Research Service Agreement to test novel TB vaccines
Role PI

RSA015 BEAMER, GILLIAN (PI) 04/02/1017-03/21/2018
WaveGuide Waveguide Antibody Testing
Role PI

R21AI115038 BEAMER, GILLIAN (PI) 04/15/2015-04/14/2018
NIH Genetic-based susceptibility to pulmonary tuberculosis
Role PI

RSA AORIAN, RAFFI (PI) 01/01/2020-12/31/2020
UMass Histopathological study of a functional food de-wormer
Role Co-I

OSRO/BGD/505/USA ANWER, SAWKAT (PI) 11/27/2018-7/31/2019
FAO Technical Support for Development of Pedagogic Skills in Nine Veterinary Schools and a Veterinary Professional Accreditation System in Bangladesh
Role Faculty facilitator