BIOGRAPHICAL SKETCH

NAME: Liang, Yun

eRA COMMONS USER NAME (agency login): yunliang07

POSITION TITLE: Assistant Professor

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	Completion Date	FIELD OF STUDY
Tsinghua University, China	BS	07/2007	Biological Sciences
University of California San Diego – Salk Institute	PhD	03/2013	Biomedical Sciences

A. PERSONAL STATEMENT

I am an Assistant Professor in the Department of Medical Microbiology and Immunology at the University of Wisconsin-Madison. My training has focused on the study of human health using both basic and translational approaches. I received my PhD in Biomedical Sciences from the University of California San Diego and the Salk Institute for Biological Studies where I studied the role of three-dimensional genome organization in stem cell differentiation. After graduation in 2013, I led efforts on the research and development of next-generation-sequencing-based genetic testing for personalized medicine as the Head of Clinical Development, AdmeraHealth, Inc. From 2015 to 2018, I pursued postdoctoral training in inflammatory and autoimmune diseases at the University of Michigan.

Current research in my lab is aimed at understanding the molecular basis of sexual dimorphisms in human immunology and immune-associated diseases. Specifically, we are interested in determining the role of sex-biased immune processes in metabolic and reproductive health. Joining the ERP program will allow us to interact with scientists with shared passion, to provide training to the next generation of scientists in this field, and to further enrich ERP with our research program.

B. POSITIONS AND HONORS

Positions and Employment

2005 - 2007 Undergraduate Student Researcher, National Laboratory of Protein Therapeutics, China
2007 - 2013 Graduate Student Researcher, Salk Institute for Biological Studies, San Diego, CA
2013 - 2013 Scientist, AdmeraHealth, Inc., South Plainfield, NJ
2013 - 2014 Head, Clinical Development, AdmeraHealth, Inc., South Plainfield, NJ
2015 - 2015 Head, Clinical Operations, AdmeraHealth, Inc., South Plainfield, NJ
2015 - 2016 Research Fellow, Department of Dermatology, Medical School, University of Michigan, Ann Arbor, MI
2016-2018 Research Investigator, Department of Dermatology, Medical School, University of Michigan, Ann Arbor, MI
2018 - Assistant Professor, Department of Medical Microbiology & Immunology, University of Wisconsin-Madison, Madison, WI

Other Experience and Professional Memberships

2012 - 2012 Consultant, Oxbridge Biotech
2012 - 2013 Project Manager, Oxbridge Biotech
2012 - 2014 Board Member, Tsinghua Biotech-Pharma Network
2014 - Ad hoc reviewer, Journal of Medical Genetics
2014 - Ad hoc reviewer, American Journal of Life Sciences

2015 - Member, Society for Investigative Dermatology
2017 - Member, National Psoriasis Foundation

Honors

2004 Scholarship for Academic Excellence, Tsinghua University
2005 Scholarship for Academic Excellence, Tsinghua University
2006 Scholarship for Academic Excellence, Tsinghua University
2010 Featured Research Publication, Salk News Release
2011 Best Cited Publication, Current Opinion in Cell Biology
2012 Selected Presentation, Cold Spring Harbor Meeting
2013 Highlighted Publication, ESC & iPSC News
2015 Award of Excellence and Dedication, AdmeraHealth, Inc.
2016 Training Fellowship in Cell and Molecular Dermatology (T32)
2016 Albert Kligman Fellowship, Society for Investigative Dermatology
2016 Invited Participation in the Research Fellow Retreat, Society for Investigative Dermatology
2016 Highlighted Research, American Academy of Allergy, Asthma and Immunology
2017 Publication Highlighted by Nature Immunology, TIME, The Independent, Medical Daily, Michigan Health Lab
2017 Invited Participation in the NPF Trainee Symposium, National Psoriasis Foundation
2017 Travel Award, Cell Symposia: Human Immunity
2017 Elsevier Family Support Award

C. CONTRIBUTION TO SCIENCE

1. Many autoimmune diseases feature strikingly increased prevalence in females (~78% overall and up to 95% for specific diseases). In an effort to understand the cause of this female-biased susceptibility to autoimmune conditions, I have identified a VGLL3-regulated gene network as a previously unknown promoter of sex-biased autoimmunity in human skin. This finding sheds light on the fundamental differences in immune regulation between men and women, and may lead to novel therapeutic strategies for the management of autoimmune skin diseases.

a. **Liang Y**, Tsoi LC, Xing X, Sarkar MK, Berthier CC, Stuart PE, Nair RP, Elder JT, Voorhees JJ, Kahlenberg JM, Gudjonsson JE (2017). A gene network regulated by the transcription factor VGLL3 as a promoter of sex-biased autoimmune diseases. Nat Immunol,18(2)152-160. PMCID: PMC5289297.

b. **Liang Y**, Kahlenberg JM, Gudjonsson JE (2017). A vestigial pathway for sex differences in immune regulation. Cell Mol Immunol, doi: 10.1038/cmi.2017.28. PMCID: PMC5520416.

2. Pustular skin disorders are a category of difficult-to-treat and potentially life-threatening conditions, with their pathogenic mechanisms largely unknown. Through molecular analysis of three pustular skin disorders, generalized pustular psoriasis, palmoplantar pustulosis, and acute generalized exanthematous pustulosis, I demonstrated that STEAP1 and STEAP4 play a role in establishing the inflammatory profiles central to these disorders. This finding opens up possibilities in targeting the STEAP pathways for the treatment of pustular skin conditions.

a. **Liang Y**, Xing X, Sarkar MK, Wolterink L, Voorhees JJ, Kahlenberg JM, Harms PW, Johnston A, Gudjonsson JE (2016). Six transmembrane epithelial antigens of the prostate comprise a novel inflammatory nexus in pustular skin disorders. J Allergy Clin Immunol. pii S0091-6749(16)31351-3. PMCID:PMC5385276.

3. In studies on the pathogenic mechanisms of inflammatory skin conditions, I have helped elucidate the role of IL17A signaling in inverse, erythrodermic, and plaque psoriasis, and the functional consequences of glucocorticoid deficiency on inflammatory responses in keratinocytes. In addition, I have helped establish a link between super-enhancers in the skin and complex skin diseases such as psoriasis and atopic dermatitis.

a. Swindell WR, Sarkar MK, **Liang Y**, Xing X, Gudjonsson JE (2016). Cross-Disease Transcriptomics: Unique IL-17A Signaling in Psoriasis Lesions and an Autoimmune PBMC Signature. J Invest Dermatol. 136(9):1820-30. PMCID:PMC5234565.

b. Xing X, **Liang Y**, Sarkar MK, Wolterink L, Swindell WR, Voorhees JJ, Harms PW, Kahlenberg JM, Johnston A, Gudjonsson JE (2016). IL-17 Responses Are the Dominant Inflammatory Signal Linking Inverse, Erythrodermic, and Chronic Plaque Psoriasis. J Invest Dermatol. 136(12): 2498-2501. PMCID:PMC5123949.

c. Sarkar MK, Kaplan N, Tsoi LC, Xing X, **Liang Y**, Swindell WR, Hoover P, Aravind M, Baida G, Clark M, Voorhees JJ, Nair RP, Elder JT, Budinova I, Getsios S, Gudjonsson JE (2017). Endogenous glucocorticoid deficiency in psoriasis promotes inflammation and abnormal differentiation. J Invest Dermatol. pii: S0022-202X(17)31157-0. PMCID: PMC5545780.

d. Klein RH, Lin Z, Hopkin AS, Gordon W, Tsoi LC, **Liang Y**, Gudjonsson JE, Andersen B (2017). GRHL3 binding and enhancers rearrange as epidermal keratinocytes transition between functional states. PLoS Genet. 13(4):e1006745. PMCID: PMC5425218.

4. Chromatin regions are known to occupy particular domains in the three-dimensional space of the cell nucleus. However, how the three-dimensional organization of the genome affects gene expression remains obscure. My studies in this area have demonstrated that from fruit flies to humans, the genome undergoes three-dimensional reorganization in relation to the nuclear pores during cell differentiation, and this reorganization is critical for the proper induction of developmental genes. The work has provided insights into the basic principles of gene regulation by nuclear architecture and molecular explanations for the tissue-specific nature of diseases caused by mutations in nuclear pore proteins.

a. Capelson M, **Liang Y**, Schulte R, Mair W, Wagner U, Hetzer MW (2010). Chromatin-bound nuclear pore components regulate gene expression in higher eukaryotes. Cell. 140(3):372-83. PMCID:PMC3821818.

b. **Liang Y**, Hetzer MW (2011). Functional interactions between nucleoporins and chromatin. Curr Opin Cell Biol. 23(1):65-70. PMCID:PMC3753814.

c. **Liang Y**, Franks TM, Marchetto MC, Gage FH, Hetzer MW (2013). Dynamic association of NUP98 with the human genome. PLoS Genet. 9(2):e1003308. PMCID:PMC3585015.

d. Buchwalter AL, **Liang Y**, Hetzer MW (2014). Nup50 is required for cell differentiation and exhibits transcription-dependent dynamics. Mol Biol Cell. 25(16):2472-84. PMCID:PMC4142618.

5. I have participated in mechanistic studies on the function of endostatin and Hsp90 during carcinogenesis. The work on endostatin has elucidated the structural basis for its interaction with physiological receptors, which is instrumental to the development of bioengineering approaches to improve the stability of endostatin-based drugs. The work on Hsp90 has revealed its role in promoting angiogenesis in cancer, leading to therapeutic approaches targeting this activity.

a. Fu Y, Wu X, Han Q, **Liang Y**, He Y, Luo Y (2008). Sulfate stabilizes the folding intermediate more than the native structure of endostatin. Arch Biochem Biophys. 471(2): 232-9.

b. Fu Y, Chen Y, Luo X, **Liang Y**, Shi H, Gao L, Zhan S, Zhou D, Luo Y (2009). The heparin binding motif of endostatin mediates its interaction with receptor nucleolin. Biochemistry. 48(49):11655-63.

c. Wang X, Song X, Zhuo W, Fu Y, Shi H, **Liang Y**, Tong M, Chang G, Luo Y (2009). The regulatory mechanism of Hsp90alpha secretion and its function in tumor malignancy. Proc Natl Acad Sci U S A. 106(50):21288-93. PMCID:PMC2795546

d. Song X, Wang X, Zhuo W, Shi H, Feng D, Sun Y, **Liang Y**, Fu Y, Zhou D, Luo Y (2010). The regulatory mechanism of extracellular Hsp90a on matrix metalloproteinase-2 processing and tumor angiogenesis. J Biol Chem. 285(51): 40039-49. PMCID:PMC30009816

Complete List of Published Work in MyBibliography:
https://www.ncbi.nlm.nih.gov/sites/myncbi/yun.liang.1/bibliography/51428012/public/?sort=date&direction=ascending

D. Research Support

Ongoing Research Support

UW-Madison Start-up Funds (Liang, PI) 11/15/18 – 12/31/99

NIH/NIAMS K01-AR073340 (Liang, PI) 04/16/19-03/31/23
"The Role of VGLL3 in Sexually Dimorphic Interferon-Driven Inflammation"
The aims of this grant are to determine sex dispairities in transcriptional responses to IFNs and their regulation by VGLL3, to establish the chromatin mechanism for sex-dependent ISG profiles, and to determine the in vivo role of VGLL3 in regulating IFN-mediated immune processes.

Past Research Support

Discovery Grant (Liang, PI) 08/01/17-07/31/18
National Psoriasis Foundation
"PLA2-mediated Lipid Metabolism as a Novel Axis in Psoriasis Pathogenesis"
The major goal of this project is to investigate the role of PLA2-mediated lipid metabolism in inflammatory processes associated with psoriasis pathogenesis.
Role: Principal Investigator

Translation Accelerator Grant (Liang, PI) 06/01/2017-05/31/18
NIAMS P30AR069625 (Clark, PI)
Human Skin Disease Resource Center
"Development of a Novel Skin-based Biomarker Panel for Lupus Erythematosus"
This project seeks to test the potential use of VGLL3 and the autoimmune genes it regulates as biomarkers for lupus erythematosus.
Role: Subcontract PI

2R01AR069071-03 (Gudjonsson, PI) 09/16/15-07/31/20
NIH/NIAMS
"Role of IL-13 and the IL-13 associated rs20541 risk variant in the pathogenesis of psoriasis"
The major goal of this project is to investigate the mechanism by which IL13 and the IL-13 associated risk variant influences the pathogenesis of psoriasis.
Role: Co-Investigator

1R01AI130025-01 (Gudjonsson, PI) 04/01/17-03/31/22
NIH/NIAID
"Role of the gender biased transcription factor VGLL3 in promoting autoimmune responses in SLE"
The major goals of this project are to investigate how VGLL3 contributes to the activation of cells relevant to SLE pathogenesis including various types of immune cells; and to establish mechanisms by which VGLL3 is activated in autoimmune conditions.
Role: Co-Investigator

5T32 AR07197-38 (Elder, PI) 08/01/15-10/31/16
NIH/NIAMS
'Training Grant for Cell and Molecular Dermatology"
Role: Trainee