BIOGRAPHICAL SKETCH

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NAME: Laurie, Sonia Jessica

eRA COMMONS USER NAME: SONIALAURIE

POSITION TITLE: Postdoctoral Fellow, Duke-UNC Immunotherapy Training Grant

EDUCATION/TRAINING

INSTITUTION & LOCATION	*DEGREE* *(if applicable)*	*Start Date* *MM/YYYY*	*End Date* *MM/YYYY*	*FIELD OF STUDY*
University of North Carolina, Chapel Hill, NC	*NCI T32 grant*	09/2021	Present	Transplant Immunology
University of North Carolina, Chapel Hill, NC	*NIGMS K12 grant*	09/2018	8/2021	Transplant Immunology
Emory University, Atlanta, GA	PHD	08/2012	08/2018	Transplant Immunology
Tufts University, Boston, MA	*NIGMS R25 grant*	09/2009	07/2012	Pathology
Beloit College, Beloit, WI	BS	08/2005	05/2010	Biology

A. Personal Statement

My long-term research focus is on the role of lymphocytes in immunotherapy and human immunology. Specifically, I aim to define the regulatory chromatin states of innate lymphoid cells in health and am interested in exploring how epigenetic programming influences responsiveness to therapeutic biologics. My academic training and research experience have provided me with an excellent background in multiple biological disciplines including immunology, molecular biology, genetics, and biochemistry. As an undergraduate, my primary research interests was in determining the genetic etiology of metabolic disease in rats and examining the interaction between coinhibitory signaling molecules on T cells and their ligands on tumor cells. As a postbaccalaureate fellow at Tufts University, I continued to explored the molecular mechanisms that underlie age-associated immunogenetic resistance and susceptibility to protozoan infections. My work as a doctoral student at the Emory University Transplant Center served to unify my interests in genetics and immunobiology. My thesis work in the Laboratory of Dr. Mandy L. Ford contributed to basic and translational aspects of our understanding of transplantation biology and immune tolerance to allografts, and I specifically worked to identify the mechanisms by which CD28 blockade alters donor-reactive T cell programming by shifting the balance of costimulatory and coinhibitory molecule expression. During the first three years of my postdoctoral training with Dr. Jonathan Serody, I have continued to build on my previous training in cellular and molecular immunology as I have developed in vitro and in vivo models to discover key epigenetic regulatory features that control the chromatin landscape and plasticity of innate lymphoid cells. This work has provided novel approaches to treating graft-versus-host disease following hematopoietic stem cell transplantation.

B. Positions and Honors

Positions and Employment	
09/2021-Present	Postdoctoral fellow, Duke-UNC Immunotherapy Training Grant, University of North Carolina, Chapel Hill, NC Laboratory of Dr. Jonathan Serody, Department of Microbiology & Immunology
09/2018-08/2021	NIGMS IRACDA K12 Postdoctoral Scholar, Seeding Postdoctoral Innovators in Research and Education Program, University of North Carolina, Chapel Hill, NC Laboratory of Dr. Jonathan Serody, Department of Microbiology & Immunology
2020	Visiting Professor, Johnson C. Smith University, Department of Natural Sciences and Mathematics, Charlotte, NC
2012-2018	Ph.D., Immunology and Molecular Pathogenesis, Laney Graduate School Emory University, Atlanta, GA, Laboratory of Dr. Mandy Ford, Department of Surgery
2009-2012	NIGMS Post-baccalaureate Research Experience Program (PREP) Scholar

Other Experience and Professional Memberships		
2020-2021	Voting Member	UNC Institutional Review Board F
2020-2021	Subcommittee co-chair	Scholars in Training Professional Development Subcommittee, National Society for Advancement of Biology Education Research
2020-2021	Co-leader	UNC Microbiology & Immunology Peer Mentorship Network Programming Committee
2020-pres	Curriculum design team	Scientists Promoting Antiracist Conversations (SPACE) Mini-Course, UNC School of Medicine, Office of Graduate Education
2020-pres	Member	National Council on Undergraduate Research
2020-pres	Member	Society for Advancement of Chicanos and Native Americans in Science (SACNAS), UNC-CH Chapter
2019	Member	
2017-2018	Trainee Member	The American Society of Hematology
2015-2018	Trainee Member	The American Association of Immunologists
2017-2018	Secretary & Founding Member	Society for Advancement of Chicanos and Native Americans in Science (SACNAS), Emory Chapter
2016-2018	Member	Association of Women in Science, National Chapter
2016-2017	Secretary & Founding Member	Emory Immunology Graduates' Group
2014-2018	Member	AST Trainee & Young Faculty Committee of Practice
2014-2018	Trainee Member	The American Society of Transplantation (AST)

C. Contribution to Science

Early Career:

My early career contributions were focused on enhancing our understanding of the genetic mechanisms underlying metabolic and immune disease. In my undergraduate research experience, I worked to define and compare the effects of a high fat diet in salt sensitive rats that contained consomic chromosomal substitutions from resistant Brown Norway (BN) rats to determine which chromosome conferred resistance to metabolic syndrome. Our results indicated that chromosome 8 substitution rats possess a trait that protects them from elevated levels of cholesterol, triglycerides, and glucose while on a high fat diet. Our findings were published in The Journal of Nutrigenetics and Nutrigenomics in 2012. After graduation, I accepted a two-year NIH-funded post-baccalaureate fellowship at Tufts University, where my project involved characterizing the role of T lymphocytes in protection against intraerythrocytic parasite infections. I established that MHC class II molecules are absolutely required to clear infection with the bloodborne protozoan parasite Babesia microti. My findings suggest that the IL-21 receptor is necessary for protection and that phagocytosis is an indispensible component of acute resistance to infection. My findings indicated that the differential parasite burden observed in resistant BALB/c and susceptible DBA/2J mice may be due to a difference in IFN-γ production, although follow-up studies are needed. Additionally, I helped to establish two congenic lines of resistant mice that have subsequently been used in mapping studies to identify alleles responsible for protection against age-acquired susceptibility to babesiosis.

a) Woods, L. C. S., Woods, B., Leitschuh, C., Laurie, S. J., Jacob, H. J. Rat Chromosome 8 Confers Protection against Dyslipidemia Caused by a High-Fat/Low-Carbohydrate Diet. 2012. *Journal of Nutrigenetics and Nutrigenomics*, 5:81-93.

b) Laurie, S. J., Vannier, E., Wortis, H. H. Phagocytosis, Interferon-γ, IL-21, and $CD4^+$ MHC class II-restricted T cells in resistance to Babesia microti infection. *J Immunol*, 188:43.14. 2012.

c) Vannier, E., Silver, Z. Wilson, C., Su, J., Chiam, J., Laurie, S. J., Telford, S., Gelfand, J., Krause, P., Wortis, H. Cellular basis for clearance of the protozoan parasite Babesia microti in immunocompromised hosts. *J Immunol*, 188:43.13. 2012.

C. Contribution to Science (con't)

Graduate Career: My graduate research contributions focused on understanding the mechanisms by which T lymphocytes mediate cellular rejection of allografted tissues following solid organ transplantation. Immunotherapeutic strategies to prevent rejection following transplantation frequently involve targeted blockade of T cell costimulatory pathways. My project assessed the contribution of coinhibitory receptors to the control of alloreactive T cell responses following transplantation in humans and animal models. I demonstrated that the T cell coinhibitory molecules 2B4 and TIGIT are expressed on $CD28^{null}$ effector memory $CD4^+$ and $CD8^+$ T cells that are associated with freedom from rejection following renal transplantation in humans. Further exploration of these pathways in a murine model of transplantation indicates that while 2B4 functions to control alloreactive T cells by limiting their glycolytic metabolism, proliferation and recruitment into the alloreactive anti-donor response. Finally, my work demonstrated that the programming of antigen specific $CD8^+$ T cells responding to graft and pathogen are dissimilar, and that antigen-specific $CD8^+$ T cells primed by a skin graft contract faster than those primed by infection, yet are able to expand more rapidly upon rechallenge. These data suggest that graft-elicited $CD8^+$ antigen specific T cells are maintained in a less terminally-differentiated state compared to pathogen-elicited $CD8^+$ antigen specific T cells. Taken together, these data suggest that the surface marker expression, metabolic prolife, and functional capacity of T cells depends on the priming conditions and may be used to predict immunologic risk following transplantation.

a) Xie, J., Chen, C. W., Laurie, S. J., Zhang, W., Otani, S., Martin, G. S., Coopersmith, C. M., and Ford, M. L. Increased Attrition of Memory T Cells During Sepsis Requires 2B4. 2019. *JCI Insight,* 4(9): e126030.

b) Laurie, S. J., Liu D., Wagener M. E., Stark P. C., Terhorst, C., Ford, M. L. 2B4 Mediates Inhibition of $CD8^+$ T Cell Responses via Attenuation of Glycolysis and Cell Division. 2018. *Journal of Immunology,* 201(5):1536-1548.

c) Bozeman, A. M.[#], Laurie, S. J.[#], Haridas, D., Wagener, M. E., Ford, M. L. Transplantation Preferentially Induces a KLRG-1^{lo} $CD127^{hi}$ Differentiation Program in Antigen-Specific $CD8^+$ T Cells. 2018. *Transplant Immunology,* 50:34-42.
[#]These authors contributed equally to this work.

d) Laurie, S. J., Ford, M. L. Epigenetic Remodeling in Exhausted T Cells: Implications for Transplantation Tolerance. 2017. *Transplantation*, 101(5):894-895.

e) Cortes, M.C., Laurie, S. J., Mathews, D. V., Winterberg, P., Larsen, C. P., Adams, A. B., Ford, M. L. Belatacept-Resistant Rejection is Associated with Increased Pre-Transplant Frequencies of $CD28^+$ $CD4^+$ T_{EM} in Human Renal Transplant Recipients. 2017. *American Journal of Transplantation*, 17(9):2350-2362.

C. Contribution to Science (con't)

Postdoctoral Career: As a postdoctoral fellow, my research in Dr. Jonathan Serody's lab at the Lineberger Comprehensive Cancer Center at UNC Chapel Hill has focused on hematopoietic stem cell transplantation (HSCT), the preferred treatment for a variety of blood malignancies. Unfortunately, HSCT is limited by the development of acute graft-versus-host disease (aGvHD). Type II innate lymphoid cells (ILC2s) are immune cells that play an important role in maintaining mucosal homeostasis, and our lab has previously shown that ILC2s in the gastrointestinal tract (GI) are sensitive to conditioning therapy prior to HSCT. Strikingly, we have demonstrated that the infusion of activated donor ILC2s markedly reduces aGvHD-associated mortality. We hypothesize that the generation of pro-inflammatory cytokines after allogeneic HSCT alters the phenotype of ILC2 cells facilitated by epigenetic changes and ILC plasticity, leading to the expansion of pathogenic ILC1 cells at the expense of protective ILC2 cells. To address this hypothesis, we are using high-throughput multiome assays to evaluate the chromatin landscape of ILC2s and establish the role of "ex-ILC2s" in the pathology of aGVHD. With the use of ChIP-seq, ATAC-seq, and protein and mRNA analyses, we have discovered a critical role for IL-12 and IL-1b in regulating the chromatin architecture of ILC2s, redirecting these cells to an alternate, pro-inflammatory ILC1-like fate. We are now to re-enforcing G9a expression and increase H3K9me1-2 marks around *Tbx21*, *Tnf*, *Eomes*, and *Ifng*. Taken together, this work will provide new insights into mechanisms by which innate lymphoid cell precursors are epigenetically regulated, providing novel approaches to treating aGvHD following HSCT.

a) Laurie, S. J., Foster, J., Bruce, D. W., Bommiasamy, H., Pattenden, S. G., Davis, I. J., and Serody, J. S., Proinflammatory cytokines induce pro-pathogenic G9a-mediated epigenome remodeling of innate lymphoid cells in the lower GI tract after allogeneic HSCT, *Manuscript in preparation.*

b) Anders, C. K., Woodcock, M. Van Swearingen, A. E. D., Moore, D. T., Sambade, M. J., Cuaboy, L. A., Garrett, A. L. McKinnon, K., Laurie, S. J., Robeson, A. C., Kolupaev, O. V., Iannone, M., Cowens, K., Bortone, D. ,Calhoun, B. C., Wilkinson, A. D., Carey, L. A., Jolly, T. Muss, H., Reeder-Hayes, K., Kaltman, R., Jankowitz, R., Gudena, V., Olajide, O., Perou, C. M., Dees, E. C., Vincent, B. G., and Serody, J. S. Evaluating the Efficacy of a Priming Dose of Cyclophosphamide Prior to Pembrolizumab to Treat Metastatic Triple Negative Breast Cancer. 2021. *J Immunother Cancer*, under revision.

c) Bruce, D. W., Kolupaev, O. V., Laurie, S. J., Bommiasamy, H., Stefanski, H., Blazar, B. R., Coghill, J., and Serody, J. S. Third Party Type 2 Innate Lymphoid Cells Prevent and Treat GI Tract GvHD. 2021. *Blood Advances*, **in press.**

d) Xu, N., Palmer, D., Shou, P., Laurie, S. J., Bommiasamy, H., Robeson, A., Wills, C., Dotti, G., Vincent, B., Restifo, N., and Serody, J. S., Single Cell Sequencing Reveals a Critical Role for STING Agonist in enhancing CAR-T against breast cancer. 2021. J Exp Med, 218(2) 20200844.

D. Additional Information: Research Support and Scholastic Performance

Research Support

09/2021-Present	Postdoctoral fellow, Duke-UNC Immunotherapy Training Grant (PI: Serody/Chao, NCI T32CA211056-01A1) , University of North Carolina, Chapel Hill, NC Laboratory of Dr. Jonathan Serody, Department of Microbiology & Immunology
09/2018-08/2021	NIGMS IRACDA SPIRE Scholarship, PI: Dr. Donald Lysle, University of North Carolina at Chapel Hill, Chapel Hill, NC, NIGMS: K12GM000678-20
2014-2018	NRSA Ruth L. Kirschstein National Research Service Award, PI: Sonia Laurie, Emory University School of Medicine, Atlanta, GA, NIAID: F31AI114250-01
2014	Recipient of NIAID RO1 Sub-Award (PI: Dr. Mandy L. Ford), Emory University School of Medicine, Atlanta, GA, NIAID: RO1AI104699-01A1
2013-2014	Emory University NIH T32 Institutional Training Grant, PI: Dr. Brian D. Evavold, Emory University School of Medicine, Atlanta, GA, NIAID: T32AI007610-15
2009-2012	Post-baccalaureate Research Experience Program Scholar (PREP), Tufts University School of Graduate Biomedical Science, Boston, MA, NIGMS: GM066567-16

Scholastic Performance: Laney Graduate School, Emory University, Atlanta, GA

YEAR	SCIENCE COURSE TITLE	GRADE	YEAR	SCIENCE COURSE TITLE	*GRADE*
2012	Concepts of Immunology	B+	2014	Advanced Graduate Research	A
2012	Introduction to Research	S	2014	Advanced Graduate Seminar	A
2012	Basic Biomedical & Biol. Sciences (I)	B	2014	Dissertation Research	A
2012	Laboratory Rotations	A	2015	Ethics: Conducting Global Work	S
2012	Colloquium Immunology	A	2015	Ethics: Responsibility of PhD	S
2013	Virology	A	2015	Advanced Graduate Research	A
2013	Basic Biomedical & Biol. Sciences (II)	A	2015	Advanced Graduate Seminar	A
2013	Introductory Graduate Seminar	A	2015	Dissertation Research	A
2013	Laboratory Rotations	A	2015	Dissertation Research	A
2013	Scholarly Integrity Core Class	S	2016	Advanced Graduate Seminar	A
2013	Advanced Graduate Research	A	2016	Ethics: Authorship & Peer-Review	S
2013	Annual Reviews of Immunology	A	2016	Advanced Graduate Seminar	A
2013	Introductory Graduate Seminar	A	2016	Dissertation Research	A
2013	Colloquium Immunology	A	2016	Dissertation Research	A
2013	Graduate School Workshop	S	2016	Advanced Graduate Seminar	A
2014	Advanced Graduate Research	A	2017	Dissertation Research	A
2014	Current Topics in Immunology	A	2017	Advanced Graduate Research	A
2014	Introductory Graduate Seminar	A-	2017	Advanced Graduate Seminar	A
2014	Teaching Assistantship	S	2017		
2014	Ethics: Human Subjects Research Risk	S	2017		
2014	Advanced Graduate Research	A	2018	**Cumulative GPA**	**3.93**
2014	Advanced Graduate Seminar	A	2018		
2015	Stat. Design & Analysis of Experiments	A			