Volume 1 | 2015

Society for Leukocyte Biology

EDITOR'S NOTES — Vijaya Iragavarapu, i-SLB Editor

Hello everyone! Welcome to the Spring issue of 2014 iSLB newsletter. Throughout the upcoming issues, we will be adding a new section called "Profile of an SLB member" and alternate this with a "Short interview with the JLB cover author" with the intention of getting to know SLB members and recognizing the contribution of JLB authors. We will be contacting SLB members for an interview regarding their profiles and those whose research article is featured on the cover of JLB during 2015. We hope that you enjoy these sections. In this issue, we will profile Dr. Rachel Caspi who was a Council member and an advisor to the Women and Diversity Committee. My Co-Editor Amanda Brown had the pleasure of interviewing this year's Bonazinga Award winner, Dr. Gail Bishop. We hope that you enjoy reading about Dr. Bishop's research and viewpoints. We are excited that Dr. Bishop will be the keynote speaker for the Women & Diversity Roundtable session at the 2015 SLB meeting in Raleigh, N.C. We encourage all of you to attend this year's meeting. Other features in this issue that merit your attention include the note from the Editor-in-Chief of The Journal of Leukocyte Biology, and the interview of Dr. Kari Ann Shirey who was the 2013 G. Jeanette Thorbecke Award Winner. Please read the notes from the Women & Diversity Committee. Stephania Libreros and Cortney Armstrong, Junior Editors, are pleased to announce the newly formed Members in Transition and Training Focus Group (MTTG). The MTTG is a student, fellows and early career scientist driven group to promote career development within the Society. We hope you enjoyed reading about the MTTG and that you will get involved in this great group.

President's Message

Ann Richmond, President

As we move into the spring, it is my pleasure to communicate updates of our society. A new initiative that I'd like to bring attention to is the Members in Transition and Training Group (MTTG). This was initially a concept introduced by SLB Council Member Bruce Levy, who had a vision of a committee that was led by junior researchers for junior researchers. In 2014 an industrious group of SLB members began forming this work group and in 2015 they will conduct their first special session at the annual SLB meeting in Raleigh. Look for more details about this exciting session and a new webpage dedicated to their mission which will be coming soon.

Speaking of the website, our redesigned presence continues to serve the community and expand the quality and quantity of its content on a regular basis. You can view the Individual Development Plan session from 2014 for a year round resource, read archived iSLB issues, and even listen to recordings of lectures from past meeting highlights. Coming later this year we will be posting a special interview between our current and past JLB Editor-in-Chiefs.

The journal continues to be a prime resource for members to submit and read about the latest developments in leukocyte biology. See later in this issue a special message from our Editor-in-Chief, Luis Montaner, calling for manuscript submissions.

I look forward to seeing everyone in Raleigh, North Carolina for an exciting scientific program that was planned by our dedicated and creative 2015 program chairs, Elizabeth Kovacs and Nick Lukacs. The program looks fantastic, so I encourage each of you to submit your research abstracts so that you may present your work at the meeting. We are dedicated to expanding the number of travel awards available so be certain to apply for travel awards and research awards as appropriate.

It is our SLB community that has facilitated the networking, career development, scientific excellence, collaborative science, and ongoing learning of leukocyte biologists across our planet. Therefore, let us renew our membership and reach out to find new colleagues to welcome into the SLB community. Our Membership Committee is working hard to grow our membership base and our best resource is the connections of our members. Through growing our membership and through increased participation in the society, we can all expand our impact and increase the benefit for all members.

Great things are still to come for SLB. We'll be welcoming Robert Clark as our new President in 2016 when we will also travel to Verona, Italy with our partners from the Neutrophil group for a one-of-a-kind spectacular meeting chaired by Marco Cassatella. This will all lead to our 2017 50th Anniversary meeting chaired by Liwu Li which is sure to bring SLB forward to the beginning of another great 50 years of science.

Please enjoy this issue of iSLB and thank you for being a part of this tremendous community.

What you cite and where you submit impacts all of us: Why we need JLB to be strong

Luis J. Montaner, PhD
Editor-In-Chief, JLB

I am reaching out to you to share our excitement for JLB and to ask that you keep JLB at the forefront for your manuscript submissions in 2015. We need you to submit your best science to JLB to insure the journal's continued move upward in quality and recognition.

JLB science impact goes well beyond its readers. It is essential to have a robust and vibrant journal with a large number of excellent papers reflecting the diversity of SLB and extending into new scientific areas. In addition to serving as the main medium for disseminating the science of the society, JLB also serves an important role for SLB through revenue generation. 100% of journal proceeds go directly to support:

- Peer community building & networking (yearly SLB meetings)
- Junior investigator professional development training
- Women and diversity initiatives for junior/established faculty
- Graduate student meeting scholarships
- Member Journal Clubs
- General SLB operations
- JLB improvement (i.e., our new journal format and website, etc.)

We are the ones that determine JLB's impact factor. Publishing in JLB benefits us as a scientific community: not shareholders, as is the case for many commercial journals our members may submit their best science to. I know the pressure to publish by impact factor alone is intense, yet the irony is that WE can decide where our best science goes and what science we cite each time we draft a paper, and this drives impact factor! So we do have a direct hand in determining which journals will remain in the higher- or lower-tier, respectively. Importantly, JLB has maintained its impact whereas many journals have dropped as the on-line journal landscape has grown. The reason JLB has remained strong is that:

- JLB is a brand name that stands for excellence and a well known community of peers
- JLB has no submission fee and no color charges
- Reviews completed within 23 days
- Reviewers/editors maintain high standards for content
- Our science gets cited in new manuscripts being drafted

We need you to act for JLB to excel. I am asking you directly to consider all factors when drafting your next manuscript and deciding where to submit it, including:

- How your manuscript will define the future impact

PIZZA AND PUBS PROGRAM

Welcome to our newest Pizza n' Pub Group – Virginia Tech!

Sign up to get $200 per year to local groups to be used at regular meetings!

To qualify, your group needs to have at least 10 people, of which at least two must be PIs who are SLB members, and one student/postdoc who will join SLB as a new member.

To apply, simply send us an email (slb@faseb.org) with the names and email addresses of the members of your group, a brief description of the lab group/journal club/discussion group, and a paid application for a student membership (you may download this off the SLB Web site (www.leukocytebiology.org).

factor of where you submit.

- Who ultimately will benefit from the revenue generated from your next high impact science
- Actively citing journals you support when relevant to the introduction or discussion of a new manuscript, thereby contributing to the impact factor of those journals.

For all the reasons above, I am calling on our membership to take pride in our progress and take an active role in building a stronger JLB beyond 2015. We are fortunate that we have a journal that is entirely run by, serves and supports our community. Thank you for your support if you already publish with us. I look forward to our greater future "investment" into a stronger JLB this year and in the years to come. It is in the end, up to us.

Women & Diversity

Vijaya Iragavarapu-Charyulu (Chair W&D)

The Women and Diversity Committee (W&D) is excited to have Dr. Gail Bishop, the 2015 Bonazinga Award winner, as their keynote speaker at the 2015 SLB Annual Meeting in Raleigh, N.C. This year's topic for the Town Hall Discussion is "Networking Strategies". The interactive topics of discussion are to include networking strategies for collaborations, for job searches and for job promotion. As you register for the meeting, please also sign-up for the Monday, September 28th breakfast meeting of W&D session.

The Women & Diversity committee is pleased to announce that it is sponsoring the "W&D Paper of the Year Award" again. All SLB members that belong to the "Women and diversity" category and have published first-author, corresponding-author or a senior-author paper within last 5 years are invited to apply for this award. The only criterion that will be used for the evaluation of the paper is its importance based on the number of citations per year. The winner will be invited to give a short presentation about his/her research at a session during the Annual SLB Conference. As well, the awardee will also be invited to participate in the panel discussion during the W&D Forum session and will be featured on the W&D web page. A travel stipend is provided with the award plaque at the time of presentation. To apply for the W&D "Paper of the Year Award" applicants should use the SLB on-line Award submission system. Submit a statement indicating how you fit the "W&D" category, a short CV and the full reference for one selected article published within the last 5 years with the calculated citations per year according to the Web of Science database. The citation report must also be included with the application. The applicant may be either first author, senior author, or corresponding author. Equal contribution will be given to first, senior and corresponding author. In order to receive this award, the applicant must register and submit an abstract(s) for the meeting.

Do not forget to visit the Women and Diversity Committee web page. Two new categories under committee resources have been added. Please visit the website to access Focus on Diversity and Internships and Professional Development Resources. Also visit the SLB Forum where members can share their thoughts on the diversity discussion.

Members in Transition and Training Focus Group (MTTG)

Silvia M. Uriarte and Bruce Levy (SLB Councilors)

In case you are wondering what is MTTG? Be prepared because you will start to read more and see more announcements related to this acronym! The SLB council has proposed the MTTG as an idea to generate an identity and voice for the younger membership of SLB. Council members Silvia M. Uriarte and Bruce Levy are helping to sponsor MTTG and several SLB members are already organizing this important effort. During last year's SLB meeting, we had a great start and recruited two energetic, talented, and amazing SLB trainee members *Juhi Bagaitkar*, currently a postdoctoral fellow at Washington University School of Medicine in St. Louis, and *Stephania Libreros*, a graduate student at Charles E. Schmidt College of Medicine at Florida Atlantic University. Since last October Juhi and Stephania have been enthusiastically working and significant progress has been made towards delineating the goals of this new task force. The main goals of MTTG are focused on career development and exciting science. The MTTG is working to provide an opportunity for SLB trainee members to have the chance to interact with and receive career-building advice from well-known scientists in the leukocyte biology field in a more personalized, targeted and informal manner. This task force will be governed by the SLB trainee members, and it will provide a forum for communication with a new voice in the Society. The MTTG will help coordinate SLB-sponsored events, including a dedicated session at the upcoming SLB meeting.

If you are a graduate student, post-doctoral fellow or early stage junior faculty, then please join us in the MTTG. Stay tuned to the SLB website to keep up to date on the progress of the MTTG activities and details regarding the MTTG session for the upcoming 2015 SLB meeting. Juhi and Stephania have already selected eminent scientific speakers to participate in the session!

Gail Bishop, Ph.D.

Holden Chair of Cancer Biology | College of Medicine Distinguished Professor of Microbiology | Co-Director, Cancer Immunology & Immunotherapy Group | Associate Director for Basic Science Research | Professor of Microbiology | Professor of Internal Medicine

The Bishop lab studies molecular mechanisms of lymphocyte activation. By working to understand how cells communicate with one another and their environment, we address questions of how normal immunity, autoimmunity and malignancy are regulated. A major focus of our work is understanding signals delivered to cells by members of the Tumor Necrosis Factor Receptor (TNFR) superfamily of molecules. This large family of receptors is expressed on many cell types, but is of special importance to regulating the activities of cells of the immune system. One of the family members that we study, CD40, is expressed primarily on immune cells that present antigen to T lymphocytes, including B lymphocytes, the immune cells that produce antibodies. CD40 plays a critical role in B cell function. In parallel, we study a protein produced by Epstein-Barr virus, called latent membrane protein 1 (LMP1), which plays important roles in the development of EBV-associated lymphomas that can arise in immunosuppressed patients, and also contributes to disease flares in Lupus and arthritis. LMP1 is a remarkable mimic of CD40 signals, but in an amplified and dysregulated manner. Using combined approaches of cell line studies, freshly isolated cells (mouse and human) and genetically altered mice, we are investigating how signaling by CD40 and LMP1 differ.

A key difference is how the two receptors regulate the function of a family of cytoplasmic proteins called TNFR associated factors (TRAFs). We are also studying how TRAF function and regulation contributes to cell signaling by TNFR family members, including CD95, TNFR2, CD27, BAFFR and TACI, as well as the innate immune TLR receptors. Some of the molecules we have begun to study regulate T cell activating and apoptotic signals delivered by the T cell antigen receptor. We have developed a method using gene targeting by homologous recombination in somatic cells to produce cell lines specifically and completely deficient in single of multiple types of TRAF molecules, as well as mice that lack particular TRAFs in certain cell types. Another line of investigation in the lab involves understanding how innate immune receptor signals interact with signals via adaptive receptors, such as antigen receptors. We are examining such signals and their roles in optimizing the use of immune cells in cellular vaccines to protect from infectious organisms and malignant cells.

Education

- BA, Biology, St Olaf College
- MS, Oncology, University of Wisconsin, Madison
- PhD, Cellular and Molecular Biology, University of Michigan, Ann Arbor
- Post Doctoral, Immunology, The University of North Carolina, Chapel Hill

Education/Training Program Affiliations

- Biosciences Graduate Program
- Department of Microbiology Graduate Program
- Interdisciplinary Graduate Program in Immunology
- Interdisciplinary Graduate Program in Molecular and Cellular Biology
- Interdisciplinary Graduate Program in Translational Biomedicine
- Medical Scientist Training Program

Center, Program and Institute Affiliations

- Cardiovascular Research Center
- Center for Immunology and Immune-based Diseases
- Center on Aging
- Fraternal Order of Eagles Diabetes Research Center
- Helen C. Levitt Center for Viral Pathogenesis
- Holden Comprehensive Cancer Center
- Institute for Clinical and Translational Science
- Stephen A. Wynn Institute for Vision Research

An Interview with 2015 Bonazinga Award Winner, Gail Bishop

Q. While complete knockout of TRAF3 results in early embryonic death, what was the initial discovery in your laboratory that revealed to you that this protein could be playing distinct functions in different types of immune cells?

A. Starting in my postdoctoral fellowship, I had a long-term interest in T-cell and B-cell interactions leading to signaling after activation. When I became a new Asst. Prof., CD40 had recently burst on the scene and there was a knowledge gap about how it signals. During my graduate work I studied the immune response to HSV, and it was really serendipity that I had kept up with the Herpesvirus literature. The Herpesvirus EBV assumes a latent infection in nearly everyone and does not cause a problem except for under conditions of immunosuppression. In this situation, when EBV partially emerges from latency, the virus makes a protein called latent membrane protein 1 (LMP1). EBV is associated with the development of B-cell malignancies. In reading a report of newly identified proteins that associate with the long tail of LMP1, the protein sequence of one of the LMP1-associated proteins looked very familiar to me. It turned out that the adapter protein binding to the intracytoplasmic

domain of LMP1 was the first CD40-associated binding protein, called TRAF3. Because EBV has tropism for B cells, we began to study LMP1 and CD40 signaling to B cells in parallel. We found that LMP1 mimics what CD40 does but in a dysregulated way, so this provided a model to determine how CD40 signaling is normally regulated. LMP1 binds the same TRAFs that CD40 does. The binding sites on LMP1 and CD40 for TRAFs 1, 2, 3, and 5 are highly overlapping, making it difficult to determine the roles for individual TRAFs in signaling by these receptors. Knockout mice lacking TRAFs 2 and 3 showed early lethality, preventing their use in studying TRAFs in mature cell types. Therefore, we developed what I call the "poor man's KO" using the same technique of gene targeting by homologous recombination, but modified for use in somatic cell lines. In this manner we found to our great surprise that in B cell line subclones specifically and completely lacking TRAF3, CD40 signaling became amplified, but LMP1 signaling was severely compromised. We then became very curious to know - if TRAF3 serves two receptors in the same cell in different ways, what does it do in different cell types? The new ability to create conditional KOs then enabled us to produce a TRAF3-conditional knockout mouse, and thus probe more deeply into roles of TRAF3 in B vs. T cells, discovering that TRAF3 functions in a highly context-dependent manner in cells of the immune system.

Q. For B-cells you have shown that TRAF3 is required for the negative regulation of homeostatic survival and also functions as a suppressor of B cell malignancies. What has your group recently discovered about TRAF3 regulation of T-cell functions?

A. In T cell-specific TRAF3-/- mice, we found a normal number of conventional T cells, but markedly compromised T cell responses to immunization or infection. To our surprise, this was associated with impaired T cell receptor (TCR) signaling, which led us to learn that TCR+CD28 ligation induces association of TRAF3 with the TCR complex. The compromised TCR signaling, whose mechanism we are now intensively pursuing, results in a profound decrease in invariant NKT cells. We also observed an increase in thymic T regulatory cells (Treg) in these mice, and recently sought to understand the mechanism for this increase. Treg precursors must receive signals through the IL-2R in order to mature. As we recently reported, upon IL-2R stimulation, TRAF3 binds to the IL-2R and recruits the tyrosine phosphatase TCPTP, resulting in the dephosphorylation of Janus-associated kinases (Jak1 and Jak3) and downregulation of STAT5 activity. We also recently reported that TRAF3 regulates IL-15R signaling to impact the homeostasis of CD8+ T memory cells. Thus, TRAF3 can impact T cell biology through regulation of multiple types of receptors.

Q. What are your ideas regarding the evolution of TRAF3 function in different immune cells? What makes it such a versatile adapter protein while at the same time, possessing a high degree of cell-type specificity?

A. TRAF3 is expressed by virtually every cell type. We speculate that cell-type specific TRAF3 functions are determined in part by distinct opportunities for protein-protein interactions involving TRAF3, specific to the unique cell type itself, by the activating signals, by the location of the activation event in the cell, etc. We would love to perform a large-scale screen for cell-type specific proteins that interact with TRAF3. Unfortunately in the current funding climate it is hard to obtain support for this kind of exploratory science, in which the results are not predictable.

Q. Do you think there is currently a sufficient knowledge base to consider thinking about ways to manipulate TRAF3 activity in specific cell types?

A. The difficulty with considering TRAF3 itself as a drug target is that TRAF3 does so many things in different cell types. Any approach would need to be targeted to a specific location and to a specific pathway, which is very difficult. We think that instead, by learning more about specific pathways of cellular function regulated by TRAF3, those pathways can be targeted for therapeutic manipulation. This is of particular interest to us in the case of B cell malignancies, in which loss-of-function mutations of the TRAF3 gene are commonly found, consistent with the chromosomal location of TRAF3 in a region prone to translocations.

Papers to read:

- Yi Z, Lin WW, Stunz LL, Bishop GA. The adaptor TRAF3 restrains the lineage determination of thymic regulatory T cells by modulating signaling via the receptor for IL-2; Nat Immunol. 2014 Sep;15(9):866-74.
- Yi Z, Stunz LL, Lin WW, Bishop GA., TRAF3 regulates homeostasis of CD8+ central memory T cells. PLoS One. Jul 10;9(7):e102120.
- Yi, Z., Stunz, LL, Bishop, GA. TRAF3 plays a key role in development and function of iNKT cells.J Exp Med 2013: 210(6): 1079-1086.
- Stunz LL, Bishop GA. Latent membrane protein 1 and the B lymphocyte-a complex relationship. Crit Rev Immunol. 2014;34(3):177-98.
- Yi Z, Lin WW, Stunz LL, Bishop GA. Roles for TNF-receptor associated factor 3 (TRAF3) in lymphocyte functions. Cytokine Growth Factor Rev. 2014 Apr;25(2):147

Rachel R. Caspi, PhD

Dr. Rachel Caspi received her doctoral training in Israel and her postdoctoral training at the National Institutes of Health (NIH) in Bethesda, MD, USA. She is a tenured Senior Investigator at NIH and serves as a Section Head of the Immuoregulation Section and Deputy Chief of the Laboratory of Immunology, National Eye Institute, NIH. She also holds an Adjunct Professorship at the University of Pennsylvannia Sch. Med.

Dr. Caspi's research centers on tolerance and autoimmunity to immunologically privileged retinal antigens in animal models of autoimmune uveitis, a potentially blinding human disease. She developed the mouse model of uveitis, now in use worldwide. Her studies have elucidated many basic mechanisms of pathogenesis and helped to devise clinically relevant immunotherapeutic approaches. She is particularly well known for her work on effector and regulatory T cells in pathogenesis of ocular autoimmunity. The work she and her group have published is broadly applicable not only to ocular immunology, but also to autoimmunity in general. She is the recipient of numerous honors and awards, most recent of which are the Friedenwald award 2010 (one of the two highest awards in the field of vision research) and the Alcon Research Institute Award 2012. She has authored and co-authored more than 200 publications.

Her interest in ocular immunology started when she was a Postdoctoral fellow. At this time she discovered in a rat model of uveitis, a potentially blinding disease, that glial Müller cells in the eye regulate activation and function of autoimmune T lymphocytes that cause retinal damage. The mechanism involved downregulation of the high affinity IL-2 receptor, required direct contact but was not antigen, strain nor species restricted. This suggested an evolutionary conserved role conferring a survival advantage, such as immune privilege. Subsequently similar phenomena were identified for other cells in the eye as well as in other tissues. These findings were published in the journal Science in 1987. Another career-changing advance was the development of a mouse model of autoimmune uveitis, which catapulted forward research on basic mechanisms of the disease and is now being used by others in the field worldwide.

During her postdoctoral and early years as an independent investigator there was much less awareness of and emphasis on the issue of mentoring than we give it today. Mentoring consisted mainly of interactions about the science being done rather than career development strategies. You had to either sink or swim. As for career and family, Rachel juggled both. She has 2 grown sons, who were born during her undergraduate and early graduate years. While the timing was a personal decision, she found that it is much easier to miss a lecture when you are not the person giving it. Clearly institutional support for women scientists with family is important, but there is only one formula for success in balancing family and career: strong family support and an equitable division of childcare and household duties between the parents.

Rachel's mantra is that success as a scientist starts with choosing a subject that is important, asking central questions and studying them using state-of-the art approaches. But this is not enough. For a woman scientist, assertiveness and tenacity can make all the difference between failure and success. Therefore, when the opportunity knocks, you should be willing to step forward, be visible and take on responsibility. She feels that you have to aim high, believe in yourself and persevere. She says, "you can really have it all, if you are willing to work for it with strength of purpose, integrity and determination.

Women and Diversity Interview: 2013 G. Jeanette Thorbecke Award Winner

Kari Ann Shirey, Ph.D.
Assistant Professor, Department of Microbiology and Immunology, University of Maryland School of Medicine

Q. Have you always been interested in scientific research?

A. Yes, I have always been interested scientific research, even from an early age. I can remember sitting in the school gymnasium as a student in the fifth grade. It was career day and we were about to hear a few people talk about their jobs in a plethora of fields. A man starts talking to us about his job at Penn State University. He first described himself as a scientist. He explained what it was that he and a group of people working with him did. I can no longer remember what it was, but it was enough to capture my interest. It was at that point that I knew I wanted to work in science. I recall asking how he got to do what he did and he explained about going to school and becoming a doctor, but not the kind of doctor most children think of, but he was still a doctor all the same. It was on that day that I knew I would go to school and get my Ph.D. I did not know exactly which field of science I would pursue as it changed throughout my primary education. It was not until I attended college that I began to narrow down what areas of the biological sciences were most appealing to me. It was my first introduction into the field of immunology as a college senior that made me

realize I wanted to continue in that field.

Q. Can you give us a brief description of your current research and what most excites you about it now?

A. My research focuses on the ability of two respiratory pathogens, Respiratory Syncytial Virus (RSV) and influenza, to modulate the host's innate immune response by altering macrophage differentiation. This macrophage "reprogramming" can either be beneficial or detrimental to either the pathogen or the host. I continue to work on dissecting the role of alternatively activated macrophages in response to these pathogens and develop new treatment strategies based on manipulation of the host response against these pathogens. Another aspect of my research looks at the contribution of viral-induced alternatively activated macrophages to enhance responsiveness to a subsequent allergen exposure.

Q. During your graduate and post-doc years did you have mentor(s) that helped guide you along the way?

A. I have been quite fortunate to have several wonderful mentors that have been and continue to be extremely helpful to me in my budding scientific career. First and foremost is Dr. Stefanie Vogel at University of Maryland, Baltimore. She took me under her wing as a post-doctoral fellow and has taught me so much. Stefanie has become both a mentor and a friend to me. She is always willing to share her experience and knowledge. I have certainly learned much more from her knowledge and experiences than I have when taking workshops on the same topics. She has helped expose me to various professional resources, opportunities, and networks as well. Additionally, she has provided both emotional and moral support and encouragement through the many ups and downs of science. Dr. Jorge Blanco (Sigmovir Biosystems, Inc.) is a wonderful collaborator and great supporter as well. He too, was a former post-doc with Dr. Vogel and has gone on to start his own company. He is always so enthusiastic with our studies and infuses that energy in all the work we do together. Dr. Blanco's never give up attitude is inspiring. Dr. Achsah Keegan (University of Maryland, Baltimore) has also been a great source for guidance and encouragement. She has helped me to effortlessly expand my research beyond innate immunology. She is always very encouraging to not give up and try again and try new things.

Q. What was (were) the biggest challenge(s) you faced in pursuing your career?

A. I think we all question ourselves at some point and ask why we chose this route for ourselves. The important thing to remember is to stay positive and not give up on what you enjoy. Early on, before graduate school, I had several people remark that I did not have what it would take to pursue a career in the sciences. These instances were not only shocking, but also hurtful because the people making these comments were some of my instructors/professors. However, I am happy to say that I had not requested them as letters of recommendation, and more importantly, I turned these experiences around into something positive. The doubt these people had in me only drove me to try harder and buckle down more to get my Ph.D.

I feel the biggest challenge I face currently is something all academic scientists are facing, funding and the lack of obtaining it. With fewer and fewer grants being funded, this is a difficult time for not only junior faculty, but also for the more experienced senior faculty. The funding climate not only affects the ability for us to do research, but it can also make it more difficult to find available academic positions. Many bigger research institutes only want to recruit PIs with funding, so new investigators/junior faculty who are struggling just as much as more senior investigators may be deterred from staying in academics due to the lack of funding and job opportunities for those without funding. I feel we may lose some really bright people in the academic arena due to this problem.

Q. Do you feel that being a woman in science came with advantages or disadvantages? What were they?

A. I do not want to say either yes or no here. I think it really comes down to who you are as a person and how much you are willing to work for what you want to achieve. I have never felt that science is a "9 to 5" job. I usually joke and say that it is a "9 to 5" job, but that it is 9 AM Monday morning to 5 AM the following Monday morning. Science doesn't stop just because it is the weekend or a holiday. My opinion is that you cannot

Thank you to our Sustaining Members!

Robert Clark

Michael Selsted

Charles Rinaldo

Lesley Doughty

Richard Kew

Jill Kramer

expect to work 40 hours a week and think all will fall into place and you will continue to move forward in your career without putting the effort in. You have to work for those goals and achievements regardless of your gender. With that in mind, yes, women may have different priorities than men when it comes to life decisions such as starting a family and how that may change their home life vs. work life. However, I have met successful women in the academic field that somehow make it work.

If I had to find one disadvantage of being a female in science, it would be that there are fewer women in senior positions in academics, government, and industry positions. This may make those positions seem less obtainable if you are a female, but the women I know in those positions would always encourage you to work hard and not be afraid to try for more. I have been fortunate to have two really wonderful female role models as mentors who have had very successful careers. They are always very inspiring, helpful, and encouraging. My advice to other women in science is to find a great female mentor and pick her brain for helpful advice.

Q. What strategies do you use to maintain balance in your life?

A. I am still working on this balancing act myself, so I may not have sound advice. I am learning to prioritize and delegate. I think this can be a difficult adjustment when as a postdoc and/or research associate, you are used to doing everything yourself. Learning to let go and delegate some of the tasks in the lab can alleviate some of the pressure. I do not know where I would be without my "girl Friday", Wendy Lai. She steps up and takes on any assignment I ask of her in the lab, and she does it with a cheery disposition. My family and friends are good at keeping me grounded. They often remind me to take time out for myself so I do not run myself into the ground.

Q. What advice would you give to female graduate students that are interested in a career as an academic scientist?

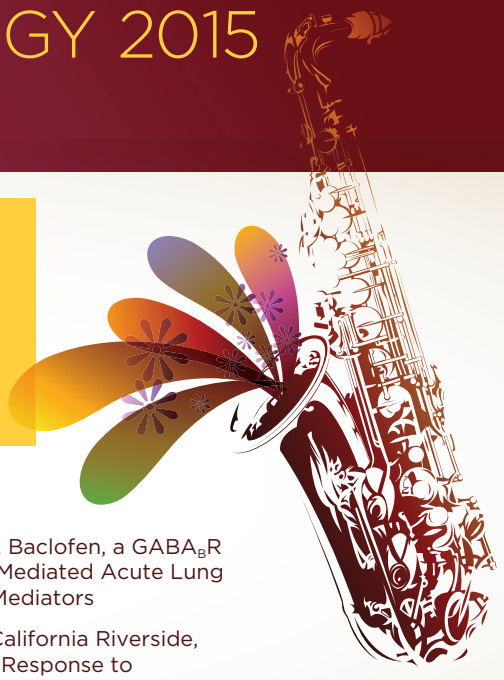
A. My advice is simple. Don't give up. There are many challenges ahead. You will face rejections many times. Laugh it off and try again. I love what I do. Science and my job are my passion. It is sometimes difficult to make people understand that you love what you do for a living when many people outside of the science world think, "I hate my job!" often. I have often commented that they should tell graduate students up front the realities of science and the academic world. Many students come in thinking I am going to get my Ph.D. and I am going to change the world. The reality is that science is hard, but if you love it and enjoy it, then it will ease some of the pain. I liken science and my job to a line graph with peaks and valleys. Most days are in the valleys when the paper got rejected, the grant was not funded, that data makes no sense and doesn't fit the hypothesis! But you live for the peaks. The paper was accepted for publication, the data was good, etc. Though the peaks may be fewer than the valleys; they make it worth it in the end. So, don't give up. Keep trying and keep moving forward.

SLB Symposium at IMMUNOLOGY 2015

New Orleans, LA ⚜ May 11, 2015

Join "Society for Leukocyte Biology: The Next Generation" at AAI 2015 where SLB will highlight some of the best up and coming junior researchers!

Co-Chairs: Louis B. Justement, University of Alabama and Silvia M. Uriarte, University of Louisville



Speakers:

Joshua J. Obar, Montana State University, Leukotriene and IL1alpha Mediated Orchestration of the Anti-Fungal Leukocyte Response to Aspergillus fumigatus

I. Coy Allen, Virginia Polytechnic Institute and State University, Evaluating NLR Modulation of Canonical and Non-Canonical NF-kB Signaling in IBD

Laura Sly, University of British Columbia, Harnessing Macrophage Phenotype for Anti-Inflammatory Therapy

Madhavi J. Rane, University of Louisville, Baclofen, a $GABA_BR$ Agonist, Ameliorates Immune Complex-Mediated Acute Lung Injury by Modulating Pro-Inflammatory Mediators

Ilhem Messaoudi Powers, University of California Riverside, Dose Dependent Modulation of Immune Response to Vaccination by Alcohol

Silvia M. Uriarte, University of Lousville, Neutrophils and Oral Pathogens: Opposing Forces in the Dysbiosis Battle

48th Annual Meeting of The Society for Leukocyte Biology

Immunity in Health and Disease

September 27-29, 2015
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- Autophagy, Unfolded Protein Response, and ER Stress in Disease Progression
- Epigenetics and Immunity
- Microbiome and Mucosal Immunity
- Neuro-Immune Interactions
- Metabolic Control of Immunity

Concurrent Sessions

- Lipid Mediator Regulation of Disease
- Immunologic Mechanisms of Vaccination
- Inflammasomes and Inflammatory Disease
- Fibrosis and Tissue Repair
- DC and Macrophages in Tumorigenesis and Inflammation
- Treg/Th17 Balance in Inflammatory Disease
- Neutrophils in Health and Disease
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