iSLB

SOCIETY FOR LEUKOCYTE BIOLOGY

Happy Holidays by Ekaterina Pylaeva

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A Message from the President


Lou Justement

As 2025 comes to a close and SLB is once again transitioning to new leadership, I want to take this opportunity to thank Jen Holland, the volunteer leadership and the members of the society for their dedication to the shared vision and mission of SLB. This past year has been extremely challenging for the biological and biomedical sciences, and for science at-large. The threats to science have never been more profound and potentially damaging than those we face today. Although that news is certainly discouraging, I am confident that the society and its members will face these challenges and will find ways to ensure that the amazing track record of research achievements that we have achieved in the US over the past 80 years, since Vannevar Bush wrote "*The Endless Frontier",* continues. *The Endless Frontier* was a visionary document, in which Vannevar Bush clearly articulated the point that "*It has been basic United States policy that Government should foster the opening of new frontiers. Moreover, since health, well-being, and security are proper concerns of Government, scientific progress is, and must be, of vital interest to Government. Without scientific progress the national health would deteriorate; without scientific progress we could not hope for improvement in our standard of living or for an increased number of jobs for our citizens; and without scientific progress we could not have maintained our liberties against tyranny."* When I read this passage and reflect on the fact that the scientific community has benefitted from bipartisan support to foster the leading research enterprise in the world for over 80 years, it renews my commitment to do everything I can to ensure that this reality continues into the future. Because if it does not, the damage to the scientific enterprise, to the health and well-being of Americans, to the US economy and to our ability as a country to fight against tyranny in all its forms, will be immeasurable.

So, what can you do to stand up for science? Each of us can play a significant role in this endeavor by doing relatively simple things. First, each of us needs to continue to support SLB by renewing our membership and by recruiting our peers to join the society. Members are the lifeblood of SLB. SLB in turn is actively engaged through its affiliation with other professional scientific organizations and advocacy groups to lobby for continued support for research and to promote sound policies that regulate the scientific endeavor. Moreover, you, the members of the society, are the ones who create such a wonderful, inclusive community that provides valuable support to one another in these trying times. Second, volunteer to become active on an SLB committee, or the Council. SLB cannot sustain itself as an organization and promote the critical changes we need to support the scientific endeavor unless members step up and volunteer. Third, submit your research to the Journal of Leukocyte Biology. Revenue from JLB provides the bulk of financial support to ensure the ability of SLB to operate day in and day out and to provide valuable member benefits. Supporting JLB is one of the most important ways that you can contribute to the society. Finally, embrace your role as a champion for science by communicating the benefits of biomedical research to friends and family, to your community and to state and federal legislators. I realize that I have called on each of you to do these things before.

However, as my term as President of SLB comes to an end, I am asking you once again to stand up for science by supporting this tremendous organization and by being an advocate for biomedical research.

I have sincerely enjoyed the opportunity to serve as President of SLB and I value the numerous friendships I have made over the years as a member of the society, while serving on committees and the council and most recently as President. I am also incredibly thankful for our Executive Director, Jen Holland, who is the literal lynch pin for the organization. Jen works incredibly hard to ensure that everything runs smoothly. I also want to welcome Cindy Leifer as our next President. Cindy is without question the right choice to lead SLB over the next two challenging years. It has been a real pleasure to interact with Cindy on the Presidents' calls over the past two years and I am consistently impressed by her vision and sound decision-making. I have absolute confidence in her outstanding leadership to take us through what is likely to be a challenging couple of years. The good news is that our President-Elect is Anne Pereira, and she has a long track record as a volunteer leader with SLB that will enable her take up the reins after Cindy and ensure that the society continues to thrive well into the future. Luckily for me, I get to stick around for the next two years as Past-President, and in that role, I look forward to continuing to work with Cindy and Anne and the volunteer leadership to position the society for long-term success. In closing, I extend my best wishes for 2026 to the new leadership of SLB and to the outstanding members of the society. Together, we can make a significant difference in ongoing effort to advance the biomedical sciences.

Host A 2026 Special Interest Group Satellite!

SLB is pleased to provide a platform for society members to organize their own 2026 session. These Special Interest Group Satellites (SIGs) will be held on Monday, September 14th, 2026 in association with the annual meeting. Proposals will be reviewed by the conference program chairs to ensure there is no conflict in topics and/or speakers. Learn more and submit your proposal by January 16th, 2026.

Member Highlight

Rosemary Bayless: *Bridging Veterinary Medicine and Translational Immunology with Equine Neutrophils*

by Kaitlyn Whitefoot-Keliin

Dr. Rosemary Bayless is carving out a unique space in leukocyte biology by integrating her veterinary training with translational immunology, focusing on neutrophils from horses, other animals, and humans. Her work bridges species, organ systems, and disciplines, exploring how naturally occurring veterinary diseases can advance understanding of neutrophil function, heterogeneity, and therapeutic targeting relevant to both animal and human health. As a member of the Society for Leukocyte Biology (SLB), Dr. Bayless offers a valuable comparative perspective on the relevance of spontaneous large animal models in developing clinically meaningful insights and interventions.

Rosemary at SLB 2023

Dr. Bayless began her scientific journey by earning her DVM from Kansas State University, completing a rigorous equine internship and equine internal medicine residency, and becoming board-certified in large animal internal medicine before pursuing a PhD at North Carolina State University. During her doctoral studies, she discovered a passion for neutrophil biology and attended her first SLB event, presenting at SLB's 2021 virtual meeting. Her first in-person SLB meeting in 2023 left a strong impression due to its welcoming culture and genuine camaraderie among members. She highlights SLB's robust networking opportunities, including the *Building Bridges in Leukocyte Biology* webinar series, and looks forward to participating in future SLB meetings and programs. For Dr. Bayless, SLB is more than a professional society, it's a community where members become friends and colleagues.

As a reviewer for the Journal of Leukocyte Biology (JLB), Dr. Bayless values the journal's rigorous peer review process. In an era of concern about predatory publishing, she appreciates JLB's commitment to quality and transparency, noting that her time and expertise are respected. These principles make her more inclined to publish in professional society-affiliated journals going forward, and she looks forward to joining SLB's *Reviewer Training Program.*

Now a faculty member at North Carolina State University and member of the Translational Predictive Biology Cluster, Dr. Bayless established her lab in 2023. She is developing a research program focused on neutrophil pathobiology, including investigating the mechanisms through which drug candidates inhibit neutrophilic inflammation, determining how neutrophil subpopulations drive disease or promote disease resolution, and developing in vitro models of neutrophil-mediated disease processes, including sepsis, that better recapitulate the immune responses that occur in patients. Her work provides insights into neutrophil function and inflammation relevant to both animal and human health. Mentorship remains a core priority for Dr. Bayless. She takes pride in her research team, including curious, motivated trainees and an experienced laboratory manager, describing the laboratory's supportive and collaborative environment as her greatest professional accomplishment to date. Her biggest advice to trainees is to form circles of science friends, across different stages of their careers — a network for collaborating, celebrating successes, and unfortunately sometimes, commiserating.

Outside the lab, Dr. Bayless balances her scientific pursuits with activities that bring her joy and mental refreshment, such as biking, solving crosswords, and spending time with her cat. She is also deeply committed to social justice, actively working to ensure her lab remains a supportive and inclusive space. With a deep love for science (especially neutrophils!), a drive to bridge disciplines, and a commitment to building supportive communities, Dr. Bayless embodies the values and spirit of SLB.

Coming FREE FOR ALL Webinars

- December 17th, 2025, 12pm eastern - Margarida Santos Saraiv, *i3S- Instituto de Investigação e Inovação em Saúde* **"Understanding tuberculosis pathogenesis and immune responses through the lens of *Mycobacterium tuberculosis*"** LIVE ONLY event.
- January 28th, 2026, 9am eastern - Stefan Oehlers, *A*STAR ID Labs, Singapore* **"Differential subversion of host immune signalling by tuberculous and non-tuberculous mycobacteria".**
- **And 6 more confirmed speakers for 2026!**

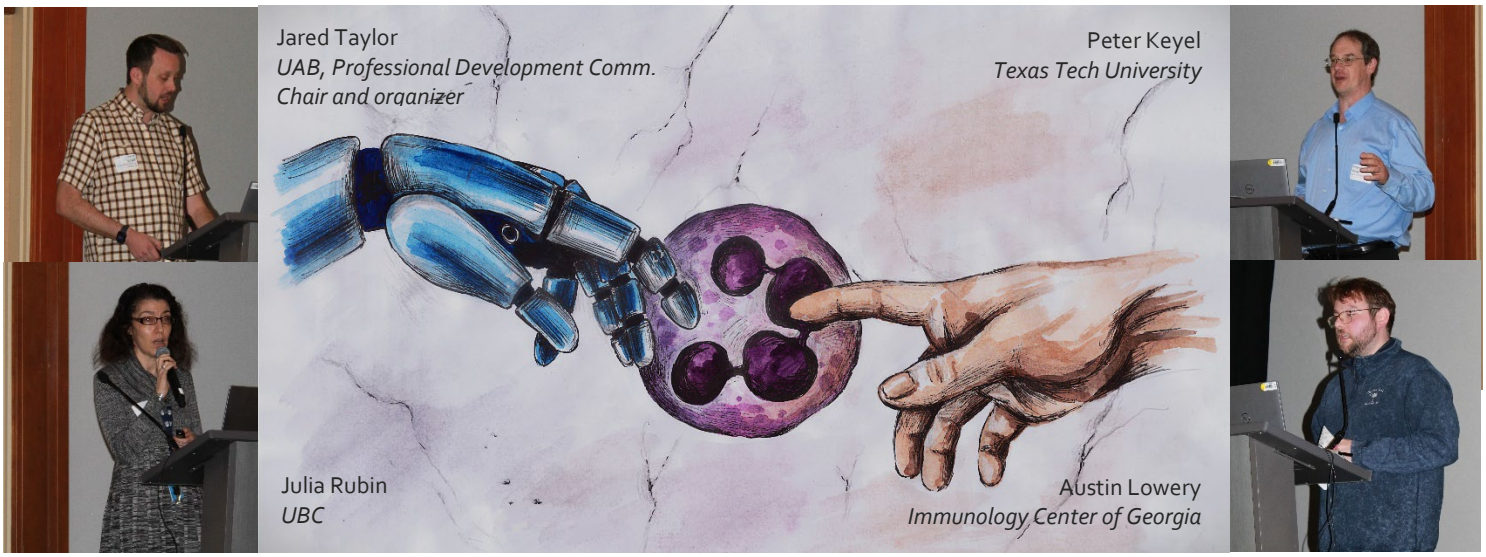
Learn more about this webinar series and register

AI Literacy: A leap into the unknown

By Austin Lowery

In recent years, AI technology has been sprouting at extraordinary rates across the globe, causing shifts in industries and making major waves across our devices, households, and workplaces. Because of this expansion, AI has brought about lingering questions as to what we may expect from its development: How will AI change the rate of scientific discovery? Will AI replace you at your place of work? Can you tell if AI used to generate the very words you're reading now?

SLB's efforts to remain at pace with cutting edge science and technological advances gave rise to this year's professional development workshop topic: AI Literacy. This year's panel consisted of talks spanning fundamental basics of AI and machine-learning models presented by Jared Taylor, PhD (UAB), the uses of AI in education and training from Julia Rubin, PhD (UBC), AI utilization in scientific journals and writing presented by Peter Keyel, PhD (TTU), as well as future considerations of AI usage from PhD student Austin Lowery (IMMCG). Amid this year's insightful presentations, inquiries from the audience further expanded the discussion to topics such as AI usage for overcoming competition during the hiring process and enhancing day-to-day skills in the workplace, such as generating lesson plans and adjusting tonality in emails based on native language and gender. While the future development of AI and its incorporation into the research world are still unclear, JLB will continue to monitor its growth with a watchful eye. Thank you to all of the SLB members that attended!



Behind the Science with JLB Authors

compiled by Ramizah Mohd Sabri

Elaheh Abdolahinia, Nova Southeastern University

Human CD24⁺CD38⁺ regulatory B cells promote pro-resolving macrophage function through the synergistic effect of IL-10 and PD-L1

Q: How would you summarize your key findings?

A: Our research shows that a specific population of B cells, CD24⁺CD38⁺ B cells, can help 'calm down' inflammation. These B cells improve how macrophages clear dying cells and help the body produce natural molecules that resolve inflammation. This gives us new insight into future anti-inflammatory therapies.

Q: What initially sparked the idea for this study?

A: The idea is from our interest in translating promising findings from mouse models into human biology. Studying human B cells can be challenging, so we focused on CD24+CD38+ B cells known to produce high levels of IL-10. This allowed us to test whether the regulatory effects observed in mice also occur in humans. Our results confirmed that this B-cell subset has strong therapeutic potential, which motivated the entire direction of the study.

Q: Was there a moment when the data surprised you?

A: When we initially isolated lymphocytes from blood and stimulated them to increase IL-10 secretion, we were surprised to see that most of cells died within just two days. This unexpected loss made it clear that isolated lymphocytes were too sensitive under those conditions. As a result, we shifted our approach and began stimulating whole PBMCs instead, which proved much more effective for generating viable IL-10–producing cells.

Q: What methods or approaches were especially important in allowing you to answer this question?

A: Using a combination of co-culture models, flow cytometry, qPCR, and functional assays was essential. This allowed us to see the molecules being expressed and how cells behave and respond under different conditions. Integrating several experimental approaches helped us get a more complete picture.

Q: What was the biggest challenge you faced during this study, and how did you overcome it?

A: One of the biggest challenges we faced was working with human primary cells, particularly when trying to isolate both CD24⁺CD38⁺ regulatory B cells and monocyte-derived macrophages from the same blood samples. Human B cells are highly sensitive to purification and stimulation, and we consistently observed significant cell loss when we attempted to expand Bregs after isolated B-cell purification. At the same time, isolating enough viable primary monocytes for macrophage differentiation—while staying within IRB limits on blood volume—was extremely difficult. These limitations made it impossible to obtain both cell types in sufficient numbers from a single donor for matched experiments. To overcome this, we optimized our workflow by first stimulating whole PBMCs to enhance Breg generation before isolation, and we used THP-1–derived macrophages as a standardized, reproducible human macrophage model. This approach allowed us to maintain non-polarized starting macrophages, avoid donor-to-donor variability, and reliably perform mechanistic assays such as efferocytosis and lipid mediator profiling. Although primary human macrophages remain ideal, this strategy enabled us to answer key mechanistic questions with rigor and reproducibility. These obstacles ultimately strengthened our study design and highlighted the need for improved technologies that allow simultaneous isolation of multiple immune cell subsets at high purity and viability—an important direction for future human in vivo studies.

Q: What do you enjoy most about doing research?

A: I enjoy the process of discovery—those moments when data begin to form a clear story. Even small findings feel meaningful when they reveal something about how biology works. Research also brings constant learning, which I find incredibly motivating.

Q: How do you approach things when you hit a roadblock?

A: I step back try to look at the problem from a different angle. Sometimes that means simplifying the question, consulting colleagues, or revisiting the fundamentals. I also find that reading outside my immediate field often sparks new ideas. Creativity in science comes from being open to rethinking assumptions.

Q: What about working in science brings you satisfaction?

A: Seeing a complex project come together, when experiments, data, and interpretations align, is satisfying. I value the collaborative nature of science. Sharing ideas, learning from others, and contributing something meaningful to the field is very rewarding.

Q: What excites you most about where the field is heading?

A: I am excited about the growing possibility of translating cell-to-cell interaction studies into actual therapeutic strategies. Our in vitro findings on how CD24⁺CD38⁺ B cells modulate macrophage function suggest that these regulatory B cells or the pathways they activate could eventually be harnessed for cell-based therapies. As the field advances in precision immunology and cell engineering, I believe this type of B-cell–macrophage crosstalk has real potential to shape future anti-inflammatory and pro-resolving interventions.

Q: What advice would you give to those at the beginning of their scientific journey?

A: Stay curious and be patient with yourself. Science is full of challenges, but persistence and an open mind make all the difference. Don't hesitate to ask questions, seek guidance, and take risks with new ideas. Every experiment—successful or not—moves you forward.

Tanya Freedman & Anders Lindstedt, University of Minnesota

Lyn expression in macrophages promotes TLR activation and restricts proliferation in an isoform-independent manner

Q: How would you summarize your key findings in plain language?

AJL & TSF: We report that the proteins LynA and LynB function similarly in macrophages, a type of immune cell responsible for fighting infection, regulating inflammation, and maintaining tissue health. LynA and LynB are the two splice variants of the Src-family kinase Lyn, which add phosphate groups to proteins inside cells to either drive or inhibit protein-protein interactions and intracellular signaling pathways. In macrophages, Lyn promotes intracellular signaling and inflammatory protein production downstream of Toll-like receptors, which respond to bacterial and viral proteins and nucleic acids. We found that macrophages lacking both isoforms of Lyn, which we call "complete Lyn knockouts," have impaired activation of downstream signaling pathways and pro-inflammatory protein production when treated with activators of Toll-like receptors. We also found that Lyn plays an important role in regulating extracellular matrix (ECM), which provides structural support for tissues and contributes to scar formation. Lyn knockout cells up-regulate ECM-forming genes and down-regulate ECM-degrading genes, which may contribute to fibrosis and regulate cancer growth and metastasis. Macrophages normally have both isoforms of Lyn, but we used cells lacking one or the other Lyn isoform to test the individual contributions of LynA and LynB to macrophage signaling. If cells express only LynA or LynB, they proliferate more in response to growth signals, similarly to complete Lyn knockout macrophages. In contrast, macrophages expressing either isoform alone have normal TLR activation responses. Overall, we found that Lyn promotes Toll-like receptor signaling and restricts macrophage proliferation and matrix deposition in an isoform-independent manner.

Q: What initially sparked the idea for this study?

AJL: This idea was sparked by the paucity of studies investigating isoform-specific functions of Lyn and differing findings of Lyn function in Toll-like receptor signaling pathways. Some work has suggested that LynA and LynB may contribute different functions in distinct cell types and signaling pathways.

Q: Was there a moment when the data surprised you?

AJL: We were surprised that LynA and LynB provide similar functions in macrophages and Toll-like receptor signaling pathways. Our studies in aged LynA- and LynB-knockout mice suggested that LynB is the dominant inhibitory isoform of Lyn, particularly in male mice. While female LynA-knockout mice developed severe splenomegaly and lupus-like manifestations, male LynA-knockout mice did not, suggesting that LynB alone was sufficient to restrict lupus development in male mice. Since Toll-like receptor signaling contributes to lupus pathogenesis, we thought we might see different contributions of LynA and LynB in Toll-like receptor signaling pathways. Furthermore, we did not see any sex-specific differences between LynA and LynB in macrophages—again, not what we expected, given our observations in aging mice.

TSF: We were surprised that either LynA or LynB expression was sufficient to restore normal gene expression and function in macrophages; this was anticipated to be the major advance. Nevertheless, the complementarity of the two Lyn isoforms was a novel finding, and we took the opportunity to double down on the broad functions of Lyn (A + B) in signaling through Toll-like receptors. Anders and I worked together to realign our expectations and construct a new narrative around the data we had. This is so common in science, and I think that it's a credit to Anders's integrity and flexibility that we were able to make a success out of this research project.

Q: What methods or approaches were especially important in allowing you to answer this question?

AJL: Developing isoform-specific knockout mice/cells and performing RNA sequencing on macrophages were important. The lack of LynA- and LynB-specific knockout mice had previously prevented us from testing isoform-specific functions, and developing these mice allowed us to investigate these proteins in isolation, without relying on overexpression systems. RNA sequencing provided the resolution to detect subtle differences in signaling and offered an unbiased picture of cellular functions regulated by Lyn.

Q: What was the biggest challenge you faced during this study, and how did you overcome it?

AJL: The biggest challenge we faced during this study was interpreting the bulk RNA sequencing dataset. RNA sequencing provides an overwhelming amount of data, and it's important to filter out the meaningful differences and be able to present them in a coherent way. We collaborated with other scientists in our department that had more experience with bioinformatics for advice on experimental design, data processing, and presentation. Computational methods like Gene Set Enrichment Analysis are also wonderful tools that help organize the data to identify relevant differences in gene expression.

Q: What do you enjoy most about doing research?

AJL: One of the most enjoyable aspects of research is finding something unexpected. In our study, we initially focused on Toll-like receptor signaling and inflammatory cytokine production, so it was exciting to find meaningful differences in extracellular matrix-related genes, like collagens and matrix metalloproteases. This might form the basis of future projects investigating extracellular matrix regulation and fibrosis, and it is always exciting to take the project in a new direction. The parts of science that bring me the most satisfaction are seeing an idea come to fruition or learning a new technique. Learning the ins and outs of RNA sequencing was incredibly satisfying. It is great to have a better understanding of how to design a quality RNA sequencing experiment but even more satisfying to be able to understand the coding behind the analysis

and data presentation. It is also gratifying to be able to apply new knowledge, like coding skills, to other aspects of life.

TSF: It's the thrill of solving a mystery, testing new frontiers of knowledge, and the experience of working with trainees and collaborators to interpret and communicate our findings. My work is different every day, and I love learning as we explore new avenues of inquiry.

Q: How do you approach creativity or problem-solving when you hit a scientific roadblock?

AJL: I think the best way to approach problem-solving is to constantly discuss your experiments with your lab and other scientists. Sometimes we can get too focused on a particular experimental avenue, and it can be helpful to bring in a fresh perspective.

TSF: I agree with Anders. While movies often depict scientists as social misfits, social skills are part of our broad toolkit. Talking to a colleague or trainee can help us escape a local minimum and spark new ideas.

Q: What advice would you give to those that are at the beginning of their scientific journey?

AJL: The two pieces of advice I have for those at the beginning of their scientific journey is to be flexible and ask for help early and often. It is virtually impossible to predict all the findings from a particular study or set of experiments. Don't fixate on one hypothesis and embrace unexpected results. This can often lead to a more fruitful project than initially anticipated, and this flexible thinking will help prevent you from getting stuck on a faulty train of thought. Similarly, it is important to ask for help from someone more experienced in a technique or knowledgeable about a topic. This will help you avoid common pitfalls and will help streamline your experiments to make the most efficient use of your time and resources. Good luck!

TSF: Don't be afraid to take risks. Change fields at career transitions. Ask questions if you don't understand, even in group settings. Volunteer for experiences and test your competence and enthusiasm for activities relying on different types of skills. Form deep friendships and collegial relationships with your peers and colleagues and keep in touch with mentors. Whenever possible, aim for synergy over competition. Lean into your curiosity, integrity, and optimism, even (especially) when times are stressful. Remember your long game.

Ruth Baigorri and Fabio Cerban, Universidad Nacional de Cordoba

Metformin improves CD8⁺ T cell responses and parasitemia control via macrophage modulation during *Trypanosoma cruzi* infection.

Chagas disease remains one of the world's most neglected tropical diseases, affecting millions across Latin America and beyond. In a recent study, researchers explored how metformin (a drug widely prescribed for diabetes) might influence the immune response during Trypanosoma cruzi infection. In this interview, the first author and the lead investigator share the motivations behind the work, the challenges faced, and the broader implications for the field.

Q: How would you summarize your key findings?

A: We explored how metformin, a drug widely used for diabetes, influences the immune response during *Trypanosoma cruzi* infection. Our findings show that metformin helps regulate the inflammatory activity of macrophages. This moderation not only benefits CD8 T cells but also plays an important role in controlling parasitemia.

Q: What initially sparked the idea for this study?

A: Our lab has studied the macrophage inflammatory response to *T. cruzi* for decades, often through in vitro experiments. In this project, we wanted to move beyond isolated systems and examine how macrophages behave in their natural environment, in vivo, and in relation to CD8 T cells. Using metformin (a drug already in common use) allowed us to explore immune modulation in a clinically relevant context. We also aimed to understand how infection impacts target tissues.

Q: Was there a moment during the project when the data surprised you?

A: Yes. A key turning point was realizing that, even though parasite replication was controlled, animal survival did not improve. We had expected that adjusting the macrophage response would strike the right balance, limiting the parasite without harming tissues or suppressing T cells. That unexpected outcome raised new questions and pushed us to rethink our assumptions.

Q: What methods or approaches were especially important in allowing you to answer this question?

A: It was essential to recognize that immune cells don't act in isolation, they're part of a complex system constantly seeking equilibrium and *T. cruzi* is a parasite highly adapted to long-term survival. To capture this complexity, we performed adoptive transfer experiments of immune cells in the infection model, combined with metformin treatment in vivo. This approach helped us see how different components of the immune system interact under real conditions.

Q: What was the biggest challenge you faced during this study, and how did you overcome it?

A: Funding was the biggest challenge. We had ambitious ideas and wanted to use advanced techniques, but our budget couldn't support them. We overcame this by finding alternative models and

approaches, and by relying on enthusiasm and teamwork. In the end, this experience strengthened our ability to design experiments critically and creatively, helping us grow as well-rounded scientists.

Q: What do you enjoy most about doing research?
A: The most rewarding moments come after long hours of study, planning, and lab work, when results support our hypotheses. But those results also open up new questions, which is the true essence of research. For a curious mind working with a strong team, that cycle of discovery is incredibly fulfilling.

Q: How do you approach creativity or problem solving when you hit a scientific roadblock?
A: There's no secret formula, it's persistence. Careful reading of the literature, long hours of thought, and teamwork, where each member contributes their expertise, are what help us move forward.

Q: What part of working in science brings you the most satisfaction?
A: Knowing that we are working together to understand how the world functions, and how that knowledge can improve human health and life, is deeply satisfying.

Q: Looking ahead, what excites you most about where the field is heading in the next few years?
A: I'm excited about the growing integration of patient data with animal models. Big data and bioinformatics have enormous potential to advance treatments, even for neglected diseases like Chagas disease. I look forward to seeing how these tools will shape the future.

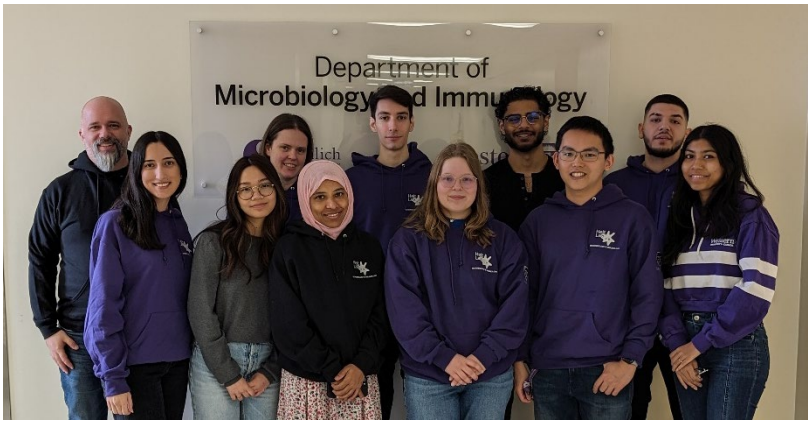
Q: What advice would you give to those at the beginning of their scientific journey?
A: Stay open to learning, be tenacious, and commit fully to your goals. Each challenge will bring its own lessons, and together they will shape you into a stronger, better-trained scientist.

This study highlights both the promise and the complexity of using existing drugs to modulate immune responses in parasitic infections. While metformin showed intriguing effects on macrophages and CD8 T cells, the unexpected outcomes underscore the importance of viewing the immune system as part of a larger, dynamic host–parasite relationship. As the field moves toward integrating patient data, animal models, and bioinformatics, the insights gained here will help pave the way for more effective treatments for Chagas disease and other neglected conditions.

Bryan Heit, The University of Western Ontario

GATA2 induces a stem cell-like transcriptional program in macrophages that promotes an atherogenic phenotype

Q: How would you summarize your key findings?
A: Macrophages are an immune cell which under some conditions can cause atherosclerosis – the accumulation of cholesterol and fats beneath the blood vessels of our hearts. Normally macrophages prevent atherosclerosis by removing lipid deposits and dying cells, but in half of people this activity is lost, leading to disease. Several years ago, we identified GATA2 – a gene which regulates the expression (production) of other genes – as a gene associated with disease. In this study we identify the disease-inducing stimuli that cause GATA2 to be expressed by macrophages and determine how GATA2 then promotes the formation of disease. This promotion of disease is a result of GATA2 inducing an immature phenotype in the macrophages that causes a loss of their cholesterol-processing and dead cell-removal activities.



Q: What initially sparked the idea for this study?
A: Several years ago, we performed a transcriptomics analysis of macrophages from early-stage human atheroma, looking for gene expression changes that may explain why these cells transition from a protective to disease-causing phenotype. GATA2 stood out in that analysis, as it is associated with myelopoiesis, with a complete suppression of GATA2 expression required for the terminal differentiation of mature myeloid cells. This study answers two key questions that arose from this first study – what induces GATA2 expression in atheroma macrophages, and what is the impact of GATA2 expression on these cells.

Q: Was there a moment during the project when the data surprised you?
A: While we expected to see some hematopoietic genes re-expressed when GATA2 was present, we were surprised at the extent to which GATA2 induced a stem-cell like expression profile.

Q: What methods or approaches were especially important in allowing you to answer this question?
A: RNAseq and ChIPseq were critical for identifying the stem cell-like phenotype of these cells and allowed us to identify key GATA2-regulated genes that generate the pro-atherogenic phenotype in these macrophages.

Q: What was the biggest challenge you faced during this study, and how did you overcome it?
A: The biggest challenge was correlating ChIPseq and RNAseq data to promoter activity data, as there was not always a 1:1 correlation between binding of GATA2 to a promoter, promoter activity, and mRNA abundance.

Q: What do you enjoy most about doing research?

A: Seeing smaller pieces of a project come together in a way which lets you see the bigger picture of how the biological system functions.

Q: How do you approach creativity or problem-solving when you hit a scientific roadblock?

A: First, read the literature. Second, consult with experts. Third, throw things at the wall and see if anything sticks.

Q: What part of working in science brings you the most satisfaction?

A: Unexpected results that challenge our models and hypotheses. Our most interesting papers arise from unexpected results.

Q: Looking ahead, what excites you most about where the field is heading in the next few years?

A: We've known for a long time that patient-specific factors such as age, sex, genetic background, and the formation of de novo mutations (such as those which cause clonal hematopoiesis) are major factors in a person's risk of developing atherosclerosis and in the rate that disease progresses. Newer sequencing and single-cell approaches may finally let us link these patient-level phenomenon to specific cellular and immunological pathways that then drive disease. This opens the door to a better understanding of atherosclerosis, as well as to the possibility of individualized therapies which address unique risk factors and disease pathways in individual patients.

Q: What advice would you give to those that are at the beginning of their scientific journey?

A: Embrace the unexpected – sometimes a seemingly "failed" experiment or unusual result is a window into the underlying biology.

SLB 2025 highlights

SLB 2025 was a great event, and we welcome you to view the program, abstracts and highlights from the event in the archives. Learn about SLB 2026 and join us next year!

Annual Survey and Volunteer Opportunities

SLB is YOUR community and we welcome your feedback via the end of year survey. Look for the invitation and reminders in your email and participate by January 5th. Be sure to enter the prize drawing! The survey also offers the opportunity to volunteer on one of SLB's amazing committees. Consider participating in these valuable activities in 2026 and beyond. Contact us with questions!

MSI winners

JLB is pleased to participate in a number of events around the globe annually. This year at the Mexican Society for Immunology meeting, several trainees received the SLB Trainee Award Certificate.

Gonzalo González Domínguez, Adrian Albarrán-Godínez, Estefania Aleman Navarro , and Saúl Arteaga Cruz with JLB EiC, Michael Schnoor

Macau winners

JLB was pleased to participate in an event in Macau with a resulting targeted science issue and 10 SLB Trainee Awardees. 2025 Lifetime Awardee, Xin Chen and JLB Editorial Board member Domenico Mavilio were present with the awardees.

Eric Alves, PhD

JLB Social Media Editor – X/Bluesky

Q: What is your background and where you are from?

A: I was born in Sydney and raised in Perth, Western Australia – often called the most isolated city in the world! I've remained in Perth for my undergraduate studies, Honours and PhD, all completed at the University of Western Australia. Currently, I'm a Postdoc at Curtin Medical Research Institute and the Harry Perkins Institute of Medical Research (also in Perth, Western Australia), and have continued at the University of Western Australia as a Sessional Teaching Academic.

Q: Please share you career path, from undergraduate studies to now?

A: After completing a Bachelor of Science at the University of Western Australia, I undertook an Honours year that introduced me to full-time research. My project focused on how HIV adapts to the CD8+ T cell response, an experience that led directly into my PhD. During my PhD, I investigated how HIV adapts to the CD4+ T cell response and expanded to explore how genetic variation at loci encoding the human leukocyte antigen (HLA), killer cell immunoglobulin-like receptor (KIR), and other related molecules influences clinical outcomes in HIV and other conditions. I spent part of my PhD at Vanderbilt University Medical Center, which was especially enriching! Upon returning to Western Australia, I've started as a Postdoc at Curtin Medical Research Institute and the Harry Perkins Institute of Medical Research.

Q: What is the focus of your current research?

A: Recently, my work has focused on the interplay between HLA variation, viral immune evasion, and T cell function, and improving approaches for profiling complex genomic regions (e.g. KIR) in multi-omic data. While multi-omic assays are a great way to study cell-cell interactions and uncover new cell states or subsets, validating results is critical, especially in the context of complex genomic regions, and doing this has revealed significant technical and bioinformatic challenges! Therefore, another major current focus is in improving how we examine complex genomic regions using these multi-omic assays and developing approaches that account for genomic complexity, while also identifying and reporting technical pitfalls that could obscure important biological insights.

Q: How do you think social media (e.g. X/Bluesky) can help support or shape scientific research, communication and publication?

A: Social media, and particularly X/Bluesky, is increasingly becoming a key source of information (or disinformation) on a global scale. I believe that X/Bluesky should be embraced as a tool for scientific communication and used to promote validated and peer-reviewed findings, support networking and foster collaboration, highlight new technical developments, and promote our achievements to both the public and within our scientific community. Unlike other social media platforms, X/Bluesky are dynamic forums, which I believe provides an exciting space where scientists can demystify their work and engage with fellow scientists, as well as the public, to make their science more accessible.

Q: What are you hoping to achieve or explore in your new role as JLB Social Media Editor?

A: JLB's X account, which was launched and grown by Irving Coy Allen (Virginia Tech) to >2,500 followers (I certainly have big shoes to fill!), has become an excellent platform for promoting the journal and its articles. I'm excited to continue expanding the journal's reach in 2026 and beyond. In addition to the X account, one of my first goals is to launch a JLB Bluesky account, so keep an eye out in early 2026!

Xiang Lin, PhD

JLB Social Media Editor – WeChat

Q: What is your background and where you are from?

A: My name is Xiang Lin. I am from Hong Kong, China. I'm currently serving as an Assistant Director (Knowledge Exchange & Technology Development), and an Assistant Professor in the School of Chinese Medicine, The University of Hong Kong.

Q: Please share you career path, from undergraduate studies to now?

A: My background is in Chemistry and Chinese Medicine. At present, there are many unsolved questions in understanding Chinese Medicine, beyond Chemistry. From clinical practice and literature, Chinese Medicine seems to elicit beneficial effects in patients with chronic inflammation, providing many potential alternative approaches. This is why I dive into the field of immunology, specifically autoimmune disease. I'm currently working on understanding the pathogenesis of and uncovering new therapeutic approaches to Sjogren's disease, a common but under investigated autoimmune disorder.

Q: What is the focus of your current research?

A: My research mainly focuses on T-B cell interactions and their responses in autoimmune pathogenesis. I'm also working on drug development to identify novel therapeutics from the components of medicinal herbs.

Q: How do you think social media (e.g. WeChat) can help support or shape scientific research, communication and publication?

A: Social media helps the exposure of novel findings in the first place, and highlights new findings with visual aids (figures, editorials, comments, etc.). It can help to feature certain important findings and help editors pick high quality work. For example, there are many journal papers presenting their novel findings after official publication in WeChat. Doing so helps to create a lasting impression of the work, as well as the journal, to a broader audience. It also allows for the latest findings to be rapidly disseminated.

Q: What are you hoping to achieve or explore in your new role as JLB Social Media Editor?

A: I hope the content from JLB, disseminated through social media, can provide the broader community with timely knowledge and a lasting positive impression, thus elevating JLB as a priority when submitting their high quality and impactful work.

FASEB CORNER

Expanding Our Advocacy for Research – On September 18, representatives from FASEB's volunteer leadership and member societies joined nearly 500 patient advocates, scientists, and researchers from 37 states for the 13th Rally for Medical Research Capitol Hill Day. FASEB also sponsored travel awards to cover costs for members of FASEB societies from targeted states to travel to Washington, DC to participate in meetings with their Senators and Representatives and their aides.

On October 1, FASEB issued a statement and an e-action alert urging Congress and the administration to immediately re-open the government. The FASEB alert generated nearly 1,000 email messages to congressional offices and the White House. As federal agencies re-opened following the historic government shutdown, FASEB sent a letter urging Congress to finalize the fiscal year 2026 budget before the current continuing resolution expires on January 30, 2026. FASEB's letter expressed concern that spending bills funding research at the National Institutes of Health, National Science Foundation, and the Department of Energy Office of Science still need to be negotiated and adopted.

FASEB Launches Virtual Advocacy Town Hall Meetings – In August, FASEB began a new series of virtual Advocacy Town Hall meeting series open to anyone interested in supporting FASEB's advocacy efforts. The town hall meetings take place on the first and third Tuesdays of each month at noon eastern. Each meeting features updates from Capitol Hill, the latest news on science policy developments from federal agencies, time for questions and answers, and an open mic segment. A one-time registration process allows participation in the entire series. Registrants receive a follow-up email message that summarizes the meeting and includes curated FASEB advocacy resources. The final 2025 town hall is on Tuesday, December 16 at noon eastern. Town halls will continue in 2026 beginning on Tuesday, January 6.

FASEB Continues Engaging in Legal Advocacy – FASEB joined three other scientific societies – ASBMB, American Society for Cell Biology (ASCB), and American Society for Microbiology (ASM) – in filing an expanded amicus brief with the U.S. Supreme Court in *American Public Health Association v. National Institutes of Health*. The brief urged the court to deny the government's request for a stay pending approval, emphasizing the immediate and irreparable harm caused by the termination of grants supporting early career scientists.

On August 21, the Supreme Court issued a fractured ruling that permitted the Administration, for now, to cancel nearly $800 million in NIH grants due to a jurisdictional issue with the original filing. However, a different majority of justices agreed the Administration's policy underlying these terminations was likely unlawful. In response, FASEB, ASM, ASBMB, and ASCB issued a statement noting the four organizations' deep disappointment with the Supreme Court's decision. Since then, the First Circuit established a briefing schedule on the merits of the government's appeal and the government's opening brief was due in mid-September. FASEB, ASBMB, ASM, and ASCB remain committed to vigorously defending the integrity of federal research funding and will continue to fight to protect the scientists it supports. To read more about FASEB's efforts in support of the plaintiffs in APHA vs NIH click here and here.

FASEB also recently signed-on to an Amicus Brief organized by the American Physical Society on administration policies affecting international scholars, including the abrupt termination of student visas and deletion of students' records in the Student and Exchange Visitor Information System (SEVIS).

Science Policy Committee Comments on Key Regulations and Policy Issues – The Science Policy Committee (SPC) develops position statements on policy issues of interest to the Federation. Between July and November, the SPC:

- Provided feedback to the Joint Associations Group on Indirect Costs (JAG) on the Financial Accountability in Research (FAIR) model, the group's proposed new approach for determining facilities and administration costs (also known as F&A or indirect costs). The FAIR model was developed as an alternative to the administration's proposal to apply a flat 15 percent F&A rate to research grants issued by the National Institutes of Health, National Science Foundation, Department of Energy, and Department of Defense.
- Issued a statement expressing concerns about the Executive Order (E.O.), "Improving Oversight of Federal Grantmaking," and calling upon the research community to take action to help inform their local communities and elected officials about the impact of this E.O. In its statement, FASEB noted that the E.O. represented the latest effort to convert previously apolitical and consensus-driven processes into vehicles for partisan priorities and codify policies currently being challenged in court.
- Responded to a Request for Information (RFI) issued by NIH seeking input on five options for limiting publishing costs charged to research grants, FASEB highlighted the ways in which the proposed strategies would nullify the agency's efforts towards fostering Gold Standard Science.
- Submitted comments opposing a Department of Homeland Security (DHS) proposal that would replace "duration of status" admissions for F and J visa holders with a fixed four-year period. FASEB warned that the rule would create unnecessary administrative and financial burdens for current international scholars, discourage rising global talent from pursuing training in the U.S., and ultimately weaken the STEM workforce at a time of growing national demand for highly trained researchers.

Supporting International Scholars – FASEB produced two new resources in support of international scholars and their contributions to the U.S. research community:

- International Scholars: Key Statistics Brief – highlights the contributions of international researchers to the U.S. scientific enterprise and identifies challenges they face, including policy changes that affect recruitment, retention, and career development.
- Navigating U.S. Visa Systems for International Scholars Factsheet – provides an overview of the primary visa categories used by international scholars in the U.S., outlining eligibility, limitations, and pathways to longer-term employment or residency.

NOTE: These resources are exclusive to members of FASEB member societies and are behind a paywall. SLB members should contact SLB to obtain the log-in credentials to access these materials.