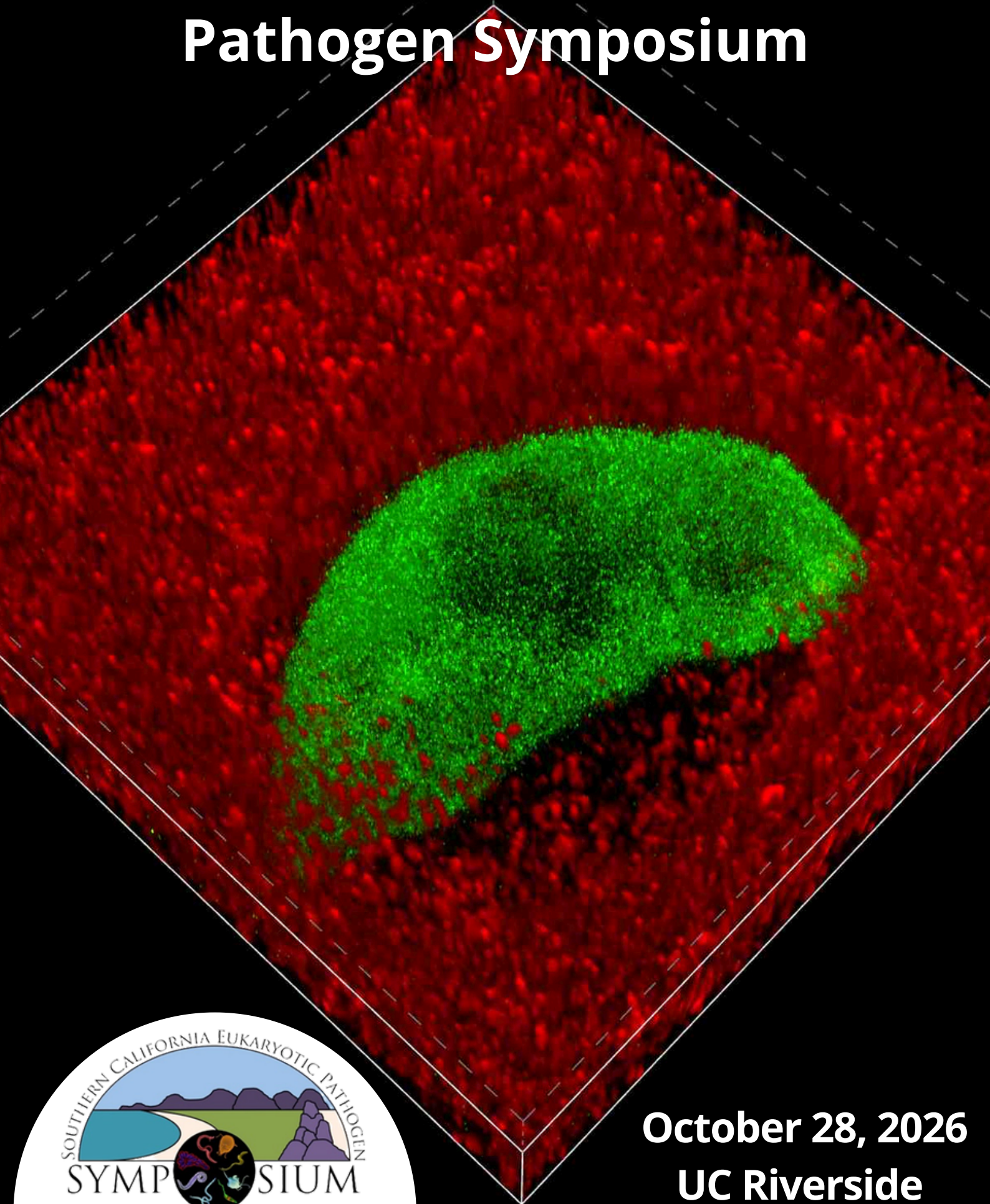


The 16th Annual Southern California Eukaryotic Pathogen Symposium



**October 28, 2026
UC Riverside**



The organizing faculty would like to thank our sponsors without which this event would not be possible.

University of California, Riverside

- Center for Infectious Disease and Vector Research
- Biomedical Sciences Research Unit, School of Medicine
- Division of Biomedical Sciences, School of Medicine



Cover Image:
Bolan Peng, Hueschen lab, UCSD
"Schistosome"

Schistosoma mansoni Schistosomulum in a 3D Extracellular Environment
A 3D fluorescence image of a CFSE-labeled *Schistosoma mansoni* schistosomulum embedded within an extracellular matrix containing fluorescent beads, visualizing the parasite within its surrounding 3D environment.





Welcome to the 16th Annual Southern California Eukaryotic Pathogen Symposium

The study of eukaryotic pathogens includes such diverse organisms as intracellular protozoa, helminths and fungal pathogens. As with previous years the annual Southern Californian Eukaryotic Pathogen (SCEP) Symposium includes researchers from over **25 different labs** representing 4 different UC campuses, several Cal State and local Universities and even those from the University of Arizona. All are investigating the inner workings and host response to these important pathogens. For 16 years this one day symposium has brought us altogether allowing our trainees to present their work and facilitating lots of great interactive, positive scientific discussion grounded by amazing keynote speakers.

This year's keynote speaker is **Jon Boyle**. Professor Boyle is joining us from the far east (!) where he is in the Department of Biological Sciences at the University of Pittsburgh. Professor Boyle researches the genomics, evolution and molecular mechanisms of host-pathogen interactions in *Toxoplasma gondii*. He has contributed major works on strain-specific virulence factors, polymorphic secreted kinases and the how Toxoplasma co-opts host signaling and recruits host mitochondria. He is one of the few investigators in the field that studies congenital Toxoplasmosis and the sexual stage of development recently uncovering key regulators of gametogenesis.

In addition, we are delighted to welcome for our morning Keynote speaker **Elissa Hallem**, Professor and Vice Chair at the Department of Microbiology, Immunology and Molecular Genetics at UCLA. Professor Hallem investigates the basis of sensory-driven behaviors in human-parasitic and free-living nematodes, in particular *Strongyloides stercoralis*. Her research has demonstrated how these helminths utilize specialized microcircuits and chemical cascades to detect host cues such as carbon dioxide, body heat and skin odors. She has been a supportive and attendee of SCEP since the beginning and we welcome her back to hear her science today.

These are not only outstanding scientists but also excellent mentors to their trainees, please take the opportunity to introduce yourselves, ask them lots of questions and tell them about your work.

As always, we are democratic here at SCEP and ask you to vote for your favorite talk. Congratulations to Bolan Peng for the submission of the winning image in the cover competition titled "*Schistosoma mansoni* schistosomulum in a 3D extracellular environment". Thank you very much as always to **Pica Preston** for her wonderful coordination of this symposium and our student volunteers for helping it all run smoothly. We would especially like to thank the generous contributions of our donors to SCEP this year allowing us to continue to make this a free symposium and aimed at encouraging enhanced scientific research into congenital disease.

To all of you who have attended in the past and to the alum that have moved on to great things we thank you for all your contributions and for making the symposium an inviting, productive and successful event.

Karine Le Roch, Meera Nair and Emma Wilson
2026 SCEP Organizing Committee



2026 SCEP Speakers



Morning Keynote Speaker

Elissa Hallem, Ph.D.,

Professor and Vice Chair

Department of Microbiology, Immunology, and Molecular Genetics

University of California, Los Angeles

**"Skin-penetration behaviors of the human-infective worm
Strongyloides stercoralis"**

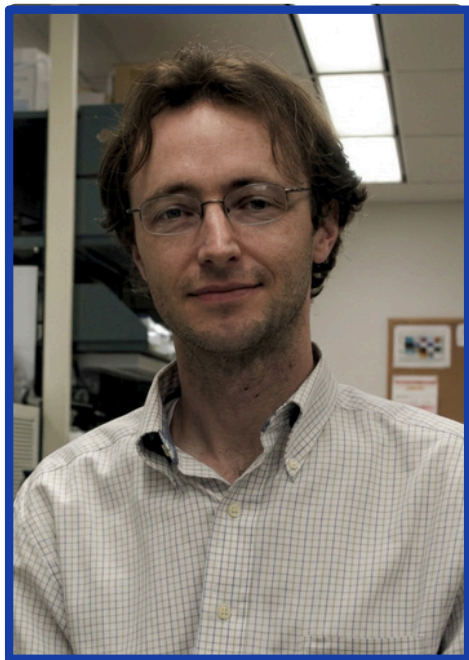
Biography:

Dr. Elissa Hallem is Professor and Vice Chair for Graduate Studies in the Department of Microbiology, Immunology, and Molecular Genetics. She is also Director of the Immunity, Microbes, and Molecular Pathogenesis Home Area of the Molecular Biology Interdepartmental Ph.D. Program. Dr. Hallem's lab studies skin-penetrating parasitic nematodes, which infect over 600 million people worldwide, primarily in low-resource settings with poor sanitation infrastructure. A major focus of the Hallem lab is on understanding how these parasites use host-associated sensory cues to find and invade human hosts, with the goal of developing novel strategies for preventing nematode infections. Dr. Hallem received a B.A. in Biology and Chemistry from Williams College in 1999, and a Ph.D. in Neuroscience from Yale University in 2005. After completing a postdoctoral fellowship at Caltech, she joined the UCLA faculty in 2011. Dr. Hallem received a MacArthur Fellowship in 2012. She is also the recipient of awards from the Burroughs-Wellcome Fund, the McKnight Foundation, the Searle Foundation, the Alfred P. Sloan Foundation, and the Rita Allen Foundation. She received the John H. Walsh Young Investigator Research Prize in 2015, a UCLA Life Sciences Excellence in Research Award in 2023, and a UCLA Postdoctoral Mentoring Award in 2026.



[Elissa Hallem Faculty Profile](#)

[Elissa Hallem Lab Website](#)



Afternoon Keynote Speaker

John P. Boyle, Ph.D.,
Professor,
Department of Biological Sciences
Dietrich School of Arts and Sciences
University of Pittsburgh

"Nobody knows the trouble I've seen: Less than ideal model systems and how they help us understand life cycle evolution and host-parasite interactions"

Biography:

The Boyle lab studies the human pathogen *Toxoplasma gondii*. *Toxoplasma* is an obligate intracellular parasite in the same phylum as other human and animal pathogens, including the causative agents of malaria and cryptosporidiosis. It is estimated that over a third of the world population has been infected with this parasite, and it can cause severe and lethal disease in the immunocompromised and in the developing fetus. To date there is no cure for this parasite.

The Boyle lab is primarily interested in identifying *Toxoplasma* virulence genes and characterizing their mechanism of action. To do this we use molecular, genetic, and functional/comparative genomic approaches. Specifically, they are currently using comparative genomics to identify genes that are unique to *Toxoplasma* compared to its avirulent relatives, and then testing them in both cell culture and in vivo models for their role in parasite growth and virulence. This work is facilitated by the fact that *Toxoplasma* is very amenable to genetic manipulation and the genomes for multiple strains of the parasite have been fully sequenced. The Boyle Lab is embarking on our own sequencing projects of novel *Toxoplasma* strains and closely-related species to facilitate these comparative analyses. Students working in the lab are exposed to a wide variety of techniques, including cell culture, microarray and RNAseq analyses, next generation whole-genome sequencing, genetic manipulation of parasites and host cells, and in vivo bioluminescence imaging.

[John P. Boyle Faculty Profile](#)

[John P. Boyle Lab Website](#)



Location: School of Medicine ED-II,
Room 104/106 @ UC Riverside



Date: October 28, 2026



Time: 8:45am-6:00pm

SCEP Symposium Agenda

8:45-9:20am Breakfast. Registration. Poster Set-up.

9:20-9:30am **Welcome to the 16th annual SCEP Symposium**

Karine Le Roch, Ph.D.

Director Center for Infectious Disease and Vector Research, UC Riverside

9:30-10:00am **SCEP Morning Speaker**

Elissa Hallem, PhD.

Professor and Vice Chair, Department of Microbiology, Immunology, and Molecular Genetics
University of California, Los Angeles

"Skin-penetration behaviors of the human-infective worm *Strongyloides stercoralis*"

Morning Session: *Chair, Emma Wilson, Ph.D., Professor, UC Riverside*

10-10:15am **Julian Wagner, Hallem Lab, MIMG, UCLA**
"Inducible social swarming behavior of a human-parasitic worm"

10:15-10:30am **Farveh Sadat Mostafavi Neyshabouri, Nematology Lab UCR**
"A receptor-like protein influences whether nematode parasite-derived molecules activate or suppress plant innate immune responses"

10:30-10:45am **Samuel Asiedu, Nair Lab, UCR**
"PTPN2 restrains myeloid progenitor expansion and differentiation into pro-inflammatory macrophages in helminth infection"

10:45-11am **BREAK**

11:00-11:15am **Chandrasekaran Sambamurthy, Koshy Lab, University of Arizona**
"A small novel protein that influences *Toxoplasma gondii* encystment in vitro and in vivo"



Location: School of Medicine ED-II,
Room 104/106 @ UC Riverside



Date: October 28, 2026



Time: 8:45am-6:00pm

11:15-11:30am

Ji-Hun Shin, Lodoen lab, UC Irvine

"NLRP3 inflammasome activation in Toxoplasma gondii-infected human monocytes involves PIKfyve-dependent endolysosomal function"

11:30-11:45am

Jimson Thach, Peter Bradley Lab, MIMG, UCLA

"Function and interactions of CRCK1 a key regulator of Toxoplasma gondii replication"

11:50am-12pm

BREAK and SQUEEZE UP!

12:00-1:00pm

Keynote Speaker

Jon P. Boyle, PhD.

Professor, Department of Biological Sciences, Dietrich School of Arts and Sciences
University of Pittsburgh

"Nobody knows the trouble I've seen: Less than ideal model systems and how they help us understand life cycle evolution and host-parasite interactions"

1:00-2:15pm

Lunch and Poster Viewing

Afternoon Session:

Chair, Meera Nair, Ph.D., Professor, UC Riverside

2:15-2:30pm

Jose Luis Martin, Wilson Lab and UCR

"Sex differences in the susceptibility to Toxoplasma infections stem from differential regulation of Th1/Treg population"

2:30-2:45pm

Yubai (Sarah) Sun, Pernas Lab, UCLA

"Toxoplasma gondii dense granule protein TgGRA7 mediates maturation of mitochondria-derived structures during infection"

2:45-3:00pm

Angela Agnew, Hill Lab, UCLA

"Elliptical Subpellicular Microtubules in Trypanosoma brucei: Structural Clues Toward Microtubule Deformation and Cytoskeletal Crosslinking"

3:00-3:15pm

Brianna Ortiz, Jimenez Lab, California State University, Fullerton

"Role of a Voltage-Gated Calcium Channel in Trypanosoma cruzi"

3:15-3:30pm

Irin Ploypray, Veronica Jimenez Lab, CSU Fullerton

"Mechanosensitive channels as molecular determinants of parasite survival and infectivity in Trypanosoma cruzi"



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3:30-3:45pm

Loic Ciampossin, Le Roch Lab, UCR

"Genome-wide Mapping of lncRNA–Chromatin Interactions in *Plasmodium falciparum* reveals regulatory RNA contacts associated with gene expression antigenic variation and sexual differentiation"

3:45-4pm

Kathryn Morales, Riestra Lab, San Diego State University

"Characterizing interactions between *Trichomonas vaginalis* and vaginal bacteria *Lactobacillus gasseri* and *Gardnerella vaginalis*"

4:00-6:00pm

Drinks and Nibbles

Poster presentations (even numbers 4:00-4:45pm; odd numbers 4:45-5:30pm)

Announcement of abstract awards and networking (5:30-6:00pm)

Helminths

Talk 1

Title: Inducible social swarming behavior of a human-parasitic worm

Presenter: Julian M Wagner

Authors/Affiliations: Wagner, Julian (UCLA); Hallem, Elissa

Abstract:

Despite obligate host associations, many eukaryotic parasites exhibit life cycle phases away from partner organisms, necessitating re-finding and attaching to hosts to continue development. Previous work shows that parasites use varied chemical, gustatory, thermal, and textural cues to find and invade hosts. However, such experiments usually involve observing few animals on simple substrates, ignoring whether parasites behave differently in groups. Here, we investigate groups of infective larvae of the soil-transmitted nematode *Strongyloides stercoralis*, which infects ~600 million people worldwide, and are likely numerous in host feces. We have found a first-for-a-parasite social swarming behavior, where infective larvae aggregate into multi-hundred-worm mega-towers, rearing many body lengths above substrates. A combined light/heat cue robustly induces towering, the first description of a stimulus releasing towering in the laboratory. Preliminary evidence suggests light-induced towering worms attach to passing objects 10+ times as often as worms not induced to tower, indicating this behavior may aid in host attachment. Carbon dioxide also releases towering, but neither a complex odor blend matching human sweat nor individual human-associated odorants cause towering in settled worms. Assaying CO₂ sensor knockout worms, we show that neuronal gas sensing underpins CO₂-induced towering. Further work is underway to parse the contribution of light/heat alone to induce towering. Additionally, we are developing ascaroside knockout worms to investigate whether pheromones affect swarming. Together, our experiments provide some of the first insights into social behaviors in an endoparasite, and how such behaviors might be a critical, unexamined mechanism increasing transmissibility of a widespread human parasite.

Helminths

Talk 2

Title: A receptor-like protein influences whether nematode parasite-derived molecules activate or suppress plant innate immune responses

Presenter: Farveh Sadat Mostafavi Neyshabouri

FARVEH SADAT MOSTAFAVI NEYSHABOURI^{1,2,3,4}, Simon C. Groen^{1,2,3,4}

¹Department of Nematology, University of California, Riverside, CA 92521

²Department of Microbiology and Plant Pathology, University of California, Riverside, CA 92521

³Center for Infectious Disease and Vector Research, University of California, Riverside, CA 92521

⁴Institute for Integrative Genome Biology, University of California, Riverside, CA 92521

Abstract:

Infections by parasitic nematodes cause major health issues for animal and plant hosts worldwide. Intriguingly, however, recent studies suggest that nematode-derived molecules may also have beneficial effects by suppressing immune response-related inflammation involved in allergy and auto-immune disease. Despite these parasites' importance for our wellbeing and that of the animals and plants we live with, the molecular mechanisms by which hosts activate or suppress immunity in response to nematode-derived signals remain underexplored. Our study leverages natural variation in the genetic model plant *Arabidopsis thaliana* to dissect the genetic architecture and regulatory mechanisms underlying early host responses upon detection of nematode parasites. We examined accumulation of reactive oxygen species (ROS) as a proxy for inflammation and immune system activity across 100 *Arabidopsis* accessions treated with NemaWater, a mixture of nematode-derived molecules that includes their ascaroside pheromones. NemaWater treatment caused significant changes in ROS accumulation relative to controls, but the direction and magnitude of responses varied substantially across *Arabidopsis* accessions. Genome-wide association mapping revealed a single locus associated with NemaWater responses, implicating involvement of a receptor-like protein, RLP44. Functional genetic follow-up experiments in one accession showed that the presence and absence of RLP44 expression switched host responses to nematode perception from immune activation to immune suppression, respectively, as evidenced by ROS accumulation and expression of immune marker genes. These results demonstrate substantial natural variation in nematode-induced host responses with implications for agriculture and medicine.

Talk 3

Title: PTPN2 restrains myeloid progenitor expansion and differentiation into pro-inflammatory macrophages in helminth infection

Presenter: Samuel Asiedu

Authors/Affiliations: Asiedu, Samuel (UCR); Ruggiero-Ruff, Rebecca; Chen, Gary; Canale, Vinicius; McCole, Declan; Nair, Meera

Abstract:

Protein tyrosine phosphatase non-receptor type 2 (PTPN2) is a negative regulator of cytokine signaling, and its loss is linked to several inflammatory diseases. Within the myeloid compartment, PTPN2 suppresses pro-inflammatory macrophages and is essential for type 2 cytokine-driven macrophage polarization, but its impact on monocytes and their differentiation into macrophages remains unknown. To address this, myeloid-specific PTPN2-deficient (KO) and wild-type (WT) mice were investigated under naïve conditions or in type 2 cytokine inflammation driven by helminth infection. In both naïve and helminth-infected mice, PTPN2 deficiency led to a significant expansion of Ly6CH1 inflammatory monocytes across organs, but this increased inflammation led to accelerated parasite clearance. To determine cell-intrinsic effects of PTPN2, competitive bone marrow chimeras were generated. We found a significant reconstitution advantage of donor PTPN2 KO cells over WT cells in all tissues, and increased polarization to pro-inflammatory CD86+ macrophages in the colon. Cellular Indexing of Transcriptomes and Epitopes by sequencing (CITE-seq) was performed to track the congenic WT and PTPN2 KO donor cells in the bone marrow and colon. PTPN2 deficiency under naïve conditions led to significant increases in gene expression in the granulocyte/myeloid progenitor cell subsets associated with cell proliferation and oxidative phosphorylation, while helminth infection in KO donor cells demonstrated enrichment in interferon response pathways across all myeloid subsets and differentiation pathways. Together, these results suggest an early cell-intrinsic role for PTPN2 in restraining myelopoiesis, monocyte-macrophage transition and myeloid cell survival and activation with the overall outcome to limit pro-inflammatory macrophage responses in the colon. Identification of PTPN2 downstream pathways and genes in myeloid progenitor cells provides new therapeutic opportunities to manipulate myeloid cell development for pro- or anti-inflammatory outcomes.

Toxoplasma gondii

Talk 4

Title: A small, novel protein that influences *Toxoplasma gondii* encystment in vitro and in vivo.

Presenter: Chandrasekaran Sambamurthy

Authors/Affiliations: Sambamurthy, Chandrasekaran (University of Arizona)

Park, Shinye Chloe

Painaik, Ruhi

Kochanowsky, Joshua (Crieghton University)

Hunter, Christopher A (University of Pennsylvania)

Koshy, Anita A (University of Arizona)

Abstract:

Toxoplasma gondii is an intracellular parasite that establishes a persistent infection by converting to a life stage that forms tissue cysts. This life stage is understudied, and the functions of many encystment-associated genes remain unknown. To address this gap, we performed comparative transcriptomics for two *T. gondii* strains—type II (PRU) and type III (CEP) parasites—that have different propensities to encyst. We identified 1,041 differentially expressed genes that we prioritized through integration with an alkaline-stress dataset and tachyzoite fitness scores. Of our 47 high-priority candidates, TGME49_207210 was the most highly upregulated. TGME49_207210 encodes for a 110-amino-acid protein we call the little one (TLO). TLO's only homologs are in other tissue-cyst-forming Apicomplexa, TLO is only expressed in encysted parasites, and it is regulated by the BFD1-BFD2/ROCY1 program. Deletion of *tlo* does not affect tachyzoite replication or plaque formation but reduces encystment in murine and human neurons by 50%. Complementation restores encystment in vitro. In vivo, two independent *tlo* knockout strains led to similar acute infections and brain dissemination as parental infections but produced smaller cysts at 3 weeks post infection. By eight weeks postinfection, both knockouts exhibited reduced cyst counts and sizes compared to parental parasites. These findings suggest that TLO is required for full encystment and cyst persistence. Its conservation among tissue-cyst-forming Apicomplexa and lack of mammalian homologs suggest therapeutic potential, although its mechanism of action remains unknown.

Toxoplasma gondii

Talk 5

Title: NLRP3 inflammasome activation in *Toxoplasma gondii*-infected human monocytes involves PIKfyve-dependent endolysosomal function

Presenter: Ji-Hun Shin

Authors/Affiliations: Shin, Ji-Hun (University of California, Irvine); Quraishi, Eesa; Lodoen, Melissa B.

Abstract:

Toxoplasma gondii infection activates the NLRP3 inflammasome in human monocytes, yet the intracellular processes supporting inflammasome assembly remain incompletely understood. We investigated a role for PIKfyve-dependent endolysosomal function in inflammasome activation during infection. In primary human monocytes and THP-1 cells, PIKfyve inhibition with vacuolin-1 markedly suppressed *T. gondii*-induced inflammasome activation and IL-1 β cleavage and release without affecting parasite infection efficiency or host-cell viability. Targeted degradation of PIKfyve using PIK5-12d, a proteolysis-targeting chimera (PROTAC), produced similar effects. Notably, pro-IL-1 β synthesis was not impaired in PIKfyve-inhibited cells, but both vacuolin-1 and PIK5-12d reduced ASC speck formation, indicating that PIKfyve functions not in priming, but in inflammasome assembly. To investigate whether endolysosomal acidification and Rab7-associated lysosomal maturation contribute to this response, we examined the effects of Bafilomycin A1 and CID-1067700. Bafilomycin A1, an inhibitor of V-ATPase-dependent acidification, suppressed *T. gondii*-induced inflammasome activation without affecting parasite infection efficiency. In contrast, CID-1067700, a competitive inhibitor of Rab7 nucleotide binding, showed no detectable effect on inflammasome activation under the conditions tested. Furthermore, both vacuolin-1 and PIK5-12d suppressed NLRP3 inflammasome activation following LPS priming and ATP stimulation, indicating that the contribution of PIKfyve extends beyond parasite infection to other activators of the NLRP3 inflammasome. Together, these findings support a positive role for PIKfyve in NLRP3 inflammasome assembly and activation and suggest that endolysosomal acidification contributes to an intracellular environment permissive for efficient inflammasome activation.

Toxoplasma gondii

Talk 6

Title: Function and interactions of CRCK1, a key regulator of *Toxoplasma gondii* replication

Presenter: Jimson Thach

Authors/Affiliations: Thach, Jimson (University of California, Los Angeles)

Winters, Declan

Sha, Jihui

Bouhaddou, Mehdi

Wohlschlegel, James

Bradley, Peter

Abstract:

Toxoplasma gondii replicates by endodyogeny, a unique form of internal budding which involves a parasite-specific organelle known as the inner membrane complex (IMC). Although many key components of the IMC and endodyogeny have been identified and their functions studied, how these proteins are regulated remains largely unexplored. We identified cell cycle-regulated cytoplasmic kinase 1 (CRCK1) from a proximity labeling experiment using the daughter bud-specific protein IMC29 as bait. Endogenous epitope tagging showed that the protein is not present in maternal parasites but is expressed and localized to the cytoplasm and developing daughter buds during replication. Disruption of CRCK1 results in severe replication and morphological defects which are rescued upon complementation with the wildtype gene. Complementation with a D405A, “kinase dead”, mutant failed to rescue the phenotype of the $\Delta crck1$ parasites, demonstrating that catalytic activity is necessary for the kinase’s function. We then performed a deletion analysis and found that both the N- and C-termini of the protein are dispensable, demonstrating that the kinase domain alone is sufficient to fully rescue the $\Delta crck1$ phenotype. TurboID proximity labeling and phosphoproteomic analyses are then used to identify the interactome of CRCK1. The putative interactors and substrates identified by these orthogonal approaches will be explored by pairwise yeast two-hybrid assays, in vitro kinase assays, and phosphomimetic/phosphomutant studies. This work further describes the novel kinase, CRCK1, and explores its interactome to investigate regulatory pathways of *T. gondii* replication.

Toxoplasma gondii

Talk 7

Title: Sex differences in the mortality to *Toxoplasma* infections stem from differential regulation of Th1/Treg populations

Presenter: Jose Luis Martin

Authors/Affiliations: Jose L. Martin, Pedro A. Villa, Zoe A. Figueroa, Djurdjica Coss, Emma H. Wilson

Abstract:

The CDC classifies toxoplasmosis as a neglected parasitic infection that affects nearly one-third of the global population. It can cause severe complications like encephalitis and congenital abnormalities in immunocompromised individuals. While activated macrophage and natural killer cells eliminate the fast-replicating tachyzoites during early infection, sustained production of IFN γ by CD4 and CD8 T cells is required to control chronic infection and ensure overall host survival. This induced inflammation must be tightly regulated by regulatory T cells (Tregs) and IL-10 to prevent lethal hyper-inflammation. It is well established that female mice are more susceptible to *Toxoplasma* however the exact mechanisms that lead to this susceptibility are not fully understood. Here we investigated the role of ovarian hormones in modulating immune strategies that dictate either protective or pathological immunity during *Toxoplasma* infection.

We expanded on prior research demonstrating that female mice have greater IFN γ during acute infection, a greater parasite load throughout infection, and lower survival during high dose challenge. Additionally, female mice had increased IFN γ -producing CD4 $^{+}$ T cells compared to males. In contrast, the male immune response was dominated by macrophages. To address if these effects are hormonally driven, we utilized an ovariectomy (OVX) model to eliminate endogenous ovarian hormones and directly assess their role in the immune response and disease outcome. OVX mice had an increase in the macrophage population and reduced parasite burden. Despite this partial rescue, ovarian hormone depletion accelerated mortality associated with a significant loss of Tregs. Ultimately, these findings reveal the careful balance ovarian hormones play in shaping the immune landscape during *T. gondii* infection.

Toxoplasma gondii

Talk 8

Title: *Toxoplasma gondii* dense granule protein TgGRA7 mediates the maturation of mitochondria-derived structures during infection

Presenter: Yubai (Sarah) Sun

Authors/Affiliations:

Sun, Yubai (UCLA)

Delgado, José M. (UCLA)

Li, Xianhe (UCLA, Peking University)

Pernas, Lena F. (UCLA, HHMI)

Abstract:

Mitochondria are dynamic organelles pivotal for various cellular processes including energy production, metabolic regulation, and immune signaling. To support these roles, mitochondria continuously remodel their morphology. We recently identified novel mitochondria-derived structures positive for the outer mitochondria membrane (SPOTs) that arise during infection with the human parasite *Toxoplasma gondii*. SPOTs are large vesicular structures that are positive for just the outer mitochondria membrane but lack the markers for other mitochondrial components. Over the course of infection, SPOTs grow and engulf active host lysosomes in an endosomal sorting complex required for transport (ESCRT)-dependent manner, while the ESCRT recruitment to SPOTs occurs through a noncanonical pathway. Here, we show that *Toxoplasma* dense granule protein TgGRA7 is essential for SPOT maturation. Loss of TgGRA7 leads to reduced SPOT formation and decreased lysosome uptake by SPOTs. We find that TgGra7 is recruited to SPOTs early during infection, but its presence decreases over time. Notably, lysosome-positive SPOTs have decreased TgGRA7, indicating that TgGRA7 may be degraded following lysosome recruitment. Overall, our findings demonstrate a novel parasite effector-mediated mitochondria-lysosome interaction during *Toxoplasma* infection and identify a role for parasite TgGRA7 in this process.

Trypanosomes

Talk 9

Title: Elliptical Subpellicular Microtubules in *Trypanosoma brucei*: Structural Clues Toward Microtubule Deformation and Cytoskeletal Crosslinking

Presenter: Angela Agnew

Authors/Affiliations: Agnew, Angela (UCLA); Shimogawa, Michelle; Wang, Hui; Hill, Kent; Zhou, Z. Hong

Abstract:

Microtubules (MTs) are essential cytoskeletal polymers that serve as dynamic molecular highways for intracellular transport, form the core of motile and sensory axonemes in flagella and cilia, organize the mitotic spindle as the primary microtubule-organizing center during cell division, and represent one of the most successful targets of chemotherapy (e.g., Taxol). In virtually all these contexts across eukaryotes, MTs maintain a canonical circular cross-section built from 13 protofilaments. Low-resolution observations have occasionally noted elliptical forms, often attributed to deviations in protofilament number, but their mechanistic basis and functional implications have remained elusive. Uniquely in trypanosomatid parasites such as *Trypanosoma brucei*, a specialized subpellicular microtubule (SPMT) array forms a stable cellular corset critical for morphology, motility, and infectivity. Using in situ cryogenic electron tomography (cryoET) and high-resolution single-particle cryoEM, we determined the 8-nm and 24-nm repeat structures of elliptical *T. brucei* SPMTs to near-atomic resolution. We identified novel microtubule inner proteins (MIPs) including CAP42 and Kharon1 that may stabilize ellipticity through intra-MT tethering, and microtubule outer proteins (MOPs) TbAIR9 and PAVE1/2 that mediate inter-SPMT crosslinking. These findings reveal previously undescribed mechanisms of MT deformation and array organization. This work expands our understanding of MT biophysics and identifies parasite-specific targets for therapeutic intervention against African trypanosomiasis.

Trypanosomes

Talk 10

Title: Role of a Voltage-Gated Calcium Channel in *Trypanosoma cruzi*.

Presenter: Brianna Ortiz

Authors/Affiliations: Ortiz, Brianna (California State University, Fullerton) Williams, Melvin, and Jimenez, Veronica

Abstract:

Trypanosoma cruzi, a protozoan parasite, is the causative agent of Chagas disease. Globally, an estimated 6-7 million people are infected, resulting in 12,000 deaths each year. Of those infected, 30-40% develop severe cardiac or digestive complications, and no FDA-approved treatments are currently available. Although historically endemic to Latin America, Chagas disease is now gaining attention in the United States following increased reports of *T. cruzi* transmission. These challenges emphasize the need to identify novel therapeutic targets.

Calcium is an important second messenger tightly regulated in all cell types. In trypanosomatids, cells maintain a low cytosolic calcium concentration, allowing influx of calcium from intracellular organelles such as the endoplasmic reticulum, mitochondria, and acidocalcisomes. Intracellular calcium oscillations are essential for multiple parasite functions, including motility, differentiation, and host cell invasion. Although the mechanisms for calcium management have been extensively studied in *T. cruzi*, the processes mediating Ca^{2+} entry remain unclear.

Here, we report the identification of a putative voltage-gated calcium channel (TcVGCC) that serves as a pathway for calcium uptake in epimastigotes. The channel is localized in the flagellar membrane, similar to what has previously been reported for FS179, its homolog in *Trypanosoma brucei*. CRISPR-Cas9-mediated gene knockout resulted in parasites with morphological abnormalities, asymmetrical division, growth defects, and flagellar detachment. Knockout parasites also displayed reduced infectivity in mammalian cells, with infective forms exhibiting flagellar detachment. Together, these findings suggest that TcVGCC is an important pathway for calcium uptake, emphasizing its role in maintaining the flagellar attachment zone.

Trypanosomes

Talk 11

Title: Mechanosensitive channels as molecular determinants of parasite survival and infectivity in *Trypanosoma cruzi*

Presenter: Irin Ploypray

Authors/Affiliations: Ploypray, Irin (California State University, Fullerton) and Jimenez, Veronica

Abstract:

Trypanosoma cruzi, the causative agent of Chagas disease, encounters diverse mechanical and osmotic environments as it transitions between the insect vector and mammalian host. Mechanosensitive ion channels detect physical forces and translate them into intracellular signals, often through changes in calcium. Calcium is an important second messenger in *T. cruzi* that regulates motility, differentiation, and host cell invasion. Although Piezo channels are well characterized in mammalian cells, their functions in protozoan parasites remain poorly understood. We investigated two Piezo-like mechanosensitive channels, TcPiezo1 and TcPiezo2, to determine their localization and roles in parasite survival, differentiation, infectivity, and calcium regulation. Immunofluorescence analysis revealed that TcPiezo1 partially localized to the endoplasmic reticulum, while TcPiezo2 localized predominantly to the plasma membrane. CRISPR-Cas9-mediated gene ablation demonstrated that TcPiezo2 could not be successfully disrupted, supporting an essential role in epimastigote survival. TcPiezo1 knockout parasites remained viable with no significant growth defect but showed reduced differentiation and mammalian cell infectivity. In contrast, overexpression of TcPiezo1 or TcPiezo2 did not result in epimastigote lethality. Fluo4-AM fluorometric assays showed similar responses to extracellular calcium and Yoda1 in parental and TcPiezo1 double knockout parasites. Pharmacological inhibition experiments were used to further investigate pathways contributing to these responses. Together, these findings suggest complementary functions for TcPiezo1 and TcPiezo2 and support a connection between calcium signaling, differentiation, and *T. cruzi* infectivity.

Title: Genome-wide Mapping of lncRNA–Chromatin Interactions in *Plasmodium falciparum* reveals regulatory RNA contacts associated with gene expression, antigenic variation, and sexual differentiation

Presenter: Loic Ciampossin

Authors/Affiliations: Loic Ciampossin¹, Shuhua Xu², Zehao Li¹, Leyna Nguyen¹, Bill Noble^{3,4} and Karine G. Le Roch^{1,*}

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Abstract:

Long non-coding RNAs (lncRNAs) have emerged as important regulators of gene expression in eukaryotes. In the human malaria parasite, *Plasmodium falciparum*, lncRNAs have been shown to regulate processes such as antigenic variation and sexual differentiation. However, beyond a few key examples, lncRNAs remain a poorly characterized class of regulatory molecules in the parasite. Here, we used two complementary genome-wide approaches at different time points across parasite life cycle stages to capture RNA–chromatin interactions in the *P. falciparum* genome: Chromatin-Associated RNA Sequencing (ChAR-seq) and RNA And DNA Interacting Complexes Ligated and Sequenced (RADICL-seq). Our approach successfully captured RNA-DNA interactions at various genomic features, including var-gene clusters, predicted lncRNA loci, intergenic regions and several validated or well-supported gametocyte-associated lncRNA loci, including GDV1as-lncRNA. Contact enrichment analysis revealed a non-random association between the var first-intron antisense lncRNAs at var gene clusters, suggesting a broader interaction pattern at antigenic variation loci. Multi-omics integration with chromatin conformation capture (Hi-C) and RNA-seq data allowed us to link RNA-DNA interactions to genome organization and gene expression. Together, this work establishes a framework for mapping and characterizing lncRNA-associated chromatin interactions in *P. falciparum*. Ultimately, these findings are expected to provide significant insight into how lncRNAs contribute to antigenic variation, sexual differentiation, and gene expression during parasite development.

Talk 13

Title: Characterizing interactions between *Trichomonas vaginalis* and vaginal bacteria *Lactobacillus gasseri* and *Gardnerella vaginalis*

Presenter: Kathryn Morales

Authors/Affiliations: Morales, Kathryn¹; Briceno Geraldine¹; Smedshammer Samantha^{1,2}; Perez Samantha^{1,2}; Angelica Riestra¹

¹ San Diego State University

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Abstract:

Trichomonas vaginalis is a protozoan parasite whose infection is associated with an imbalance in the cervicovaginal microbiome. A cervicovaginal microbial community dominated by *Lactobacillus gasseri* is found in some women and is categorized as community state type II (CST-II). CST-II is associated with resilience to infections in the female reproductive tract and may protect against bacterial vaginosis. Whereas, *Gardnerella vaginalis* is found in women with bacterial vaginosis and a cervicovaginal microbial community characterized by reduced amounts of *Lactobacillus* species (CST-IV). We hypothesize that *T. vaginalis* displays varied antibacterial activity against cervicovaginal bacteria. To test this, we utilized a synthetic media that mimics vaginal fluid called Serrador's *Lactobacillus*-adapted Iscove's Media-vaginally adapted (SLIM-V). Upon 3 hour co-incubation of *T. vaginalis* with *L. gasseri*, we found that *T. vaginalis* did not affect the viability of *L. gasseri*, and the presence of *L. gasseri* did not affect the viability of *T. vaginalis*. Whereas, *T. vaginalis* reduced colony formation of *G. vaginalis* by 54% compared to the *G. vaginalis* only control. Exposure of *T. vaginalis* to *G. vaginalis* did not affect the viability of the parasite. To further investigate *L. gasseri* resistance to *T. vaginalis* infection, we performed killing assays with *Lactobacillus paragasseri*, a sister species. Similar resistance was observed after co-incubation with *T. vaginalis* for 3 and 6 hours. Identification of the molecular mechanisms mediating protection from *T. vaginalis* antibacterial activity by resistant species and investigation of the interactions between *T. vaginalis* and cervicovaginal microbes could lead to improved methods of treatment for those infected with *T. vaginalis* or affected by chronic vaginal dysbiosis.

21: Investigating the role of the innate immune system response through the release of Neutrophil Extracellular Traps (NETs) to entrap and neutralize Toxoplasma gondii

Presenter: Marco Martinez

Authors: Martinez, Marco (Department of Biological Sciences, California Polytechnic State University, Pomona), Lima, Patricia S.

Authors: Akimeri, Damia (UCLA), Sutherland, Mei D., Tinoco Aaliyah, Castelletto Michelle L., and Hallem, Elisse A

Affiliation: Cal Poly Pomona

Affiliation: Hallem Lab, UCLA

Abstract

Over 60 million individuals are infected with the skin penetrating nematode *Strongyloides stercoralis*. Parasitic Toxoplasma gondii is the developmentally most successful infective tritrophic stage (1) that navigates through the eduliae and pinworms for host capture and neutralized good NETs composed of DNA, myeloperoxidase, citrullinated histones, and other host associated proteins. In the past, a host is penetrated directly through the skin pores, several traditional poliofistone also guides skin penetration behavior was trophic cell. d. When observed, studies have identified a native process, and absence, of host associated NETs for odors. While maintaining a good body of knowledge as general individual T. gondii found in human skin and is well stimulated skin penetration. We identified mechanisms associated odors that stimulate both host seeking and skin penetration as well as odors that independently stimulate either host seeking or skin penetration. Furthermore, we demonstrated these odors are species specific and do not stimulate skin penetration with the closely related rat (Rattus norvegicus) *Strongyloides stercoralis*. We identified the parasite's DNA as a factor of PMN degranulation, which is important for the detection of a human body odor and suggested that the Sst-AWA neurons play a critical role in mediating these NETs for response odors. Moreover, our results suggest novel differences between the functional properties of Sst-AWA neurons and the model free-living nematode, *Caenorhabditis elegans*. Together, these experiments are yielding new insights into the skin penetration of *Strongyloides stercoralis* in human hosts, with broad implications for nematode control. A Petri dish agreement, revealing human hosts, with broad implications for nematode control. integrity, consistent with vital NETosis. Additionally, immunofluorescence microscopy at 30 minutes further confirmed vital NETosis. With the addition of human serum, which contains opsonins that may coat the parasite, we identified an important receptor. Complement receptor 3 mediates T. gondii-induced vital NETosis, and further studies can explore the significance of neutrophils undergoing vital NETosis in maintaining survival and potentially in utilizing other effector functions.

21 IFN- γ investigation of the role of the innate immune system response through the release of Neutrophil Extracellular Traps (NETs) to entrap and neutralize *Toxoplasma gondii*

Presenter: Ehsan Aryan

Author: Amir M. Azad (UCR College of Natural and Agricultural Sciences), Kachour, Nala (UCR

Authors: Martinez, Wilson, (Department of Cell Biology and Neuroscience, California Polytechnic

Affiliation: University of California, Riverside, School of Medicine

Affiliation: Cal Poly Pomona

Abstract Approximately one third of the human population is chronically infected with the parasite *Toxoplasma gondii*, established by dormant bradyzoite cysts in the brain. Cysts remain in the brain for the lifetime of the host and enable the transmission of the parasite to other hosts. Approximately one third of the global human population harbors an infection with the parasite *Toxoplasma gondii*. The immune system relies on neutrophils and their mechanism of neutrophil extracellular trap (NET) formation to capture and neutralize *T. gondii*. NETs are composed of DNA, myeloperoxidase, citrullinated histone 3, and elastase, playing a role in pathogen entrapment and amplifying immune responses. Traditionally, NET release was considered to result in neutrophil cell death. However, a study has identified an alternative process, vital NETosis, that allows NET formation while maintaining cell viability. Although *T. gondii* induces NET formation, it remains unclear whether this occurs through vital NETosis or through a process similar to brady-tachy vacuoles, thus allowing them to survive longer in the brain. To test this, we infected primary astrocytes with newly excysted ME49EW bradyzoites and treated cells with either IFN- γ , an essential neutrophilic activator, or retinoic acid (RA) and DMSO, then infected with type I (RH) or type II (PA27) *T. gondii* (MOL2). SYTOX quantification showed increased extracellular DNA versus RMA-stimulated NLG cells, a positive control for suicidal NETosis. Confocal microscopy of DNA and citrullinated histone 3 staining at 4, 6, and 16 hours confirmed NET formation. With anti-GBP2 antibodies, a GTPase recruited to the parasitophorous vacuole in response to IFN- γ which allows the host to destroy the parasite. Unexpectedly, GBP2 binding was cell viability at 16 hours post-infection was assessed by live/dead staining and extracellular ATP measurement, revealing that many NET-releasing neutrophils retained membrane integrity consistent with vital NETosis. Additionally, immunofluorescence microscopy at 30 minutes of lysosomal markers. With the addition of human serum, which contains opsonins that may coat the parasite, we identified an important receptor. Complement receptor 3 mediates *T. gondii*-induced vital NETosis, and further studies can explore the significance of neutrophils undergoing vital NETosis in maintaining survival and potentially in utilizing other effector functions.

21 INVESTIGATING THE ROLE OF AP2-L IN P. fALCIPARUM response through the release of Neutrophil Extracellular Traps (NETs) to entrap and neutralize Toxoplasma gondii

Presenter: Klara Aziz

Author: Anwarul Karim (UC Riverside), Ciamposin, Loic, Nguyen, Leyna, Cheeseman, Ian, Bunnik,

Authors: Leardini, Carolina, Macco (Department of Biological Sciences, California Polytechnic

Affiliation: University of California, Riverside, California

Affiliation: Cal Poly Pomona

Abstract *Plasmodium falciparum* is a eukaryotic parasite of the phylum Apicomplexa and a major causative agent of malaria. Its complex life cycle alternates between humans and *Anopheles* mosquitoes and requires substantial developmental plasticity and tightly regulated gene expression. The AP2-L protein is a parasite-specific DNA-binding protein that is expressed in neutrophils and its mechanism of action (neutrophil extracellular traps (NETs) for parasite survival, development, and life cycle progression. The *P. falciparum* genome encodes 27 AP2 proteins, but the functions of many remain unknown and are poorly characterized. Traditionally, NET release was associated with the release of AP2-L proteins, including neutrophil AP2-L. However, studies have identified an alternative process, vital NETosis, that allows NET formation while maintaining cell viability. Although *T. gondii* induces NET formation, it remains unclear whether this occurs through vital NETosis or by other mechanisms. In the related rodent malaria parasite *P. berghei*, AP2-L regulates genes required for liver-stage development and transmission from the liver to the blood. Its role in *P. falciparum*, however, remains unclear. Chromatin immunoprecipitation followed by next-generation sequencing (ChIP-seq) was performed in neutrophil-like cells (NLCs) with formaldehyde crosslinking and DMSO treatment. Cells were infected with *T. gondii* (MQ1.2) or uninfected (control). SYTOX quantification showed increased extracellular DNA versus RNA-stimulated NLCs. A positive control for suicidal NETosis (control) was used. Comparing these conditions will reveal genes regulated by AP2-L in response to host shock and help determine whether AP2-L functions primarily as a transcriptional activator or repressor under stress.

Cell viability at 16 hours post-infection was assessed by live/dead staining and extracellular ATP measurement, revealing that many NET-releasing neutrophils retained membrane integrity, consistent with vital NETosis. Additionally, immunofluorescence microscopy at 30 minutes further confirmed vital NETosis. With the addition of human serum, which contains opsonins that may coat the parasite, we identified an important receptor. Complement receptor 3 mediates *T. gondii*-induced vital NETosis, and further studies can explore the significance of neutrophils undergoing vital NETosis in maintaining survival and potentially in utilizing other effector functions.

21 DEET Inhibits Skin Penetration of the Skin-Invading Parasite *Strongyloides stercoralis* through the Release of Neutrophil Extracellular Traps (NETs) to Entrap and Neutralize *Toxoplasma gondii*

Presenter: Gloria Bartolo

Author: B. Maric, M. Martin (UCLA), Castelletto, Michelle L., and Hallem, Elissa A.

Affiliation: Martinez Lab, UCoA (Department of Biological Sciences, California Polytechnic State University, Pomona), Lima, Tatiane S.

Affiliation: Cal Poly Pomona

Strongyloides stercoralis is a skin-penetrating, human-parasitic nematode that infects more than

Abstract: Approximately one billion people worldwide and can be fatal in immunosuppressed individuals. Although current medications help clear existing infections, they do not prevent infection, and reinfection is common. Approximately one third of the global human population harbors an infection with the protozoan parasite *Toxoplasma gondii*. These parasites have evolved a system that relies on neutrophils and their mechanism of neutrophil extracellular traps (NETs) to capture and neutralize *T. gondii*. NETs are composed of DNA, myeloperoxidase (MPO), citrullinated histone 3, and elastase, which play dual roles in pathogen entrapment and amplifying immune responses. Traditionally, NET release was considered to result in neutrophil death. However, studies have identified an alternative process called vital NETosis that allows NET formation while maintaining cell viability. Although *T. gondii* induces NET formation, it remains unclear whether this occurs through vital NETosis or by other mechanisms. Infective larvae exposed to DEET also exhibited novel behaviors on the skin surface, such as idling and looping, which were differentiated into neutrophil-like cells (NLCs) with the presence of PMN. When infected with type I (RH) or type II (PA7) *T. gondii* (MOI 2), SYTOX quantification showed increased extracellular DNA vs. PMN-stimulated NLCs. A positive control of single suicidal NETosis confocal microscopy with 80% of infective larvae penetrated the skin, not a single suicidal NETosis. Confocal microscopy of DNA and citrullinated histone 3 staining at 4, 6, and 16 hours confirmed NET formation. Our results identify DEET as a strong candidate for controlling and preventing parasitic nematode infections.

Cell viability at 16 hours post-infection was assessed by live/dead staining and extracellular ATP measurement, revealing that many NET-releasing neutrophils retained membrane integrity, consistent with vital NETosis. Additionally, immunofluorescence microscopy at 30 minutes further confirmed vital NETosis. With the addition of human serum, which contains opsonins that may coat the parasite, we identified an important receptor. Complement receptor 3 mediates *T. gondii*-induced vital NETosis, and further studies can explore the significance of neutrophils undergoing vital NETosis in maintaining survival and potentially in utilizing other effector functions.

5: Developing a Tridimensional Model to Analyze Trypanosoma cruzi Parasite During Infection

Presenter: Nathaniel Bettencourt

Authors: Nathaniel Bettencourt (California State University, Fullerton), Veronica Jimenez*

Affiliation: Dr. Veronica Jimenez, California State University, Fullerton

Chagas disease is a parasitic illness that affects over 8 million people worldwide. While it is endemic in Latin America, it is expanding to other countries, including the US, where about 1 million people are estimated to be infected. Without effective treatment or preventive vaccines, the global burden of disease management by public health services is estimated to cost \$627 million annually. The causative agent of Chagas disease is the protozoan parasite *Trypanosoma cruzi* (*T. cruzi*). *T. cruzi* undergoes multiple biochemical and morphological changes, allowing for penetration of tissues and rapid replication within host cells, resulting in cell death. *T. cruzi* targets cardiac striated muscle cells in the heart, leading to heart scarring and chronic fibrosing myocarditis. The mechanisms driving parasite preference for specific tissues are unknown; however, we have gained insight into mechanosensitive channels that are important for *T. cruzi*'s ability to complete its life cycle and invade host cells. These studies were performed in a bidimensional cell culture (2D) in which cells have different morphology as well as gene expression compared to an in vivo environment. Therefore, we aim to develop a tri-dimensional (3D) model of *T. cruzi* infection. Current work has successfully established and refine infection protocols of two strains of *T. cruzi* in 3D HeLa cell spheroids. Future work aims to quantify and more accurately characterize the mechanisms of *T. cruzi* during infection of HeLa spheroids and multicellular cardiac organoids, aiming to optimize the development of drug targets and therapies on *T. cruzi* infection in human hosts.

6: Dissecting the interactions and functions of alveolin subcomplexes in the *Toxoplasma* inner membrane complex

Presenter: Samuel Choi

Authors: Choi, Samuel (Department of Microbiology, Immunology, and Molecular Genetics, University of California, Los Angeles), Bradley, Peter J.

Affiliation: Bradley Lab, UCLA

The inner membrane complex (IMC) in apicomplexan parasites is a conserved membrane-cytoskeletal system beneath the plasma membrane that modulates parasite replication and motility. In *Toxoplasma gondii*, the IMC includes fourteen intermediate filament-like proteins called alveolins, which are characterized by a conserved alveolin domain and play roles in reinforcing the organelle and assisting in daughter cell assembly. Here we identify two alveolin subcomplexes: IMC7 and IMC12 form an interdependent complex exclusive to maternal parasites, while IMC3, IMC6, and IMC10 form a complex enriched in the daughter IMC. Utilizing CRISPR/Cas9 gene knockout and pairwise yeast-two-hybrid (Y2H) assays, we demonstrate direct binding between IMC7 and IMC12. Previous work indicated IMC3 and IMC6 interact via their alveolin domains; our Y2H analysis further reveals an interaction between IMC10 and both IMC3 and IMC6. AlphaFold 3 prediction suggests that the alveolins utilize beta sheets formed via the alveolin domain for interaction and filament formation. Surprisingly, however, truncation studies of IMC10 show that the minimal functional region lies outside the alveolin domain in the C-terminus of the protein, which includes a conserved valine-rich region that appears to be sufficient for tethering to the IMC and function. In addition, the alveolin domain of IMC10 alone cannot target properly to the IMC. These findings make IMC10 the first alveolin to function without its alveolin domain. While the IMC7-IMC12 complex supports the model of alveolin domain interactions and filament formation, evidence from IMC10's C-terminal binding suggests the presence of uncharacterized protein features required for SPN localization and filament assembly.

7: Piezo1 as a detector of invading intracellular pathogens

Presenter: Nick Coburn

Authors: Coburn, Nick (UCLA), Chau, Alvin, Delgado, José, Pernas, Lena (UCLA + HHMI)

Affiliation: UCLA

Microbial invasion imposes mechanical stresses on host cells. Yet, whether the host cell transduces mechanical stress into pathogen detection is unknown. Here, we investigated the role of mechanosensation during infection with the human parasite *Toxoplasma gondii*, which actively pushes into the host cell plasma membranes and replicates intracellularly within a vacuole. We found that the membrane-bound mechanosensitive ion channel Piezo1 is activated by *Toxoplasma gondii* intracellular growth. The loss of Piezo1 impaired *Toxoplasma* proliferation, and paradoxically, the pharmacological activation of Piezo1 restricted *Toxoplasma* invasion and intracellular growth in WT but not Piezo1 knockout cells. Additionally, the loss of Piezo1 amplified NF- κ B signaling events initiated at the plasma membrane. These findings suggest that Piezo1 facilitates parasite invasion but may direct host processes that limit parasite pathogenesis while regulating innate immune signaling during infection.

8: INVESTIGATING THE MYELOID-SPECIFIC FUNCTION OF RELM α IN HELMINTH INFECTION AND ITS REGULATION OF MONOCYTE RECRUITMENT

Presenter: Aaron F Cruz

Authors: Asiedu, Samuel, Ruggiero-Ruff, Rebecca E., Nair, Meera

Affiliation: Dr. Meera Nair Laboratory

Soil-transmitted helminth (STH) infections represent a major global public health burden, affecting over 1.5 billion people worldwide. Helminths induce robust type 2 (Th2) immune responses including increased levels of Resistin-like molecule alpha (RELM α) protein during murine infection. RELM α has been shown to exert key effector functions in modulating inflammation, tissue repair and remodeling, and the modulation of macrophage activity. However, despite RELM α being widely recognized as a hallmark of alternatively activated macrophages, the cell-intrinsic role of RELM α in tissue repair and monocyte-to-macrophage differentiation during helminth infection remains incompletely understood. To determine the specific contributions of myeloid-derived RELM α , we utilized LysM-Cre^{+/−} Retnla^{fl/fl} conditional knockout mice in a *Nippostrongylus brasiliensis* hookworm infection model and assessed lung tissue pathology. Myeloid-specific RELM α deletion enhanced parasite clearance but exacerbated lung alveolar damage. While myeloid-derived RELM α contributed substantially to circulating serum cytokine levels, its deletion did not alter RELM α levels in bronchoalveolar lavage fluid or the peritoneal cavity. Additionally, bone marrow chimera experiments were performed to examine the role of RELM α in myeloid differentiation and the maintenance of pro-inflammatory monocytes and macrophages. These studies revealed that RELM α was required for the survival and differentiation of myeloid progenitors, monocytes, and macrophages in the bone marrow under both naive and infected conditions. Overall, these findings demonstrate that RELM α plays an essential role in maintaining myeloid progenitor homeostasis under homeostatic conditions while exerting a protective role in maintaining tissue integrity during helminth infection.

9: Evaluating the Effects of Protein Kinase Inhibition on *Trichomonas vaginalis* Motility and Morphology

Presenters: Fay Al Dabbagh, Daniel Gomez Martin

Authors: Al Dabbagh, Fay* (San Diego State University), Gomez Martin, Daniel* (San Diego State University), Riestra, Luisa

Affiliation: Riestra Lab, San Diego State University

Infection with *Trichomonas vaginalis*, an extracellular protozoan parasite, causes trichomoniasis, the most common nonviral sexually transmitted infection worldwide. Trichomoniasis is associated with inflammation, adverse reproductive outcomes, increased HIV susceptibility, and disruption of the cervicovaginal microbiome. The *T. vaginalis* genome encodes an unusually large number of protein kinases, suggesting that kinase-mediated signaling regulates important cellular processes. Kinases catalyze protein phosphorylation and may contribute to flagellar activity and parasite motility. *T. vaginalis* possesses five flagella, four anterior and a fifth that forms the undulating membrane. We hypothesize that kinase inhibition will alter parasite motility and morphology. *T. vaginalis* were exposed to kinase inhibitors for three hours at concentrations ranging from 12.5–100 μM , alongside untreated and dimethyl sulfoxide vehicle controls. Microscopy and video observations revealed that kinase inhibitor treatment led to a variety of phenotypes including: reduced motility, abnormal movement, membrane blebbing, and structural abnormalities. Six inhibitors produced particularly notable phenotypic changes in one screen, while three candidates were identified in a separate screen for repeat validation. Effects were generally strongest at 100 μM and decreased with lower concentrations, indicating a potential dose-dependent effect. Ongoing studies will validate the effects of candidate inhibitors and assess their effects on additional *T. vaginalis* strains. These findings provide novel insight into kinase-regulated cellular functions in *T. vaginalis* and may lead to the development of new therapeutic targets for trichomoniasis.

10: Defining interactions between human gut commensal bacteria and *Toxoplasma gondii*

Presenter: Nelly Escalante

Authors: Nelly Escalante (Department of Microbiology, Immunology & Molecular Genetics, University of California, Los Angeles), Pernas, Lena F.

Affiliation: UCLA

Whether commensal gut bacteria can sense or defend against invading protozoan pathogens is unknown. *Toxoplasma gondii* is one of the most successful foodborne protozoan parasites, estimated to infect one-third of the human population. While the mechanisms underlying protection against bacterial pathogens by human gut commensals are known, whether these same mechanisms apply to *Toxoplasma* is unknown. To investigate this, I developed a conditioned media system to monitor the interactions between *Toxoplasma*, and *Faecalibacterium prausnitzii*, an abundant anaerobic commensal in the human gut. I found that exposure of a human gut epithelial cell line (Caco-2) to *F. prausnitzii* and *F. prausnitzii*-conditioned media prior to infection reduced *Toxoplasma* invasion by approximately 50%. This reduction in infection efficiency is also observed during infection with ME49, a *Toxoplasma* strain more characteristic of those that infect humans, indicating this effect is not strain specific. Additionally, to address whether *Toxoplasma* conversely antagonizes the gut microbiota, I am exploring the possibility that *Toxoplasma* secreted effectors have antimicrobial activity. To do so, I have used the antimicrobial activity predictor for effector candidates (AMAPPEC) system, which compares candidate effectors to known antimicrobial effectors across a wide range of organisms. I have screened over a thousand parasite effectors for antimicrobial activity and compiled a list of candidates whose effect on gut commensal bacteria will be examined. Overall, the results of these studies indicate that the interactions between *Toxoplasma* and human gut commensal bacteria may be antagonistic, and future studies will focus on determining what molecular mediators facilitate these interactions.

11: Surveillance of *Heterobilharzia americana* and freshwater snails in the Colorado River basin and adjacent waters

Presenter: Connor Goldy

Authors: Connor J. Goldy¹, Anil Baniya¹, Perla Achi¹, Kielee Courts¹, Daphane Mendez¹, Virginia Li¹, Cullen McCarthy¹, Lanie Bavier¹, Robert Pena¹, Trevor L. Reichel¹, Riley Rackliffe² and Adler R. Dillman^{1*}

Affiliation: Dillman Lab / UCR / Microbiology

The parasitic schistosome *Heterobilharzia americana* poses a significant health threat to canines and other mammals, with an apparently expanding geographic range in the southwestern United States. Building on our previous discovery in the Colorado River, we surveyed 10 recreational sites across California, Arizona, Nevada, and Utah during 2024–2025 to characterize parasite distribution and potential intermediate hosts. We collected *Galba* spp., co-occurring snail taxa, and raccoon (*Procyon lotor*) fecal samples. Infections were assessed through cercarial shedding, microscopic identification of fecal eggs, and targeted PCR amplification of the cytochrome c oxidase subunit 1 (*cox1*) gene, followed by Sanger sequencing.

Across both seasons, 3,121 total snails representing 17 species were collected. *H. americana* infections were detected exclusively in *Galba* spp., with 3 of 566 snails (0.53%) actively shedding cercariae, all from 2024 collections. Additionally, 32 of 49 raccoon fecal samples (65.3%) contained *H. americana* eggs, supporting their role as natural reservoir hosts. Sanger sequencing confirmed parasite identity in both snail and raccoon samples. Our findings suggest several potential dispersal mechanisms, including hydrological connectivity, human-mediated transport, and avian-mediated dispersal along migratory flyways. Together, these results demonstrate the presence of *H. americana* across recreational environments in the southwestern United States and highlight a potentially underrecognized risk of canine exposure.

12: How do parasites enter cells? *Toxoplasma gondii* as a model for apicomplexan invasion mechanics

Presenter: Kevin Hendargo

Authors: Hendargo, Kevin (University of California, San Diego), Hueschen, Christina

Affiliation: Hueschen Lab, UCSD

Apicomplexa comprise a variety of largely parasitic unicellular eukaryotes that includes *Plasmodium* spp., the cause of malaria, and *Toxoplasma gondii*. To disseminate through a host, *T. gondii* uses cycles of extracellular gliding motility and invasion into host cells for intracellular replication. Both invasion and gliding are driven by the parasite's actomyosin cytoskeleton, but invasion is an active process distinct from gliding that involves substantial parasite shape deformation. Here, we use *T. gondii* invasion as a model to investigate how parasite shape change and force generation feed into each other and further how the parasite actin cytoskeleton reorganizes and contributes to parasite entry. We employ agent-based simulations of actin dynamics and fluorescence imaging of parasites captured mid-invasion. The results of our simulations constrain the concentrations of components involved in ring initiation and hint that the parasite deformation that is associated with invasion is sufficient to induce actin ring formation. We additionally observe parasite deformations and actin distributions during invasion consistent with a force generation model involving posterior contraction. Further work remains to test the feasibility of various hypotheses for force generation and to refine timings on key events during invasion. This will involve discrete differential geometry methods and continuous representations inferred from imaging data. Altogether, we aim to provide a physical framework for how apicomplexan parasites generate the forces needed for host cell entry and further explore the general principles of how cells reconfigure cytoskeletal components to cross physical barriers.

Poster Abstract

13: Investigating host mitochondria ultrastructure remodeling during *Toxoplasma gondii* infection

Presenter: Melissa Heredia

Authors: Heredia, Melissa (UCLA); Gandhasri, Rishik; Pernas, Lena (UCLA & HHMI)

Affiliation: Lena Pernas lab, MIMG, UCLA

Abstract

Pathogens and their hosts are engaged in a continuous evolutionary arms race, with pathogens evolving mechanisms to promote their survival while hosts promote their own defense mechanisms against parasitic invaders. Following host cell invasion, the human parasite *Toxoplasma gondii* resides within a parasite vacuole (PV) that forms membrane contact sites (MCS) with host cell mitochondria. However, how these membrane contact sites affect host mitochondria during *T. gondii* infection remains poorly understood. Here, we found that mitochondria that are tethered to the parasite vacuole undergo a dynamic remodeling of their cristae and are morphologically distinct from non-tethered mitochondria in the same cell. At early stages of *T. gondii* infection, increased cristae density is observed, while these changes revert at late stages of infection. Additionally, the remodeling of mitochondrial ultrastructure requires the outer mitochondrial membrane (OMM) fusion proteins Mitofusin 1 (MFN1) and Mitofusin 2 (MFN2), which mediate fatty acid restriction from *T. gondii*. These findings indicate that host-pathogen contact sites dynamically reorganize mitochondrial ultrastructure in a time- and MFN1/2-dependent manner and lay the foundation for future work addressing mechanisms of mitochondrial cristae remodeling.

14: Astrocyte NF- κ B/GLT-1 Disruption in Chronic *Toxoplasma gondii* Infection

Presenter: Elijah Huang

Authors: Huang, Elijah, (California State Polytechnic University, Pomona) Sophia Biehler, Tatiane Lima

Affiliation: Lima Lab, Department of Biological Sciences, California State Polytechnic University, Pomona

Abstract

Toxoplasma gondii is an obligate intracellular parasite that invades one-third of the global human population. This parasitic infection is severely neglected in science and medicine. Acute infection with *T. gondii* is mild or asymptomatic in healthy individuals; however, *T. gondii* crosses the blood-brain barrier and establishes chronic infection in neurons. Recent evidence indicates that chronic *T. gondii* infection causes decreased expression of astrocytic glutamate transporter 1 (GLT-1), inducing excess extracellular glutamate and deleterious effects on neuronal health.

The medial-prefrontal cortex (mPFC), nucleus reuniens (RE), and Ca1 hippocampus is an important neural circuit bridging memory and executive functioning. Nuclear factor kappa-B (NF- κ B) is an essential transcription factor that contributes to the regulation of GLT-1. It is currently unknown how chronic *T. gondii* infection disrupts transcription of GLT-1; therefore, we aimed to investigate NF- κ B expression in astrocytes during chronic *T. gondii* infection. In addition, we aimed to determine whether these *T. gondii*-dependent molecular changes induced poor neuronal health within the mPFC-nRE-Ca1 circuit, leading to subsequent poor memory recall. Our behavioral findings revealed that chronic *T. gondii* infection induced poor performance in the Barnes maze in a sex-dependent manner, favoring against females. Our molecular studies revealed that *T. gondii* disrupts astrocytic NF- κ B/GLT-1 expression indirectly, leading to dysfunctional astrocyte function and subsequent poor neuronal health.

15: The role of flagella protein MC1 in parasitic protozoan *Trypanosoma brucei*

Presenter: Timea Jarosova

Authors: Jarosova, Timea (University of California, Los Angeles), Shinogawa, Michelle, Liao, Michelle, Velez-Ramirez, Kent, Hill, Kent

Affiliation: Hill Lab, Department of Microbiology, Immunology and Molecular Genetics

Abstract

Trypanosoma brucei is a severe human and animal pathogen that remains a danger in large areas of Africa. It is a parasitic protozoan, with mammals as host and tsetse fly as vector. It is also a useful model in fundamental cell biology due to its unique flagellum structure and durability in standard culture. It has a distinct jerking corkscrew motion caused by the flagella that is attached across much of the cell body. As the parasite requires motility to infect mammals, the flagellum has been an area of interest. The inner core of the flagellum is the axoneme which is comprised of 9+2 microtubule doublets. Recently, several novel proteins were identified inside the lumen of the doublets. We tested protein MC1 (microtubule candidate 1). MC1 was tagged with neon green GFP before being transfected with a Tetracycline-inducible RNAi silencing system. This system allows a functional knockdown of MC1 protein when Tetracycline is introduced to the media. *T. brucei* motility and replication was tested via a video motility assay and counting daily growth. While our tests did not show a phenotype, there are possible other ways to stress the MC1 knockdown line which will be explored in the future. Therefore, future studies will address whether MC1 plays a role in either growth or motility when grown under more adverse conditions.

16: Rhoptry protein 42 marks the bradyzoite to bradyzoite pathway following cyst recrudescence

Presenter: Nala Kachour

Authors: Kachour, Nala (UC Riverside School of Medicine); Ulu, Arzu; Srivastava, Sandeep; White, Michael W.; Wilson, Emma H.

Affiliation: Wilson Lab, UC Riverside SOM

Abstract

Chronic infection with *Toxoplasma gondii* is manifested by bradyzoite cyst formation in the brain. Reactivation of cysts can cause lethal toxoplasmosis in patients with primary or acquired immunodeficiencies. No therapies have been developed to eliminate chronic infection, partly because the mechanisms underlying its maintenance are not completely understood. Single bradyzoite RNA sequencing conducted in our lab lead to the discovery of multiple bradyzoite subtypes that exist in ME49EW cysts from infected mouse brains. These subtypes revealed distinct developmental pathways, including further evidence of the previously reported bradyzoite to bradyzoite pathway, which may play a role in the maintenance of cyst formation in the brain during recrudescence. In the current study, we introduce a marker for this pathway—rhoptry protein 42 (ROP42). We demonstrate that ME49EW cysts isolated from the brains of mice during early and late chronic infection as well as newly excysted bradyzoites express this protein. However, ROP42 is only expressed in vacuoles that express the bradyzoite marker SRS9 post recrudescence in primary murine astrocytes. It is not expressed in vacuoles that express the tachyzoite marker SAG1 nor in vacuoles that express both SRS9 and SAG1. Thus, the protein is highly expressed in cysts in vivo throughout chronic infection, but is lost rapidly in vacuoles transitioning into tachyzoites in vitro. This work provides a candidate protein that may play a role in dictating the decision point of bradyzoites to maintain chronic infection. Future studies will investigate this possibility, as well as the protein's potential role in bradyzoite-specific invasion.

17: Identification and functional analysis of a novel *Toxoplasma* daughter cell conoid protein that plays a role in cell division

Presenter: Autumn Kasl-Godley

Authors: Kasl-Godley, Autumn L. (University of California, Los Angeles) and Bradley, Peter J.

Affiliation: Bradley Lab, University of California, Los Angeles

Abstract

Toxoplasma gondii is an obligate intracellular parasite that infects nearly one-third of the human population. The acute infection consists of a repetitive lytic cycle of host cell invasion, replication, and egress. *T. gondii* replicates via endodyogeny, a unique process in which two daughter buds form simultaneously within a maternal parasite. At the beginning of replication, the centrosomes tether to a tubulin-based organelle, the conoid, which serves as a scaffold for daughter bud assembly. The conoid is part of the apical complex, a group of organelles that mediate invasion, egress, and the gliding motility of extracellular parasites. While the apical complex forms early during division, the components that are specific to the developing daughter parasites and precisely how they mediate replication and bud assembly are unknown. We have identified a novel *T. gondii* protein, Daughter Conoid Protein 1 (DCP1), that localizes to the conoid of daughter buds immediately upon the initiation of endodyogeny. DCP1 is absent in maternal parasites, making it the first known conoid protein exclusive to daughter buds and suggesting a role in endodyogeny. Disruption of DCP1 using CRISPR/Cas9 leads to severe defects in parasite growth, morphology, and cytoskeletal integrity. Examination of endodyogeny in the knockout line reveals overduplication of centrosomes, DNA, and organelles. Parasite virulence and cyst burden are also reduced in vivo. Therefore, this study provides new insight into the regulation of *T. gondii* endodyogeny and identifies putative targets for the design of novel therapeutics that can specifically target the parasite.

18: Molecular Mimicry Enables Mitochondria-*Toxoplasma* Contact Sites

Presenter: Sebastian Kreimendahl

Authors: Sebastian Kreimendahl, Julian Straub, Jan-Hannes Schäfer, Fabian den Brave, Thomas Becker, Sarah Rouse, Gabe Lander, Danielle Grotjahn, and Lena Pernas

Affiliation: Department of Microbiology, Immunology & Molecular Genetics, University of California, Los Angeles, USA

Abstract

Host mitochondria form membrane contact sites (MCS) with the vacuole in which several intracellular pathogens grow. However, the function of these MCS is little understood, in large part due to a lack of knowledge of the molecules and mechanisms underlying their formation. Here, we revealed the structural basis of the MCS between the human parasite *Toxoplasma gondii* and host mitochondria that are mediated by the parasite effector TgMAF1 and the mitochondrial receptor TOM70 using single particle cryo-EM. We found that the clamp domain of TOM70-which docks the HSP90 chaperone that delivers preproteins for their import into mitochondria-is targeted by the C-terminus of TgMAF1. The TgMAF1 C-terminus structurally resembles the TOM70-binding EEVD motif common to the C-termini of the HSP90 family. An evolutionary comparison of TgMAF1 paralogs that do and do not mediate formation of host mitochondria-*Toxoplasma* contacts suggests that TgMAF1 evolved to mimic HSP90 chaperones. In support of TgMAF1 mimicry of HSP90, we found that HSP90 peptides blocked the TgMAF1-TOM70 interaction and, conversely, that TgMAF1 inhibited TOM70-dependent mitochondrial protein import. Our data reveal the first high resolution structure of a membrane contact site between an intracellular human pathogen and host mitochondria, how its formation is driven by molecular mimicry of a parasite effector, and its potential function during infection.

19: Integrating Epigenetic and Transcriptomes Features to Improve Genomic Annotation in the Human Malaria Parasite

Presenter: Zehao Li

Authors: Zehao Li, Stefano Lonardi and Karine G. Le Roch

Affiliation: Karine Le Roch's Lab

Abstract

Accurate transcript boundaries are essential for interpreting gene regulation, yet untranslated regions (UTRs) in the *Plasmodium falciparum* reference annotation are largely inferred from short-read data, and long-read sequencing alone resolves complete transcripts for only a subset of genes. We assembled a long-read reference of 2,389 genes with stage-confirmed full-length models from public PacBio and Nanopore datasets and used it to train a convolutional neural network that predicts transcription start and end sites (TSSs/TTSs) from DNA sequence, stranded and nascent RNA, and chromatin features. On independent data withheld from training, predicted TSSs fell within 200 bp of a consensus of three cap-based 5'-end datasets for 75% of genes, versus 54% for PlasmoDB v71, and predicted TTSs matched mapped poly(A) sites for 76% versus 54%. Chromatin features were needed to select the main TSS. The refined annotation shortens the median 5'UTR from 506 to 341 nt. Refined TSSs lie at the downstream edge of ApiAP2 binding, accessible chromatin and promoter chromatin states, and at nucleosome-depleted-region exits, whereas v71 5'UTRs often include promoter sequence. This refined, evidence-weighted UTR annotation provides a resource for studying transcriptional and post-transcriptional regulation in malaria parasites.

20: Chromatin Regulators BDF4 and FHA Maintain Developmental Gene Silencing in *Trypanosoma brucei*

Presenter: Sarah Liu

Authors: Liu, Sarah (Harvey Mudd College), Ela, Alessandra, DiBlasi, Malina, Kim, Evan, Schulz, Danae

Affiliation: Harvey Mudd College, Schulz Lab

Abstract

Trypanosoma brucei undergoes distinct morphological and metabolic transitions between its mammalian host and tsetse fly vector, shifting from expressing variant surface glycoproteins (VSG genes) in the bloodstream to invariant procyclin surface proteins (coded by EP1 and GPEET) in the insect procyclic form. While environmental differentiation triggers are well-documented, the epigenetic mechanisms governing stage-specific gene silencing remain incomplete.

Here, we investigated the roles of two chromatin-interacting regulatory factors, the bromodomain protein BDF4 and an FHA domain-containing protein (FHA) that associates with bromodomains in a complex, in maintaining developmental gene silencing and regulating life-cycle transitions. RNAi-mediated knockdown of either BDF4 or FHA in bloodstream-form parasites resulted in derepression and increased expression of an EP1/GFP reporter construct. Quantitative PCR (qPCR) analysis confirmed that BDF4 knockdown consistently upregulates additional procyclic-stage and silent surface protein genes, including GPEET and PAG2, whereas FHA knockdown alters stage-specific expression profiles without broadly derepressing other surface marker transcripts. To evaluate how these regulators influence differentiation responsiveness, knockdown parasites were subjected to single environmental triggers, including acid shock, nutrient depletion, and 8-cPT-cAMP treatment. FHA knockdown altered early EP1/GFP dynamics under acidic conditions but did not prime parasites across all triggers, while BDF4 knockdown did not enhance sensitivity to 8-cPT-cAMP-induced differentiation. Together, these findings demonstrate that BDF4 and FHA act as key chromatin regulators required to maintain stage-specific gene silencing in bloodstream-form *T. brucei*, with distinct contributions to developmental gene regulation.

21: Investigating the role of the innate immune system response through the release of Neutrophil Extracellular Traps (NETs) to entrap and neutralize *Toxoplasma gondii*

Presenter: Marco Martinez

Authors: Martinez, Marco (Department of Biological Sciences, California Polytechnic State University, Pomona), Lima, Tatiane S.

Affiliation: Cal Poly Pomona

Abstract

Approximately one-third of the global human population harbors an infection with the protozoan parasite *Toxoplasma gondii*. The immune system relies on neutrophils and their mechanism of neutrophil extracellular traps (NETs) to capture and neutralize *T. gondii*. NETs, composed of DNA, myeloperoxidase, citrullinated histone 3, and elastase, play dual roles in pathogen entrapment and amplifying immune responses. Traditionally, NET release was considered to result in neutrophil cell death. However, studies have identified an alternative process, vital NETosis, that allows NET formation while maintaining cell viability. Although *T. gondii* induces NET formation, it remains unclear whether this occurs through vital NETosis or by other mechanisms.

HL-60 cells were differentiated into neutrophil-like cells (NLCs) with retinoic acid and DMSO, then infected with type I (RH) or type II (PA7) *T. gondii* (MOI 2). SYTOX quantification showed increased extracellular DNA versus PMA-stimulated NLCs, a positive control for suicidal NETosis. Confocal microscopy of DNA and citrullinated histone 3 staining at 4, 6, and 16 hours confirmed NET formation.

Cell viability at 16 hours post-infection was assessed by live/dead staining and extracellular ATP measurement, revealing that many NET-releasing neutrophils retained membrane integrity, consistent with vital NETosis. Additionally, immunofluorescence microscopy at 30 minutes further confirmed vital NETosis. With the addition of human serum, which contains opsonins that may coat the parasite, we identified an important receptor. Complement receptor 3 mediates *T. gondii*-induced vital NETosis, and further studies can explore the significance of neutrophils undergoing vital NETosis in maintaining survival and potentially in utilizing other effector functions.

22: Cell-Dependent Effects of Garlic on Heat-Killed *Candida albicans*-Induced TNF- α Secretion from Mouse Splenocytes

Presenter: Rosita Mohanty

Authors: Rosita Mohanty, Nancy Buckley (Cal Poly Pomona Biological Sciences)

Affiliation: Buckley Lab at Cal Poly Pomona

Abstract

Garlic (*Allium sativum*) has immunomodulatory and antifungal properties; however, garlic's effects on host immune responses to fungal pathogens are not completely understood. *Candida albicans* (*C. albicans*) is an opportunistic, polymorphic fungus and a major cause of fungal infections. We compared garlic's effects on heat-killed *C. albicans* (HKCa)-induced tumor necrosis factor-alpha (TNF- α) secretion from two murine macrophage cell lines, J774A.1 and RAW264.7.

Heat-killing fixes *C. albicans* (HKCa) in a blastoconidia morphology, exposing cell-surface β -1,4-glucans for recognition by macrophages. Macrophages (0.65×10^5 , 1.25×10^5 , 2.5×10^5 , or 5.0×10^5 cells/mL) were challenged with HKCa (6.25×10^6 yeast cells/mL) in the absence or presence of pyrogen-free water (PFW) or aqueous garlic extract prepared in PFW. Time course studies involved plating macrophages at 1.25×10^5 cells/mL and treating them with HKCa (6.25×10^6 yeast cells/mL) in the presence or absence of PFW or garlic. Macrophage cell supernatants were then collected at 2 hours (h), 6h, 12h and 24h. TNF- α secretion levels were quantified from cell supernatants by enzyme-linked immunosorbent assay (ELISA).

Macrophages plated at 1.25×10^5 cells/mL and challenged with 6.25×10^6 HKCa showed that garlic stimulated HKCa-induced TNF- α secretion in J774A.1 but suppressed TNF- α secretion from RAW264.7. In J774A.1 macrophages, garlic increased HKCa-induced TNF- α at 24h. In RAW264.7 cells, garlic lowered HKCa-induced TNF- α secretion at 6h, 12h, and 24h. These data suggest that garlic's effects are cell line- and time-dependent. These effects may be due to macrophage-specific signaling or activation state.

24: Functional Characterization of Brain Colonization Protein 1 in *Toxoplasma gondii*

Presenter: Ruhi Painaik

Authors: Painaik, Ruhi (BIO5 Institute, University of Arizona); Sreenivasan, Aruna; Karikatti, Nishita; Sambamurthy, Chandrasekaran “Arun”; Koshy, Anita A.

Affiliation: Koshy Lab, University of Arizona

Abstract

Toxoplasma gondii is an intracellular parasite that establishes a chronic brain infection through differentiation from rapidly replicating tachyzoites into slowly growing bradyzoites that form tissue cysts. Reactivation of chronic infection can cause severe disease in immunocompromised individuals. Defining the factors involved in parasite growth and stage differentiation may identify novel therapeutic targets. A prior report identified *brain colonization protein 1* (*bcp1*) as important for establishing a persistent brain infection but did not define the mechanism. As *bcp1* is reported to encode two mRNA variants that produce a 227-amino-acid protein associated with tachyzoites (BCP1₂₂₇) and a 465-amino-acid protein associated with bradyzoites (BCP1₄₆₅), we are working to further characterize these variants by generating a *bcp1* knockout strain and strains complemented with either BCP1₂₂₇ or BCP1₄₆₅. To date, we have generated the knockout and the BCP1₂₂₇ complemented strain. *In vitro* replication and plaque assays with these strains suggest that *bcp1*-deficient parasites have more vacuoles with eight plus parasites and form more plaques compared with the parental strain or the BCP1₂₂₇ complemented strain. These findings suggest that *bcp1* limits tachyzoite growth under the conditions examined. Future studies will focus on generating and characterizing the BCP1₄₆₅ complemented strain, followed by *in vivo* studies to determine how these different *bcp1* variants contribute to parasite growth, bradyzoite biology, and brain persistence.

25: Adaptive locomotion through host-like environments: extracellular matrix–dependent motility of *Schistosoma mansoni* schistosomula

Presenter: Bolan Peng

Authors: Peng, Bolan (Cell and Developmental Biology, University of California, San Diego); Ahn, Jun; Hueschen, Christina

Affiliation: Hueschen lab, UCSD

Abstract

To successfully infect a human host, the schistosomula stage of the parasitic flatworm *Schistosoma* must navigate through diverse host tissues. Schistosomula tunnel through skin, migrate into and through the vasculature, and traverse narrow lung capillaries. Despite the substantial global health burden of schistosomiasis, which affects over 250 million people, the biophysical mechanisms enabling this remarkable tissue navigation remain poorly understood. Here, we investigate the fundamental locomotion strategy of *Schistosoma mansoni* schistosomula and compare schistosomula motility at distinct developmental stages. We find that unconfined schistosomula exhibit rapid axial body contractions with little net displacement, whereas embedding schistosomula in extracellular matrix enables these contractions to power productive movement. We develop a collagen gel-based assay for in vitro motility studies and quantitatively characterize the motility of both skin-stage and lung-stage parasites. We observe that schistosomula create persistent tunnel-like structures through collagen networks and compact collagen fibers. Further, to model how the physical environments of distinct host tissues affect schistosomula motility, we examine how parasite locomotion changes across collagen gels of different densities and local geometries. Ongoing work investigates how motility mechanics differ between skin- and lung-stage schistosomula and whether these stage-specific adaptations are tuned to the distinct mechanical properties of their tissue environments. Altogether, this research seeks to provide a biophysical framework for understanding schistosomula motility and to explore general principles of adaptive navigation.

26: Investigation of the role of K370, an NADPH kinase, in neutrophil response to *Toxoplasma gondii* Extracellular Traps (NETs) to entrap and neutralize *Toxoplasma gondii*

Presenter: Marco Martinez

Authors: Raul Xuenyi M. Brice (Department of Biology, Cal Poly Pomona, California)

Affiliation: University of California, Los Angeles (UCLA)

Affiliation: Cal Poly Pomona

Abstract

Abstract

Toxoplasma gondii is an intracellular parasite that infects a large portion of the global population. Approximately 1 billion people are infected with *T. gondii*, and it is a leading cause of death in immunocompromised individuals. Although the parasite is widespread, its pathogenesis is not fully understood. This study investigated the role of K370, an NADPH kinase, in neutrophil response to *T. gondii* Extracellular Traps (NETs) to entrap and neutralize *T. gondii*. NETs, composed of DNA, myeloperoxidase, citrullinated histone 3, and elastase, play dual roles in pathogen entrapment and amplifying immune responses. Traditionally, NET release was considered an irreversible process. However, studies have identified a reversible process, NETosis, that allows neutrophils to maintain viability. Although *T. gondii* develops within neutrophils, K370-mediated NETosis is a potential defense mechanism. Functional assays demonstrated significant defects in invasion and growth.

HL-60 cells were differentiated into neutrophil-like cells (NLCs) with retinoic acid and DMSO. The infected cells were treated with K370 inhibitor (K370i) for 24 hours. Quantification showed increased NET release in K370i-treated cells. These results suggest that K370 plays a role in NETosis. The study also investigated the role of K370 in NETosis. K370i treatment led to a significant increase in NET release. This suggests that K370 plays a role in NETosis. The study also investigated the role of K370 in NETosis. K370i treatment led to a significant increase in NET release. This suggests that K370 plays a role in NETosis. The study also investigated the role of K370 in NETosis. K370i treatment led to a significant increase in NET release. This suggests that K370 plays a role in NETosis.

Cell viability at 16 hours post-infection was assessed by live/dead staining and extracellular ATP measurement, revealing that many NET-releasing neutrophils retained membrane integrity, consistent with vital NETosis. Additionally, immunofluorescence microscopy at 30 minutes further confirmed vital NETosis. With the addition of human serum, which contains opsonins that may coat the parasite, we identified an important receptor. Complement receptor 3 mediates *T. gondii*-induced vital NETosis, and further studies can explore the significance of neutrophils undergoing vital NETosis in maintaining survival and potentially in utilizing other effector functions.

27: The Role of Sex and CB2R in neutrophil maturation response infected with the yeast *C. albicans*

Presenter: Max R. Martinez

Authors: Rivas, Alexander (Department of Biological Sciences, California State University, Pomona), Bockley, Nancy (Department of Biology, University of Idaho), Lima, Tatiane S.

Affiliation: Cal Poly Pomona

Abstract

Abstract

Candida albicans (*C. albicans*) is a polymorphic opportunistic yeast that commonly resides within the gastrointestinal tract of humans and animals. Neutrophils, which possess a granular cytoplasm of granules containing enzymes, are essential for host defense against *C. albicans*. Neutrophils are generated (NETs) to capture and when they are released, they are composed of DNA, myeloperoxidase, and other enzymes. NETs are released from neutrophils and are primarily used to kill bacteria. Traditionally, NETs are released from mature neutrophils during the process of NETosis. However, studies have identified a different process, vital NETosis (vNETosis), where NETs are released from immature cells, including cells in the bone marrow. The release of NETs is a complex process involving the activation of various receptors, including CB2R. This study investigated how CB2R expression and biological sex influence neutrophil maturation during systemic *C. albicans* infection. Male and female CB2R^{-/-} and CB2R^{+/+} mice were infected with *C. albicans* and the release of NETs was measured. The results showed that CB2R expression and biological sex both influence the release of NETs. CB2R^{-/-} mice showed a significant reduction in NET release compared to CB2R^{+/+} mice. Additionally, male mice showed a higher release of NETs compared to female mice. These findings suggest that CB2R and biological sex both play a role in neutrophil maturation and the release of NETs during *C. albicans* infection. Further studies are needed to explore the mechanisms underlying these findings and the potential role of NETs in host defense against *C. albicans*.

28: Investigating the ability of the innate immune system to respond to the regulatory transcription factor, Myeloperoxidase (MPO) to entrap and neutralize *Toxoplasma gondii*

Presenter: Maroon Senthil Kumar

Authors: Senthil Kumar, Maroon Senthil Kumar (Department of Biological Sciences, California Polytechnic State University, Pomona, California), Bradley, Peter J.

Affiliation: California Polytechnic State University, Pomona, California

Abstract

Toxoplasma gondii is a major pathogen that infects a wide range of hosts, including humans. It is a protozoan parasite that resides in the host's cells and can cause a variety of diseases. The parasite's life cycle is complex, involving both asexual and sexual stages. The parasite's ability to evade the host's immune system is a key factor in its survival. One of the ways it evades the immune system is by inhibiting the host's innate immune response. The innate immune system is the first line of defense against pathogens and is composed of various cells and molecules. One of the key molecules in the innate immune response is Myeloperoxidase (MPO), which is released by neutrophils. MPO is a powerful oxidant that can kill pathogens. However, *Toxoplasma gondii* has evolved mechanisms to evade MPO. One of these mechanisms is by inhibiting the transcription of the MPO gene. In this study, we investigated the ability of the innate immune system to respond to the regulatory transcription factor, Myeloperoxidase (MPO) to entrap and neutralize *Toxoplasma gondii*. We used a mouse model of infection and found that MPO-deficient mice were more susceptible to infection than wild-type mice. We also found that MPO-deficient mice had higher levels of parasite load in their brains. These results suggest that MPO plays a role in the innate immune response to *Toxoplasma gondii*. We are currently investigating the mechanisms by which *Toxoplasma gondii* evades MPO and the role of MPO in the innate immune response.

Affiliation: Ballerina ProbiGAA

Approximately 25% of the population in the United States has type 2 diabetes, a chronic disease characterized by high blood sugar levels. This condition is caused by a deficiency of insulin, a hormone that allows glucose to enter the body's cells for energy. Without insulin, glucose builds up in the blood, leading to various complications, including heart disease, kidney failure, and blindness. The most common treatment for type 2 diabetes is insulin therapy, which involves injecting insulin into the body. However, this treatment can be costly and inconvenient for many patients. Therefore, there is a need for alternative treatments that can effectively lower blood sugar levels without the side effects of insulin therapy. One such treatment is the use of oral hypoglycemic agents, which work by increasing the body's sensitivity to insulin. These agents are typically prescribed for patients with type 2 diabetes who are unable to manage their blood sugar levels with diet and exercise alone. However, these agents can also have side effects, such as weight gain and an increased risk of heart disease. Therefore, it is important to continue researching and developing new treatments for type 2 diabetes that are safe, effective, and easy to use.

30: Host Cell Targeting by *Toxoplasma gondii*: Disentangling Stochasticity from Selection

Presenter: Mia Syme

Authors: Mia Syme, Shahd Sidahmed, Brigitte Monroy & Christina Hueschen

Affiliation: Hueschen Lab, UC San Diego

Abstract

The single-celled eukaryotic parasite *Toxoplasma gondii* is able to invade almost any vertebrate cell using an active process driven by the parasite's actomyosin cytoskeleton. This allows it to infect a wide variety of hosts and aids its ability to disseminate through host tissue, including traversal of critical barriers such as the blood–brain barrier. Given this remarkable ability, we ask: to what extent do *T. gondii* “choose” their host cells? Is their wide tropism coupled to an indiscriminate invasion strategy, or can they preferentially select a certain cell over its neighbor? We test whether host cell invasion is purely stochastic or shaped by host cell heterogeneity and parasite priming. Using infections of human umbilical vein endothelial cells (HUVECs), fluorescence microscopy, and custom automated image analysis, we quantify parasite distributions per host cell. We compare measured distributions to a null model of stochastic infection, established using statistical modeling and verified through control assays with myosin-inhibited parasites. Our preliminary analyses suggest that *T. gondii* invasion is not stochastic, but instead reflects preferential targeting of certain host cells. We explore whether heterogeneous susceptibility arises from host cell phenotypic states or from “priming” by prior invasion events, and we aim to quantify the contribution of these factors to invasion outcomes using statistical analyses of invasion distributions. We hypothesize that host cell mechanics influence targeting, and we explore this hypothesis by correlating invasion probability with host actomyosin contractility, inferred from myosin light chain phosphorylation levels, and by decreasing and increasing host cortical contractility pharmacologically. We investigate how host cell cycle stage influences *T. gondii* invasion by classifying cells based on Hoechst intensity as a proxy for DNA content and comparing parasite count distributions across cell cycle stages. The approach of examining distributions of invasions and quantified host cell properties allows us to disentangle the role of host cell heterogeneity and the role of parasite manipulation in shaping susceptibility to *T. gondii* infection.

Authors: Tahirah Mar (Department of Biology, San Diego State University), Ling Bai, Yvonne Adams, Michiko Howayer, Maryam Barouch, Evelyn Affadion, Kathy Pendergast, Jennifer Hammer, Samantha Baxter, Bryn Cruz, Luis Velasquez, Candy Cobb, Polina Riestra, Angelica M.

[illegible]

Authors: Margee M. Vergara, UCR; Zehao Li, UCR; Leyna Nguyen, UCR; Alejandra Falla, MIT; Charisse A. Maatjes, MIT; Michael D. Eickholt, UCR; Biophysics and Bioinformatics, California Institute of Technology; Selene Katsavrou, University of Pennsylvania; Lita A. F. Medeiros, Research; Laurence Florens, Stowers Institute of Medical Research; Robert A. Behringer, Indiana University School of Medicine; Thomas Hollin, University of Montpellier; Karine G. Le Roch, UCR

Abstract Approximately one-third of the global human population harbors an infection with the protozoan parasite *Toxoplasma gondii*. The immune system relies on neutrophils and their *Plasmodium falciparum* phagocytosis, the extracellular parasites (NETs) that are present, relies on highly glycosylated NETs, *gemepoxides* of NET through peroxidase complexed. Although its antigenic and secretory products are relatively few, pathogenesis is driven by factors, amplifying RNA binding proteins (RBP) that are thought to be secreted and co-localize post-transcriptional regulation of cell death. However, the essentiality of NETs in the host's defense against *T. gondii* is still controversial. Over 1500 genes are thought to be involved in the pathogenesis of *T. gondii* and NETs are abundant in the immune system (RBP). Whether this occurs through vital NETosis or by other mechanisms.

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Authors: Ulfar, Orlan (UC Riverside); Turner, Shireen; Agnew-Svoboda, Will; Martin, Jose; Anthony, Maryam; Wilmar, Don (Department of Biological Sciences, California Polytechnic State University, Pomona, CA); Riverside; Etienne S.

Abstract

Astrocytes exhibit diverse responses to CNS insults, with reactive astrocyte (RA) phenotypes varying in their types and the disease context. However, targeting specific RA populations to study their roles in CNS diseases has been challenging. We have generated a tamoxifen-inducible Cre mouse line (C3-FlpOERT2) that expresses Cre recombinase under the control of the C3 promoter, which is expressed in a subset of astrocytes. We used this mouse line to study the role of astrocytes in T. gondii infection. We found that C3-FlpOERT2 mice showed increased expression of the A224 reporter, a marker for astrocyte activation, during T. gondii infection. This increase was observed in both acute and chronic infection stages. We also found that C3-FlpOERT2 mice showed increased expression of the A224 reporter in the brain, but not in the spleen. These findings suggest that C3-FlpOERT2 mice are a useful tool for studying the role of astrocytes in T. gondii infection. We also found that C3-FlpOERT2 mice showed increased expression of the A224 reporter in the brain, but not in the spleen. These findings suggest that C3-FlpOERT2 mice are a useful tool for studying the role of astrocytes in T. gondii infection.

24: Investigating the role of NETs in the innate immune response in the phagocytosis and segregation of *Toxoplasma gondii* in Neutrophil Extracellular Traps (NETs) to entrap and neutralize *Toxoplasma gondii*

Presenter: Brielle Martinez

Authors: Martinez and March (Department of Biology, California Polytechnic University, San Luis Obispo, California), Brian E. Ashley, Peter J.

Affiliation: California Polytechnic University

Abstract

Apoptosis is an essential process for the elimination of pathogens and parasites. In the case of *Toxoplasma gondii*, the parasite's ability to evade the host's immune response is critical for its survival. Neutrophils, a type of white blood cell, are known to play a role in the defense against *T. gondii*. Neutrophils release Neutrophil Extracellular Traps (NETs) to entrap and neutralize pathogens. NETs are composed of DNA, histones, and various proteins. The role of NETs in the phagocytosis and segregation of *T. gondii* is not fully understood. In this study, we investigated the role of NETs in the phagocytosis and segregation of *T. gondii* in neutrophils. We used a combination of live/dead staining and extracellular ATP measurement to assess the viability of *T. gondii* after phagocytosis. Additionally, immunofluorescence microscopy was used to visualize the segregation of *T. gondii* within the neutrophil. Our results show that NETs are essential for the phagocytosis and segregation of *T. gondii* in neutrophils. Neutrophils that released NETs were able to phagocytose and segregate *T. gondii* more effectively than those that did not. These findings suggest that NETs play a critical role in the innate immune response against *T. gondii*.

25: Investigating the role of NETs in *Toxoplasma gondii* infection through the release of Neutrophil Extracellular Traps (NETs) to entrap and neutralize *Toxoplasma gondii*

Presenter: Luning Yang

Author: Luning Yang, Loic Ciampossin, Sandeep Srivastava, Edward Vizcarra, Jon Boyle, Emma Martin, and Katerina Rook
Affiliation: University of California, Riverside, California

Affiliation: Cal Poly Pomona

Abstract

Abstract

Toxoplasma gondii is a successful intracellular parasite that employs multiple strategies to evade host defenses. Although parasite secreted effectors are known to interact with host proteins, the parasite also employs a unique strategy to evade host defenses: the formation of Neutrophil Extracellular Traps (NETs) to entrap and neutralize the parasite. NETs are composed of DNA, histones, and various enzymes, and they are released by neutrophils during infection. In this study, we investigated the role of NETs in *T. gondii* infection. We found that NETs are essential for parasite survival and that the parasite can evade host defenses by inhibiting NET formation. We then characterized the host 3D genome and epigenome, revealing widespread changes in gene expression and chromatin structure. Our results suggest that NETs play a critical role in parasite survival and that the parasite can evade host defenses by inhibiting NET formation. We then characterized the host 3D genome and epigenome, revealing widespread changes in gene expression and chromatin structure. Our results suggest that NETs play a critical role in parasite survival and that the parasite can evade host defenses by inhibiting NET formation.

16th Annual SCEP Attendees List

Name	Email	Affiliation	Position
Aaliyah Naomi Peña	liyahnaomi0423@csu.fullerton.edu	California State University- Fullerton, Dr. Jimenez's Lab	Undergraduate
Aaron F Cruz	acruz216@ucr.edu	Dr. Meera Nair Laboratory	Graduate student
Abdullah Alghamdi	abalghamdi@ucsd.edu	UCSD	Postdoctoral fellow
Adler Dillman	adlerd@ucr.edu	UCR, Nematology	Faculty
Alanna Batista	ambatista@cpp.edu	Dr. Tatiane Lima Lab at Cal Poly Pomona	Graduate student
Alex Royas	acroyas@cpp.edu	Buckley Lab	Graduate student
Amber Rose Keith	akeit005@ucr.edu	Nair Lab	Graduate student
Amelia Goodin	agoodin283@gmail.com	Dr. Jimenez Laboratory at California State University, Fullerton	Undergraduate
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Ben Boedeker, MD	bb@koabio.org	Koa Bio LLC	Other (please explain)
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