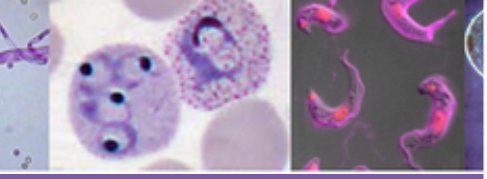

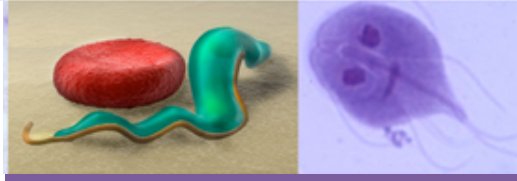

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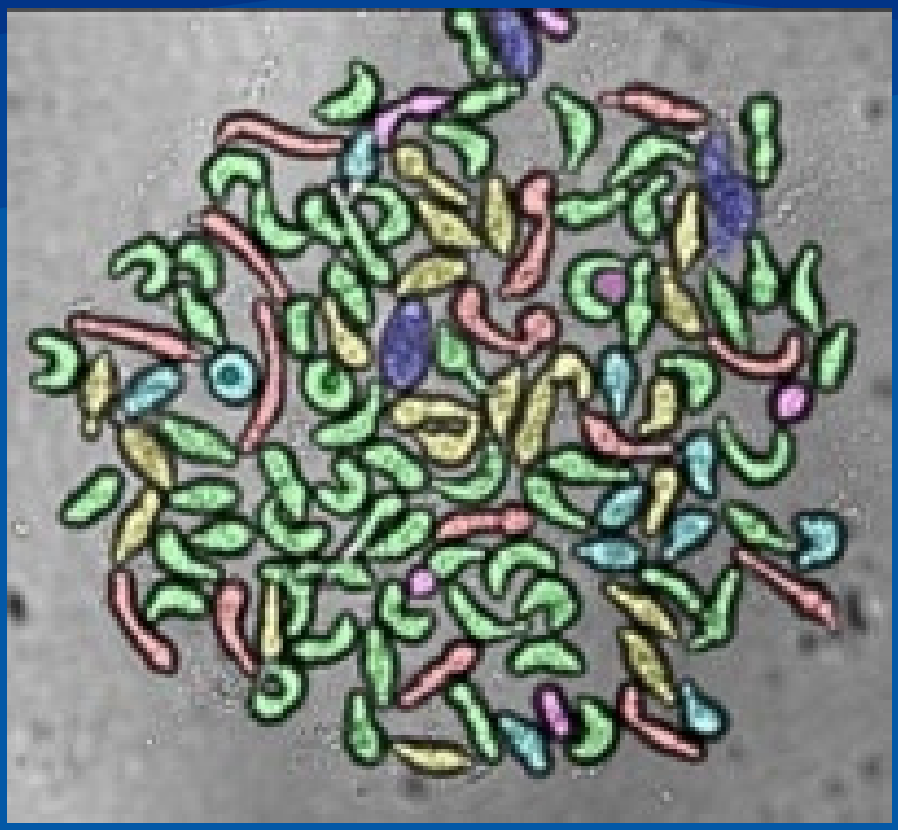
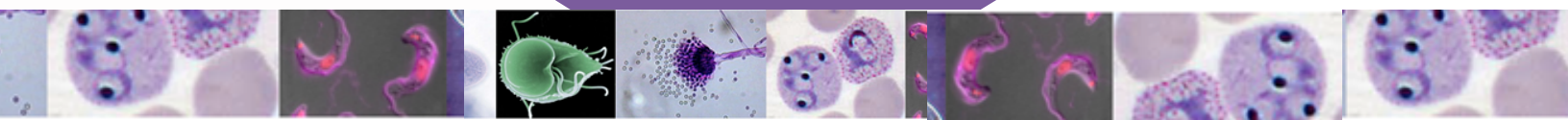
SOUTHERN CALIFORNIA EUKARYOTIC PATHOGEN SYMPOSIUM

UC RIVERSIDE

11th Annual Southern California Eukaryotic Pathogen Symposium (SCEP)



Wednesday, October 27, 2021



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

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- Graduate Student Association
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Cover Image
Nelly El-Sakkary, Postdoctoral Fellow
University of California San Diego
Developed by the lab of Dr. Conor Caffrey at UC San Diego with Steven Chen from Molecular Devices, LLC.

High-Content Coloring in a Flatworm Parasite "JellyBeans"
Schistosomula "somules" in a flatbottom well at 10x magnification in brightfield. Using INCarta High-Content imaging tools, worms were "colored" based on morphometric features: green = "normal"; yellow = "dark"; purple = "round"; long = "red.

2021 SCEP Welcome

Welcome to the 11th 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 20 different labs representing 4 different UC campuses, several Cal State Colleges and many visitors from out of state. All are investigating the inner workings and host response to these important pathogens. It is the aim of this symposium to bring together these like-minded but individual groups to facilitate interaction and collaboration and to enjoy some great science.

Unfortunately, due to a UCR request to keep large gatherings to a minimum, we changed from an in-person format to online. We are all a little gutted but hope that in 2022 we can all come back together again and enjoy chats, drinks and nibbles.

This year's keynote speaker is **Jane Carlton** the Silver Professor and Professor of Biology at New York University. Dr. Carlton received her PhD from the University of Edinburgh in Parasite Genetics and has studied the genome of several of our favorite eukaryotic protozoa including publishing the genomes of *Plasmodium falciparum* and Plasmodium yoelii in 2002. Her comparative approach has led to several major findings on *Plasmodium*, *Babesia* and *Cryptosporidium*. Today she will be telling us all about the wonderful qualities of *Trichomonas*! We are delighted to welcome Dr. Carlton to SCEP and are only sorry that this year it is not in person.

As always, we are democratic here at SCEP and encourage everyone to vote for your favorite talk. You will have that opportunity through a zoom poll at the end of the symposium. Congratulations to Nelly El Sakkary for the submission of the winning image in the cover competition titled "High-Content Coloring in a Flatworm" or as we are affectionately terming it "JellyBeans". Thank you very much as always to **Pica Preston** for her wonderful coordination of this symposium that has taken extra work during these online times.

It is amazing that this is the 11th symposium! 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 for all. We hope to see you in person very soon.

Karine Le Roch, Meera Nair and Emma Wilson

2021 SCEP Organizing Committee

2021 SCEP Keynote

Keynote Speaker: Jane Carlton, Ph.D.

Silver Professor; Professor of Biology & Global Public Health, New York

"The trickiness of Trichomonas genomes"

Professor Jane Carlton, also known as "Genome Jane" on Twitter, is the Julius Silver, Roslyn S. Silver, and Enid Silver Winslow Professor of Biology, and of Epidemiology, and Faculty Director of Genomics at the Center for Genomics & Systems Biology, New York University.

She received her PhD in Genetics at the University of Edinburgh, Scotland, and has worked at several scientific institutions in the United States, including NCBI/GenBank at the National Institutes of Health, and The Institute for Genomic Research (TIGR) founded by J. Craig Venter.

Professor Carlton is passionate about genomics and its power to revolutionize the study of parasites. Her research involves using comparative genomics to interrogate the biology and evolution of the malaria parasite, and trichomonads that infect humans, wildlife, and domesticated animals. For the past 15 years she has worked with researchers, clinicians, and public health practitioners in India as Director of an International Center of Excellence in Malaria Research funded by the NIH. She has published more than 150 research articles, been profiled by CNN, BBC, and The Economist, and received awards from the American Society for Parasitologists and the American Association for the Advancement of Science.

For more information, please visit: https://janecarltonlab.org/

UC RIVERSIDE

11TH ANNUAL SCEP SYMPOSIUM

WEDNESDAY OCTOBER 27, 2021
9:00AM-4:00PM

AGENDA

Welcome to the 11th Annual Southern California Eukaryotic Pathogen Symposium

9:00 -9:15 am - Welcome to 11 years of SCEP

Opening remarks for the 11th Annual SCEP Symposium

Morning session: Chair, Dr. Emma Wilson

9:15 -9:30 am - Angeline Wijono (UCLA , Hill Lab)

Identification of Lineage-Specific Microtubule Inner Proteins (MIPs) in Trypanosoma brucei

9:30 -9:45 am - Melvin Williams (CSUF, Jimenez lab)

Mitochondrial function is regulated by mechanosensitive channels in Trypanosoma brucei

9:45 -10:00 am - Lucy Paddock (HMC, Schulz Lab)

Bromodomain proteins show increased occupancy at binding sites as African trypanosomes differentiate from bloodstream to procyclic forms

10:00 -10:15 am - Todd Lenz (UCR, Le Roch Lab)

Chromatin structure and var2csa – a tango in regulation of var gene expression in the human malaria parasite Plasmodium falciparum?

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IITH ANNUAL SCEP SYMPOSIUM

Lightning talks (10:15-10:30am)

10:15 -10:18 am - Akshara Kannan (CSUF, Jimenez lab)

Regulation of the contractile vacuole function and flagellar homeostasis by mechanosensation

10:18 -10:21 am - Izra Abbaali (UCI, Morrisette lab)

Analysis Pipeline for Candidate Apicomplexan Parasite-Selective Microtubule-Targeting Agents

10:21 -10:24 am - Skylar Rains (UCR SOM)

There's a Fungus Afoot! A Case Study of Disseminated Coccidioidomycosis

10:24 -10:27 am - Michelle Shimogawa (UCLA , Hill Lab)

Developing a microfluidic-based chemotaxis assay for Trypanosoma brucei

10:27 -10:30 am - Stephanie Orchanian (UCI, Lodoen lab)

Defining the Molecular Basis of Immune Cell Recruitment to the Central Nervous System

10:30 - 10:45 am - BREAK

10:45 -11:00 am - Nelly ElSakkary (UCSD, CaffreyLab)

High-Content (HC) Imaging and Gene Expression Profiling (GEP) in a Flatworm Parasite

11:00 -11:15 am - Ivan Chavez (UCLA, Hallem Lab)

Parasitic nematodes exhibit life stage-specific interactions with host-associated and environmental bacteria

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IITH ANNUAL SCEP SYMPOSIUM

11:15 -11:30 am - Suhani Bhakta (Cal Poly, Mercer lab)

Determining how protozoan parasite Trichomonas vaginalis is degraded and digested during trogocytosis

11:30 -11:45 am - William Maciejowski (U of A, Decks lab)

Evolution of Membrane Trafficking Machinery in Tritrichomonas foetus

11:45 -12:00 pm - BREAK and ready for Keynote!!!

12:00 -1:00 pm - Keynote lecturer: Professor Jane Carlton, PhD. (New York University)

The trickiness of Trichomonas genomes

1:00 -2:30 pm - LUNCH. Poster viewing and interactions via SLACK

Afternoon session: Chair, Dr. Meera Nair

2:30 - 2:45 pm - Sarah Bobardt (UCR, Nair lab)

Endocannabinoid Receptor-Signaling Regulates Host-Parasitic Nematode Interactions

2:45 -3:00 pm - Kristina Bergersen (UCR, Wilson lab)

Chronic Brain Neutrophils Protect Against Toxoplasma gondii Infection

3:00 -3:15 pm - Peter Back (UCLA, Bradley Lab)

An essential IMC protein complx governs Toxasma invasion and egress

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IITH ANNUAL SCEP SYMPOSIUM

3:15 - 3:30 pm - Stephanie Matsuno (UCI, Lodoen lab)

The inflammatory role of caspase-8 during T. gondii infection of human monocytes

3:30 - 3:45 pm - Chandrasekaran "Arun" Sambamurthy (U of A, Koshy lab)

Using Toxoplasma gondii to redefine the neuronal innate immune response

3:45 - 4:00pm - Awards and Thank You's!!!

SEE YOU ALL NEXT YEAR!

UCR College of Natural and Agricultural Sciences
Center for Infectious Disease and Vector Research

Molecular, Cell and Systems Biology

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Talk abstract #1

Identification of Lineage-Specific Microtubule Inner Proteins (MIPs) in Trypanosoma brucei

ANGELINE WIJONO[a], Michelle Shimogawa[a], Hui Wang[e], Jihui Sha[d], James Wohlschlegel[d],Z. Hong Zhou[a,b], and Kent Hill[a,b,c]

[a]Department of Microbiology, Immunology and Molecular Genetics, University of California, Los Angeles, CA, 90095, USA.

[b]California NanoSystems Institute (CNSI), University of California, Los Angeles, CA, 90095, USA.

[c]Molecular Biology Institute, University of California, Los Angeles, CA, 90095, USA.

[d]Department of Biological Chemistry, University of California, Los Angeles, CA, 90095, USA.

[e]Department of Bioengineering, University of California, Los Angeles, CA, 90095, USA.

Flagella are microtubule-based organelles that are essential for motility of many eukaryotic parasites, including *Trypanosoma brucei*. *T. brucei* has a unique corkscrew motility and a vigorously beating flagellum with specialized requirements for maintaining structural integrity. In recent years, it has been found that the inside of microtubules is not hollow, but contains proteins called microtubule inner proteins (MIPs) that assemble to form an inner sheath on the microtubule lattice. Previous studies in other organisms have shown that MIPs help maintain microtubule integrity during flagellar beating. *T. brucei* has some MIPs that are conserved and some MIP structures that are lineage-specific. However, the identities of proteins that comprise these lineage-specific MIPs are unknown. We hypothesize lineage-specific MIPs contribute to flagellum stability and unique motility of trypanosomes. To identify lineage-specific MIPs, we examined flagellum composition and structure in *T. brucei* cells following knockdown of FAP106, a conserved MIP. Loss of FAP106 results in shortened flagella, suggesting that FAP106 helps in flagellum assembly and/or stabilization. Moreover, using cryo-electron tomography we found that some MIP structures are missing in the FAP106 mutant. Using quantitative proteomics to compare flagellum protein composition of WT and FAP106 KD cells, we identified ~2500 proteins that were unaffected and seven that were reduced at least two-fold in the FAP106 mutant. Proteins reduced in the mutant are FAP106, two conserved MIPs known to contact FAP106, and four novel trypanosome-specific MIP candidates. Further study will focus on defining which MIP structures these proteins correspond to, how they interact with other flagellum structures, and their role in maintaining flagellum integrity and motility. Given their absence in the human host, lineage-specific MIPs represent potential targets for antiparasitic drugs.

Talk abstract #2

Mitochondrial function is regulated by mechanosensitive channels in Trypanosoma brucei

MELVIN WILLIAMS1, Elissa Moller2, Tiffine Pham1, Sergei Sukharev2 and Veronica Jimenez1

1Center for Applied Biotechnology Studies and Department of Biological Science

2College of Natural Sciences and Mathematics

3California State University, Fullerton, CA.

4Department of Biology, University of Maryland, College Park, MD.

The lifecycle of Trypanosoma brucei involves adapting to various mechanical challenges as it travels through both its insect vector the Tsetse fly and its mammalian hosts. These challenges include changing osmotic conditions, shear stress, and extravasation to host tissues. It is currently unknown, how T. brucei can sense these mechanical stimuli and respond to them. We have identified a mechanosensitive channel (TbrMscS) that shares homology to mechanosensitive channels of small conductance found in E. coli. Expression in spheroplasts followed by electrophysiological recordings indicate that TbrMscS is directly activated by tension and has a conductance of 0.2 pS. When expressed in mechano-deficient E. coli strains, TbrMscS complemented the function of these mutants and restored their ability to regulate volume, protecting them from osmotic shock. These results confirm TbrMscS is a bona fide mechanosensitive channel that plays a role in volume regulation. In the parasites, the channel is expressed in both life stages, with higher levels in procyclic forms, and localizes to the mitochondria, as expected by its genetic homology with bacterial-like channels. When downregulated through RNAi the absence of the channel caused a decrease in cell growth and reduced social motility behavior. Additionally, TbrMscS downregulation caused dissipation of the mitochondrial membrane potential resulting in a depolarization of the mitochondria. Our results provide the first evidence of mechanosensation in T. brucei and the roles it plays in mitochondrial homeostasis.

Talk abstract #3

Bromodomain proteins show increased occupancy at binding sites as African trypanosomes differentiate from bloodstream to procyclic forms.

LUCY PADDOCK (1), Ethan Ashby (2), Hannah Betts (1), Anya Porter (1), Jo Hardin (3), Danae Schulz (1)

(1) Harvey Mudd College

(2) University of Washington

(3) Pomona College

Our lab is interested in how Trypanosoma brucei, the causative agent of Human and Animal African Trypanosomiasis, adapts to the differing environments of its mammalian host and fly vector. We and others have shown that chromatin interacting bromodomain proteins localize to transcription start sites in bloodstream forms. However, it's unclear if localization of bromodomain proteins changes as parasites differentiate from bloodstream to procyclic forms. To address this question, we performed Cleavage Under Target and Release Using Nuclease (CUT&RUN) timecourse experiments using a tagged version of Bromodomain Protein 3 (Bdf3) in parasites differentiating from bloodstream to procyclic forms. We found that the majority of Bdf3 peaks show increased protein occupancy at 3 hours following onset of differentiation. A number of peaks with increased bromodomain protein occupancy lie proximal to genes known to have altered transcript levels during differentiation, such as procyclins, procyclin associated genes, and invariant surface glycoproteins. While most peaks are observed throughout differentiation, a very small number appear as differentiation progresses. One such peak lies proximal to the procyclin genes. Overall, these studies will help shed light on whether transcriptional changes that occur during differentiation might in part depend on occupancy of bromodomain proteins found proximal to the corresponding genes. Additionally, the optimization of CUT&RUN for use in T. brucei may prove helpful for other researchers as an alternative to ChIP-seq.

Talk abstract #4

Chromatin structure and var2csa – a tango in regulation of var gene expression in the human malaria parasite Plasmodium falciparum?

TODD LENZ[1], Xu Zhang[2], Mohit Gupta[1], Kirk Deitsch[2], and Karine Le Roch[1]

1- Department of Molecular, Cell and Systems Biology, University of California Riverside, 900 University Avenue, Riverside, CA 92521, USA

2 Department of Microbiology and Immunology, Weill Medical College of Cornell University, New York, NY.

Over the last decades, novel methods have been developed to study how chromosome positioning within the nucleus may play a role in gene regulation. Adaptation of these methods in the human malaria parasite, Plasmodium falciparum, has recently led to the discovery that the three-dimensional structure of chromatin within the nucleus may be critical in controlling expression of virulence genes (var genes). During asexual, mitotic replication within erythrocytes, extensive non-allelic recombination events in regions containing var genes produce chimeric genes, helping to explain one method the parasite utilizes to avoid the immune response. To further understand how recombination occurs within var gene regions while the rest of the genome remains stable, var2csa transcription was disrupted using targeted DNA double-strand breaks (DSBs) within the sub-telomeric region of chromosome 12, resulting in a cascade of recombination events involving the sub-telomeric regions of other chromosomes and the production of chimeric var genes. To characterize the changes in chromatin architecture stemming from homologous recombination and how that could affect var gene expression, we used Hi-C to pinpoint chromatin structural modifications. CRISPR-cas9 cell lines were used to generate a Hi-C library. We also developed a novel computational pipeline to identify regions within the genome containing significant changes in both intra-chromosomal and inter-chromosomal interactions. We observed a net gain of interactions in all sub-telomeric regions and internal var gene regions, indicating a gain of the tightly controlled heterochromatin structures. Our results suggest that disruption of var2csa results not only in changes in var gene transcriptional regulation but also a significant increase of heterochromatin clusters thereby disrupting coordinated activation and silencing of var genes throughout the genome. Altogether our result confirms a strong link between chromatin structure and gene expression.

Poster abstract and lighting talk #1

Regulation of the contractile vacuole function and flagellar homeostasis by mechanosensation.

AKSHARA KANNAN1, Ingrid Augusto 2, Kildare Miranda 2, and Veronica Jimenez 1

1. Department of Biological Science, College of Natural Sciences and Mathematics, California State University, Fullerton.

2. Instituto de Biofísica Carlos Chagas Filho, Federal University of Rio de Janeiro.

Mechanosensation is an evolutionary conserved phenomenon found in all organisms. Mechanosensitive channels (MSc) are transmembrane proteins that sense mechanical forces and transduce them into biochemical or electrical signals. In bacteria, MSc are associated with traits that regulate virulence, surface detection, and biofilm formation. In plants, these channels play a crucial role in organelle formation and calcium homeostasis. However, in protozoan parasites, the role of MSc is not completely understood.

In Trypanosoma cruzi, the protozoan parasite that causes Chagas disease, we have identified a mechanosensitive channel (TcMscS) that shares structural and functional features with small conductance mechanosensitive channel (MscS) found in E. coli.

TcMscS is localized to the contractile vacuole complex of the parasite. CRISPR-cas9 mediated gene knock out of TcMscS impaired the parasites' growth, osmotic regulation, and infectivity. Global transcriptomic analysis by RNA seq shows the differential expression of several genes with flagellar and motility-related genes significantly downregulated in the TcMscS KO.

We verified the expression at protein level and found that PFR2, FCaBP and other key flagellar components are reduced. Additionally, the parasites showed abnormal morphology, shorter flagella and the presence of multiple basal bodies. This is consistent with motility and infectivity defects observed in the TcMscS-KOs. Trafficking experiments with brefeldin A (BFA) on revealed a decreased labeling of FCaBP, mimicking the phenotype observed in TcMscS KO suggesting a role for the mechanosesitive channel in the trafficking of proteins between the contractile vacuole and the flagellar pocket. Our results reveal new mechanisms of organellar crosstalk that could be essential for parasites' survival and infectivity.

Poster abstract and lightning talk #2

Analysis Pipeline for Candidate Apicomplexan Parasite-Selective Microtubule-Targeting Agents

IZRA ABBAALI, Truong, D., Day, S.D., and Morrissette, N.

Department of Molecular Biology and Biochemistry, University of California, Irvine, Irvine, CA

Toxoplasma gondii is a human protozoan parasite that causes one of the most common food-borne parasitic infections. Approximately one third of the world is affected by the resultant chronic infection. This parasite grows and replicates intracellularly, thereby causing extensive tissue damage and cell death. Those with weak immune systems, including developing fetuses and immunocompromised individuals, are particularly vulnerable and can develop hydrocephaly, blindness, or life-threatening encephalitis. Standard treatment for toxoplasmosis is a combination therapy of pyrimethamine and sulfadiazine, which synergistically block parasite folate metabolism. However, pyrimethamine is teratogenic and sulfa drugs cause hepatotoxicity. Though there are other anti-parasitic drugs that can be used as alternative therapies, they are also poorly tolerated. As such, there is an urgent need for safer and more effective anti-toxoplasmosis treatments. This work is complicated because both the parasite and the human host are eukaryotes, and thus have many of the same essential proteins. Successful anti-parasitic drugs must target the parasite with minimal toxicity to the host cells. Our previous work indicates that protozoan tubulin can be selectively disrupted by small molecules to inhibit parasite growth. During this analysis, we developed an optimized pipeline of assays that delineate effects of candidate drugs on Toxoplasma gondii growth and host cell viability. These two-pronged analyses quantify efficacy, selectivity, and specificity of candidate compounds against Toxoplasma gondii. This methodology will allow us to standardize a testing cascade, thereby making compound profiles comparable. We have since used this pipeline to analyze the activity of clemastine, an obsolete antihistamine that has been shown to inhibit Plasmodium growth by competitively binding to the TRiC/CCT tubulin chaperonin. To demonstrate the broad applicability of this pipeline, we are concurrently analyzing oryzalin, a known parasite-selective tubulin-targeting agent, parabulin, a rationally designed parasite-selective colchicine site inhibitor, and astemizole, a distinct antihistamine that inhibits multiple stages Plasmodium replication and is postulated to target heme detoxification. Our data indicates that clemastine and astemizole have EC50 values ~ 2 μM despite having different proposed molecular targets. Analysis of these drugs illustrates our standardized testing pipeline and unbiased quantification to identify compounds with selective activity.

Poster abstract and lightning talk #3

There's a Fungus Afoot: A Case Study of Disseminated Coccidioidomycosis

SKYLAR RAINS[1], Brandon Nathaniel, MD.2,

1 University of California, Riverside School of Medicine, Riverside

2 Riverside University Health System, Riverside

Coccidioidomycosis (also known as Valley Fever) is a fungal infection endemic to the Southwestern United States caused by the inhalation of spores of Coccidioides immitis. Sixty percent of infections are asymptomatic and in the remaining cases of symptomatic infection, coccidioidomycosis presents with mainly pulmonary symptoms. In extremely rare cases, disseminated infection to distant tissues can occur through hematogenous spread, however, these infections are usually only seen in the immunocompromised population. A 32-year-old African American man from Southern California with no past medical history presented to the hospital with a 3-month history of progressive swelling and pain in his left foot. On admission, the patient was found to have a mass at the head of the left 4th metatarsal concerning for malignancy, with infectious causes and osteomyelitis lower on the differential diagnosis. The patient had no clinically significant leukocytosis, the only notable laboratory findings included elevated inflammatory measures of erythrocyte sedimentation rate (ESR) and C-reactive protein. On imaging studies, an MRI of the foot showed an enhancing mass with bony destruction of the head of the fourth metatarsal, most concerning for malignancy and a CT scan of the chest revealed a 1.18 cm ill-defined nodule on the right lower lobe. These findings, along with the patient's presentation, pointed towards a potential diagnosis of Giant Cell tumor of bone. A CT guided biopsy with fungal cultures positive for Coccidioides immitis, making a final diagnosis of disseminated coccidioidomycosis and ruling out malignant etiologies. The patient was started on oral antifungal medications and a fourth ray total amputation is scheduled. This case illustrates an extremely rare phenomenon of fungal osteomyelitis from disseminated coccidioidomycosis affecting a healthy young patient with no past medical history. It highlights the need for clinicians to consider rarer causes of osteomyelitis in patients with atypical presentations especially in endemic regions and to obtain appropriate fungal cultures in the diagnostic workup of osteomyelitis.

Poster abstract and lightning talk #4

Developing a microfluidic-based chemotaxis assay for Trypanosoma brucei

MICHELLE SHIMOGAWA1, Bijie Bai2, Yuzhu Li2, Hatice Ceylan Koydemir2, Aydogan Ozcan2, and Kent Hill1,3

1Department of Microbiology, Immunology and Molecular Genetics

2Electrical and Computer Engineering Department

3Molecular Biology Institute

African trypanosomes (T. brucei and related species) cause devastating human and animal disease and limit economic development in some of the world's most impoverished regions. To become infectious to mammals, these unicellular parasites must navigate through a series of diverse tissue environments within the tsetse fly vector. The chemotactic cues that drive these directional migrations are still unknown. Social motility assays have previously demonstrated the ability of T. brucei communities to undergo both negative and positive chemotaxis, however these assays are not very amenable to high-throughput screening. We are working to develop a liquid-based chemotaxis assay that will enable quantitative analysis of chemotaxis over short time scales and allow us to measure parameters of individual cell motility that may be correlated with the chemotactic response to a stimulus. Recent work from Isabel Roditi's lab has implicated pH as a chemotactic cue that directs parasite movements during social motility on semi-solid agarose plates. In ongoing experiments, we are attempting to recapitulate pH-dependent chemotaxis of insect-stage parasites in a liquid-based assay and determine whether there are changes in individual cell motility that are correlated with this response.

Poster abstract and lightning talk #5

Defining the Molecular Basis of Immune Cell Recruitment to the Central Nervous System

STEPHANIE B. ORCHANIAN1, Katherine M. Still2, Tajie H. Harris2, Melissa B. Lodoen1

1University of California, Irvine, 2University of Virginia

Toxoplasma gondii is an obligate intracellular parasite and a leading cause of death attributed to foodborne illness. T. gondii is a unique pathogen due to its ability to invade across the blood-brain barrier and persist for the remainder of the host's lifespan. CCR2+ monocytes are required for host survival in both acute and chronic infection. We have found that these inflammatory immune cells are recruited to the meninges within 7 days and to the brain within 15 days post-infection. Using CCL2 reporter mice, our data indicate that CCL2, a key chemokine for CCR2+ monocytes, is produced initially by meningeal macrophages and microglia during acute infection, and subsequently by astrocytes during chronic infection. Intriguingly, in GFAP-Cre x CCL2 fl/fl mice, in which astrocytes are deficient in CCL2 expression, monocyte and T cell recruitment to the brain are reduced during the chronic stage of infection, and parasite cyst burden is increased. This study analyzed the effects of infection on monocyte infiltration of the meninges and the brain and identified microglia and astrocyte CCL2 as a possible driver for monocyte and T cell recruitment and host protection in the CNS.

Talk abstract #5

High-Content (HC) Imaging and Gene Expression Profiling (GEP) in a Flatworm Parasite

NELLY EL-SAKKARY1, Uli Sun1, Vanessa Fu1, Ali Syed1, Supacha Denprasertsuk1, Rafay Syed1, Steven Chen2, Hayley Bennett3, Anthony O'Donoghue1, Conor R. Caffrey1

1Center for Discovery and Innovation in Parasitic Diseases, University of California San Diego, 9500 Gilman Dr., La Jolla, CA 92093, USA;

2Molecular Devices, LLC. 3860 N First Street, San Jose, CA 95134;

3Genentech, 1 DNA Way, South San Francisco, CA 94080

Schistosoma mansoni causes schistosomiasis, a neglected tropical disease which infects over 200 million people worldwide. Treatment of the disease is primarily with praziquantel (PZQ). With widespread use of the drug in mass drug administration and the lack of an available alternative, there are concerns that resistance will develop. It is therefore important to develop novel drugs against the parasite. We use high-content (HC) imaging to identify changes in worm phenotype and identify drug groups of interest. One of these drug groups is the proteasome inhibitors.

Larval parasites are distributed into wells at ~50 worms per well in 96-well, clear, round-bottom plates. An ImageXpress Micro XLS plate reader was used to obtain brightfield images at 10x magnification. Worms were segmented and morphometric parameters, were identified using an iterative machine learning SINAP module in INCarta (Molecular Devices, LLC). Morphometric features identified included: darkness, roundness, length and opacity.

A drug group we identified with efficacy against schistosomes includes the proteasome inhibitors. Further, we've validated the proteasome as a drug target in the parasite. The proteasome is important for protein degradation and parasite survival, though it has not been much studied as a drug target. The proteasome has been validated as a drug target in other parasites including malaria, trypanosoma and leishmania. In S. mansoni, in vitro, exposing schistosomes to short-interfering RNAs in culture targeting the proteasome reduces parasite viability by ~80%.

Exposing parasites to proteasome inhibitors carfilzomib (CFZ) and bortezomib (BTZ) causes reduced motility by more than 85% after 24 h and is associated with an increase in caspase 7 activity (apoptosis). Proteasome inhibitors (PIs) are currently used in myeloma treatment, and these inhibitors have potential for drug repurposing to design schistosomiasis treatments. In vivo studies are associated with a 96% reduction in parasite burden.

While the proteasome is a proven drug target in schistosomes, the gene expression profile (GEP) associated with effects on parasite viability and associated phenotypic effects are not known. We perform GEP to provide the basis wherein the chemical and genetic "profile" of these PI effects on the parasite can be integrated and analyzed such that emerging patterns can inform the discovery of markers of drug efficacy. Using this approach, we exposed parasites to PIs in culture at 1 μM of BTZ, CFZ, or MG132, followed by RNA-Sequencing (RNA-Seq). We use RNA-Seq to identify the mechanism of action of PIs, identify markers of drug efficacy, and identify other drug targets.

Talk abstract #6

Parasitic nematodes exhibit life stage-specific interactions with host-associated and environmental bacteria

IVAN N. CHAVEZ[1], Taylor M. Brown[1], Adrien Assié[2,3], Astra S. Bryant[1], Buck S. Samuel[2,3], and Elissa A. Hallem[1]

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Skin-penetrating nematodes of the genus Strongyloides infect over 600 million people, posing a major global health burden. Their life cycle includes both a parasitic and free-living generation. During the parasitic generation, infective third-stage larvae (iL3s) actively engage in host seeking. During the free-living generation, the nematodes develop and reproduce on host feces. At different points of their life cycle, Strongyloides species encounter bacteria from various ecological niches. However, the microbial interactions between Strongyloides and bacteria remain uncharacterized. We first investigated the microbiome of the human parasite Strongyloides stercoralis using 16S-based amplicon sequencing. We found that S. stercoralis free-living adults have a distinct microbiome, suggesting that they selectively associate with specific fecal bacteria. We then investigated the behavioral responses of S. stercoralis and the closely related rat parasite Strongyloides ratti to an ecologically diverse panel of bacteria. We found that S. stercoralis and S. ratti showed similar responses to bacteria. The responses of both nematodes to bacteria varied dramatically across life stages: free-living adults were strongly attracted to most of the bacteria tested, while iL3s were attracted specifically to soil bacteria. The behavioral responses to bacteria were dynamic, consisting of distinct short- and long-term behaviors. Finally, a comparison of the growth and reproduction of S. stercoralis free-living adults on different bacteria revealed that the bacterium Proteus mirabilis inhibits S. stercoralis egg hatching, greatly decreasing parasite viability. Our results identify bacteria that serve as key sensory cues for directing movement, as well as bacteria that decrease the parasite's reproductive fitness.

Talk abstract #7

Determining how protozoan parasite Trichomonas vaginalis is degraded and digested during trogocytosis

SUHANI BHAKTA and Frances Mercer

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Trichomonas vaginalis (Tv) is a flagellated, extracellular protozoan parasite that infects the urogenital tract and is transmitted sexually resulting in the sexually transmitted infection trichomoniasis. There are roughly 276 million new cases of trichomoniasis each year, however, this number may be greater due to undetected asymptomatic cases. Common symptoms of trichomoniasis include but are not limited to discharge, pain during urination, and inflammation of the prostate and cervix. The inflammatory response to Tv is attributed to the immune response, specifically neutrophils, which are the most abundant immune cell types present at the site of infection. Neutrophils kill Tv in a contact and dose-dependent manner, wherein multiple neutrophils swarm and surround one trichomonad and remove “bites” of the Tv plasma membrane in a process known as trogocytosis. The mechanisms involved in neutrophil trogocytosis of Tv have not been well established, thus we aim to elucidate the subcellular events involved in the degradation and digestion of Tv material. Neutrophil serine proteases (NSPs) have been shown to be essential for the trogocytosis of Tv, as the killing was reduced in the presence of serine protease inhibitor. Many NSPs are found within the granules of neutrophils, thus we aim to knockout stxbp2, a gene that plays a role in extracellular degranulation, to determine whether NSPs within granules play a role in the degradation of Tv during trogocytosis. Here, we show that we used CRISPR Cas9 technique to construct stxbp2 knockouts within neutrophil like cells (NLCs) —myeloid progenitor cells that have been differentiated and serve as a suitable model to test neutrophil degranulation and trogocytosis. Next, we will pit stxbp2 knockout NLCs against Tv to determine whether neutrophil granules are required for killing and trogocytosis of the parasite. Furthermore, we investigated how “bites” of Tv are digested following trogocytosis. The protozoan Entamoeba histolytica kills live epithelial cells using trogocytosis, a process that was shown to be dependent on lysosomes, since inhibition of lysosomes resulted in reduced trogocytosis. We therefore hypothesize that neutrophils trogocytose Tv and digest ingested material using lysosomes. To test this, we used two inhibitors, ammonium chloride (NH_4Cl)—a weak base, and concanamycin—V-ATPase inhibitor to prevent lysosome acidification in NLCs. Killing assays, and preliminary trogocytosis assays between NLCs and Tv demonstrate no significant difference in parasite killing or trogocytosis in the presence of lysosome inhibitors, indicating that unlike amebic trogocytosis, neutrophil trogocytosis may not require lysosomal digestion of bites in order to kill a target.

Talk abstract #8

Evolution of Membrane Trafficking Machinery in Tritrichomonas foetus

WILLIAM MACIEJOWSKI (1), Joel Dacks (1)

The Parabasalia is a eukaryotic lineage largely composed of parasitic protists with heavy medical, social, and economic implications. Trichomonas vaginalis, the causative agent of Trichomoniasis, affects 270 million people, making it the most prevalent non-viral STD in the world. This condition increases the risk of other sexually transmitted conditions such as HIV and may cause issues with pregnancy. Tritrichomonas foetus is responsible for Bovine Trichomoniasis, which causes infertility and early fetal death within cattle herds.The membrane trafficking system plays a critical role in the parasitic nature of Trichomonas and has been greatly expanded, even compared with humans. Resolving the complement of encoded proteins and the details of their evolution in globally relevant parasites, such as T. vaginalis and T. foetus, solidifies the understanding of their pathogenic qualities and broadens our scope for future treatments.

Adaptins are a family of proteins responsible for vesicle formation and cargo selection within the membrane trafficking system. Throughout evolutionary history adaptin complexes have been relatively well conserved, making them a good target for homology searching. In 2007, the adaptin complement of T. vaginalis was reported to be vastly expanded, with 73 individual protein coding genes. Humans, in comparison, only have 24.

We began by searching for adaptin complexes and associated genes in T. vaginalis to both verify the previous findings, and create a dataset of queries for further homology searching in other parabasalid lineages. Subsequently, we used these data to perform the first analysis of this membrane trafficking system in T. foetus. Analyses were undertaken using homology searching (BLASTP) and phylogenetics (RAxML, MRBAYES).

We report an expanded adaptin complement of 64 protein coding genes in T. foetus. Most duplications appear to be unique to the individual parasite, though one pre-dates its divergence from T. vaginalis. These species represent the deepest split in parabasalids, therefore this represents an ancient gene duplication. Overall, this is the first analysis of adaptins in this important veterinary parasite and gives us a glimpse into the membrane trafficking system of this important parasitic lineage.

Talk abstract #9

Endocannabinoid Receptor-Signaling Regulates Host-Parasitic Nematode Interactions

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Many helminth infections have a mandatory lung stage that can cause severe damage to host tissue due to parasite migration. The endocannabinoid system has been shown to have anti-inflammatory effects in the gut, but the role of this system in the lungs has not been well-characterized in the context of helminth infection. In our previous work, we demonstrated that loss of the endocannabinoid receptor CB1R results in increased eosinophilia of the lungs of mice in response to infection with the parasite N. brasiliensis (Nb), a model of hookworm. Here we show that macrophages isolated from the lungs of secondary infected mice lacking CB1R show an increase in Relmα, a marker of type 2 immunity, to these parasites. Utilizing an ex vivo approach, macrophages from mice lacking CB1R had increased levels of Relmα, a marker for the Type 2 Immune Response. Similarly, both macrophages and eosinophils sorted with beads from the lungs of secondary infected mice showed an increased affinity for live and dead worms in a coculture. This demonstrates a role for the endocannabinoid system in mediating host-pathogen interactions in the lungs and is the premise for the use of cannabis as a treatment to ameliorate the inflammatory immune response to helminth infection.

Talk abstract #10

Chronic Brain Neutrophils Protect Against Toxoplasma gondii Infection

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Infection with the protozoan parasite Toxoplasma gondii leads to the formation of lifelong cysts in neurons of the brain that can have devastating consequences in the immunocompromised. However, despite the establishment of a chronic inflammatory state and infection-induced neurological changes in the brain, the majority of neurons show no overt signs of clinical pathology resulting in an asymptomatic infection in the immunocompetent. This suggests the work of neuroprotective mechanisms to prevent clinical manifestations of infection, but sources of neuroprotection during infection remain mostly unknown. Previous work in other models of central nervous system (CNS) injury and infection demonstrates neuroprotection by CNS-resident and peripheral immune cells via the expression of neuroprotective molecules. Targeted gene expression analysis during chronic Toxoplasma infection has also previously demonstrated upregulation of neuroprotective genes and repair pathways.

We have identified a population of chronic neutrophils in the brain during Toxoplasma infection that expresses neuroprotective molecules such as NRG1, ErbB4, and MSR1. Further phenotyping of this chronic neutrophil population via flow cytometry and singe-cell RNA sequencing reveals two distinct subsets of neutrophils that display functional heterogeneity. Functional analyses of these cells demonstrate cyst-specific responses, and depletion of neutrophils in the brain suggests this population is required for control of the parasite. Collectively, these results demonstrate a novel neutrophil population that may play a dynamic role in controlling chronic infection and inflammation in the CNS by balancing classical responses with pro-resolution functions.

Talk abstract #11

An essential IMC protein complex governs Toxoplasma invasion and egress

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Toxoplasma gondii is an opportunistic intracellular pathogen that uses specialized organelles to invade their host cells and ultimately cause disease. One organelle is the inner membrane complex (IMC), a membrane system composed of flattened vesicles that underlie the plasma membrane, coupled to a cytoskeletal meshwork of intermediate filament-like proteins. IMC proteins localize to three subcompartments – the base, the body, and the apical cap. Recently, we demonstrated that the apical cap plays a critical role in forming the apical complex, a group of cytoskeletal structures that functions in regulated secretion and parasite motility. Specifically, the conditional depletion of any of the three essential apical cap proteins AC9, AC10 and ERK7 eliminates the apical complex, blocks organelle secretion, and completely inhibits invasion and egress. AC9 accomplishes this by recruiting the MAP kinase ERK7 to the apical cap and regulating its kinase activity. However, how the three elements interact together and are organized in the apical cap remains unknown. In this study, we use a combination of deletion analyses and yeast-2-hybrid experiments to reveal multiple domains in AC9 and AC10 that are necessary for assembling the complex at the apical cap and for the maturation of the conoid. Importantly, we demonstrate that these domains mediate independent pairwise interactions between AC9, AC10, and ERK7, suggesting that this protein complex is organized by multivalent interactions. Thus, we propose that these multimeric interactions drive the oligomerization of the AC9:AC10:ERK7 complex into the apical cap cytoskeleton, providing a platform for ERK7 to coordinate the proper biogenesis of the apical complex.

Talk abstract #12

The inflammatory role of caspase-8 during T. gondii infection of human monocytes

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Toxoplasma gondii is a food-borne obligate intracellular parasite that infects one-third of the global human population. Innate immune cells, such as monocytes, are among the first cells recruited to sites of infection and produce the potent proinflammatory cytokine IL-1β. We previously showed that T. gondii-infected primary human monocytes produce IL-1β through a Syk-PKC-δ-CARD9-MALT1-NF-κB signaling pathway, and IL-1β release requires the NLRP3 inflammasome and caspase-1 activity. We recently investigated the roles of other caspases in IL-1β release by knocking out caspase-1, -4, -5, or -8 in THP-1 cells using CRISPR/Cas-9 genome editing. Genetic ablation of caspase-1 or -8, but not caspase-4 or caspase-5, decreased IL-1β release during T. gondii infection. Furthermore, dual pharmacological inhibition of caspase-8 and RIPK1 in primary human peripheral blood monocytes decreased IL-1β release without affecting cell viability or infection efficiency. Caspase-8 was not required for the production or cleavage of IL-1β but rather, caspase-8 inhibition leads to the retention of mature IL-1β within the cells. In investigating caspase-8 processing, we found that infection with type II T. gondii, which induces IL-1β release, leads to cleavage of caspase-8 from full-length protein to the p30 subunit. In contrast, type I infection, which does not induce IL-1β release, did not trigger caspase-8 cleavage. Our data suggests that during type II T. gondii infection of human monocytes, caspase-8 functions in a novel mechanism of controlling IL-1β release from viable cells. This study expands on the molecular mechanisms of IL-1β release from human immune cells and on the inflammatory role of caspase-8 in host defense.

Talk abstract #13

Using Toxoplasma gondii to redefine the neuronal innate immune response

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Toxoplasma gondii naturally causes a lifelong, asymptomatic central nervous system (CNS) infection in immunocompetent humans and rodents. This tropism for and persistence in the CNS underlies T. gondii's ability to cause devastating neurologic disease, and even death, in the immunocompromised. Despite being able to invade any nucleated cell in vitro, T. gondii persists primarily in neurons in vivo. Prior in vitro work suggested that neurons are persistently infected because, unlike other CNS cells, they lack the ability to clear intracellular parasites even in the setting of IFN-γ stimulation. Using Cre-expressing parasites (RopCre) that allow us to permanently mark and track CNS cells injected with T. gondii effector proteins, we found that up to 95% of the T. gondii-injected neurons are not actively infected, suggesting that cytokine-stimulated neurons may clear intracellular parasites. Given these conflicting in vitro and in vivo results, we used primary murine neuronal cultures from wild-type and genetically modified mice in combination with cytokine stimulation and parental and transgenic parasites, including a new T. gondii-Cre line (GraCre), to reassess the ability of neurons to clear intracellular parasites in the setting of IFN-γ stimulation. These data reveal that neurons respond to IFN-γ— including up-regulating STAT1 and the GTPases known to be involved in IFN-γ dependent clearance of T. gondii in non-neuronal murine cells— and that a subset of neurons (~20%) clear intracellular parasites via these GTPases. In addition, in Cre reporter mice infected with the GraCre parasites that mark CNS cells only after full invasion, whole neuron reconstructions showed that ~40% of these T. gondii-invaded neurons no longer harbor parasites. Finally, IFN-γ stimulation of human stem cell derived neurons led to an ~50% decrease in parasite infection rate when compared to unstimulated, infected cultures. Collectively, these data highly suggest that IFN-γ stimulation leads to parasite resistance in murine and human neurons and that a subset of murine neurons clear intracellular parasites both in vitro and in vivo, likely via the previously identified GTPases.

Poster Abstract #1

Determining PMN Surface Receptors and Subcellular Events Involved in PMN Trogocytosis of Trichomonas vaginalis

ASHLEY RAMIREZ, Emely A.P. Giron and Frances Mercer - Cal Poly Pomona

Trichomonas vaginalis (Tv) is a flagellated unicellular parasite responsible for causing the highly prevalent and most common non-viral sexually transmitted infection (STI) worldwide, trichomoniasis. Clinical symptoms associated with trichomoniasis include severe inflammation at the site of infection and premature rupture of the placental membranes in pregnant women. It is known that Tv binds to and induces cytolysis of human mucosal epithelial cells. However, the immune response to the parasite remains poorly characterized. It has been demonstrated that neutrophils (PMN) are critical in the killing of Tv and use a contact-dependent mechanism called trogocytosis. In this process, PMN surround the parasite and take “bites” preceding parasite death; however, the specific molecular players in the trogocytic killing process are unknown. PMNs are known to exhibit several opsonin receptors on their cell surface that bind to serum components of the adaptive immune system, such as antibodies. One focus of our study, is to determine if PMN cell surface opsonin receptor, FcYRIIa (CD32a), a constitutively expressed IgG receptor, is required for PMN trogocytosis of Tv. Additionally, we are determining the fate of these trogocytic bites of the parasite within the PMN by asking whether trogocytosed parasite material colocalizes to PMN lysosomes. Here, we show that CD32a is expressed on a neutrophil-like-cell (NLC) cell line model of PMN used to study trogocytosis of Tv, and that we have successfully designed a strategy to knockout CD32a using the CRISPR/Cas9 homology-directed repair method. We also show that we have obtained clonal populations of NLCs with a stop or silent mutation encoded at the CD32a locus. Additionally, we show preliminary results of lysosomal marker (LAMP-1) staining on neutrophils in the presence of Tv. The conclusions of our study will help to uncover the subcellular and molecular mechanism that immune cells use to kill this highly prevalent parasite.

Poster Abstract #2

Assembly of RNAi Construct for Genetic Screen Verification in Trypanosoma brucei

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Trypanosoma brucei is a parasite endemic to regions of Sub-Saharan Africa that infects humans and cattle with sleeping sickness, causing significant health and economic burdens. The parasites exist in a mammalian bloodstream stage, then differentiate to a procyclic stage within the tsetse fly vector. Bloodstream-stage trypanosomes express variant surface glycoproteins (VSGs) that help the parasite evade the mammalian adaptive immune system, while procyclic parasites express non-variant surface procyclin proteins. Although there are known environmental triggers and signal transduction mechanisms associated with the downregulation of VSGs and the upregulation of procyclin, the factors responsible for increasing transcript levels of procyclin are unknown. We will attempt to identify these factors via a genetic screen with an overexpression plasmid library. Each plasmid contains a single T. brucei gene, and plasmids are transfected in bulk into an EP1/GFP reporter cell line, which produces GFP as procyclin transcripts increase. Increased GFP expression in the reporter line might indicate that the plasmid contained a gene important for procyclin expression. Given the high-throughput nature of the screen, verification methods are needed to confirm that candidate genes are true positives. We modified an RNAi plasmid backbone that can inducibly knock down candidate genes. When the reporter parasites transfected with the RNAi plasmid are differentiated to their procyclic form, we would expect to see decreased GFP expression for genes that truly induce procyclins. We tested our RNAi construct with tagged RBP21 and showed inducible knockdown via Western blot.

Poster abstract #3

cAMP signaling during the intracellular infection cycle of Trypanosoma cruzi

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Trypanosoma cruzi is a protozoan parasite and causative agent of Chagas disease, a vector-borne disease historically restricted to Latin America. Nowadays, global warming is causing vector's distribution to expand to northern territories in Mexico, reaching southern parts of US, in which up to 300,000 people are infected. Chagas disease manifests clinically as cardiovascular disease, characterized by heart hypertrophy. A major cause of heart pathology in Chagas disease damage is caused by the host immune system, as it attacks chronically infected tissue. Therefore, pathology of the disease is a direct consequence of the ability of the parasite to invade host cells, establishing a chronic infection. To achieve this, T. cruzi must sense and adapt to the host environment, but the underlying mechanisms are poorly understood. In particular, parasite signaling pathways used to sense and transduce signals from the host environment are most completely unknown. We are interested in studying the role of cAMP during the intracellular infection cycle of T. cruzi. Several lines of evidence suggest T. cruzi cAMP signaling is important for host cell invasion, as well as parasite differentiation and replication, which in turn underlies heart tissue pathology of Chagas disease. A previous transcriptome analysis identified that mRNAs of proteins involved in cAMP metabolism, i.e. receptor-like adenylate cyclases (AC) and phosphodiesterases (PDE), are either upregulated or downregulated during the intracellular infection cycle. Homology modeling of the extracellular domain of an AC, upregulated during the host cell invasion, revealed that could bind leucine. Homology modeling of the intracellular (catalytic) domain of another AC, upregulated during the host cell invasion, revealed that catalytic domains could form homodimers in vivo. The homologue of a PDE, upregulated during the parasite differentiation inside the infected cell, has a flagellar localization in a related trypanosome. Suggesting that in T. cruzi, the flagellum could also be a signaling platform. Sequence alignment of the catalytic domain of a PDE, downregulated during the intracellular replication, revealed T. cruzi-specific residues that could have a regulatory role. Altogether, our bioinformatics analyses indicate that cAMP has an important role in T. cruzi intracellular invasion cycle.

Poster abstract #4

Combinatorial nanotherapy reduces adipocyte-derived inflammation during Trypanosoma cruzi infection

1 DEBORA SCARIOT, Austeja Steneviciute, Evan Scott 2 David Engman 1

Trypanosoma cruzi is the causative agent of Chagas disease (CD), a neglected disease endemic to vast regions of the Americas from the United States to Argentina. CD is rarely diagnosed in the acute stage since infection is usually asymptomatic. T. cruzi is an obligate intracellular parasite that can infect any nucleated cell, particularly cardiomyocytes. In chronic infection, which is typically lifelong, T. cruzi remains dormant in long-lived cells, not only myocytes but also adipocytes, causing silent tissue damage and inflammation, and low-level parasite production for decades following infection. As a consequence, about 30% of infected people develop chronic chagasic myocarditis. Only two drugs – Benznidazole (BNZ) and Nifurtimox (NFX) – are available to treat acute CD but the limited efficacy and high toxicity of these drugs in the chronic stage are both significant problems. Additionally, BNZ and NFX are not able to prevent heart failure in those with chronic chagasic myocarditis, although they may reduce progression to myocarditis in those without chronic heart involvement. Our previous study showed that BNZ-loaded poly(ethylene glycol)-b-poly(propylene sulfide) nanocarriers (PEG-b-PPS NCs) reduce acute heart parasitosis using a 466-lower dose than free BNZ without causing typical BNZ toxic effects, such as hepatotoxicity and weight loss. Additionally, previous findings showed that vitamin D-loaded PEG-b-PPS NCs can mitigate chronic inflammation in obese mice via induction of regulatory T cells. Now, we hypothesize that a combinatorial nanotherapeutic strategy of BNZ and vitamin D (VD) can address both the T. cruzi parasitemia in reservoir cells and the inflammation associated with chronic CD. To check the potential trypanocidal and anti-inflammatory effects of BNZ-loaded and VD-loaded PEG-b-PPS NCs, 3T3-L1 fibroblasts were chemically differentiated into adipocytes, infected with T. cruzi for 24 h, and then treated with BNZ-NCs, VD-NCs, BNZ-NCs + VD-NCs, blank unloaded NCs and free-form BNZ for 48 h. The number of infected cells and intracellular parasites was counted and the levels of cytokines in the supernatant were quantified by flow cytometry. Parasite growth inhibition after BNZ-NC, VD-NC and BNZ-NC + VD-NC treatments were similar, suggesting that the parasiticidal effect of BNZ is not affected by the anti-inflammatory action of VD. However, only VD-NCs and BNZ-NCs + VD-NCs treatments promoted a significant reduction of IL-6 levels. In adipose tissue, IL-6 is a major inflammatory mediator associated with high systemic inflammation in chronic inflammatory diseases, autoimmune diseases, and cancer. Our preliminary results suggest that BNZ-NC+VD-NC combination nanotherapy is a potential strategy to treat chronic T. cruzi infection and systemic inflammation simultaneously. The impact of this combination will be evaluated in a murine model of chronic CD.

Poster abstract #5

Group 1 metabotropic glutamate receptor's influence on T cells in T. gondii infection

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Toxoplasma gondii (T. gondii) is one of the most effective transmissible pathogens in the world, infecting approximately two billion people. Encystment of the parasite in neurons in the brain results in a lifelong chronic infection. Within the brain, a pro-inflammatory response is essential to prevent parasite reactivation. Infection in the immunocompromised leads to lethal Toxoplasmic encephalitis while in the immunocompetent, there is persistent low-grade inflammation which lacks clinical symptoms. This suggests that there is a tightly regulated inflammatory response to T. gondii in the brain. T cells are the dominant immune cell that control recrudescence and parasite replication through secretion of effector molecules such as perforin and IFNγ. However, the regulation of these cells in this tissue is poorly understood. During chronic infection there is an increase in extracellular (EC) glutamate that is normally tightly controlled in the brain. High extracellular glutamate is not specific to T. gondii infection and can occur during multiple pathologies but may be an important environmental signal to tissue specific immune cells. We hypothesize that this glutamate-rich environment plays a role in T cell function and regulation.

Here we demonstrate that T cells from the T. gondii -infected brain express the G-protein coupled metabotropic glutamate receptors (mGluR's) mGluR1 and mGlur5. This expression is enriched in T cells recruited to the brain compared to secondary lymphoid derived cells. Furthermore, expression can be determined by T cell phenotype. We further hypothesize that T cells recruited to the brain are regulated by glutamate through mGluR modulation. Using activators and inhibitors of these receptors we will test glutamate dependent signaling mechanisms that are implicated in T cell function and regulation in response to T. gondii in the chronically infected brain. Understanding the effect of exogenous glutamate on immune cells in the brain is a critical avenue to explore as changes in glutamate concentrations may disrupt the balanced inflammatory response to T. gondii and may have implications for glutamate-lowing therapies for neurodegenerative disease.

Poster abstract #6

Extracellular Vesicles Derived from Toxoplasma gondii Infection Contribute to Intracellular Communication

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Infection with the obligate intracellular parasite Toxoplasma gondii leads to an increase in T cells in the brain and Toxoplasmosis in immunocompromised patients. During infection astrocytic glutamate regulation is disrupted and extracellular levels of glutamate in the central nervous system (CNS) reach non-homeostatic ranges. Astrocytes regulate CNS glutamate by adjusting uptake, release and synthesis into glutamine. Through previous work in the lab we have found that following T. gondii infection there is a decrease in astrocytic glutamate transporter, GLT-1, leading to an increase in the amount of extracellular glutamate, which can lead to neurotoxicity and seizures. GLT-1 can be regulated by exosomes secreted by neurons. Exosomes are extracellular vesicles containing proteins, lipids, mRNA, and miRNAs. Exosomes are derived from the fusion of multivesicular bodies with the plasma membrane and extracellular release of the intraluminal vesicles. They are known to function in intercellular communication and modulate host-parasite interactions. Exosomes are produced by any cell, including T. gondii. To test if Toxoplasma induces changes in extracellular vesicles (EVs) production, the number of exosomes was measured from infected and uninfected cells. In addition, purified EVs were added to fibroblasts and host cell response was measured. Our preliminary results suggest that Toxoplasma infection induces changes in EV production from host cells that can alter the inflammatory response.

Poster abstract #7

The role of complement receptors in mediating the cell-to-cell contact between neutrophil like cells and Trichomonas vaginalis

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Trichomonas vaginalis (Tv) is an extracellular parasite that is responsible for ~276 million cases of trichomoniasis worldwide. Trichomoniasis is the most common non-viral sexually transmitted infection, yet despite this high rate of infection, it is considered a neglected disease due to its lack of research. Symptoms of trichomoniasis can include cervicitis, prostatitis, and vaginal/penile discharge. Many of these symptoms could be a result of the innate immune system's response to Tv. Neutrophils are one of the major players during acute inflammation and are recruited to the site of infection. Neutrophils "nibble" Tv in a novel contact dependent mechanism called trogocytosis to kill the parasite. While it is known that neutrophils surround the parasite and trogocytose it until it is dead, it is not known how the initial contact is made between neutrophils and Tv, to initiate trogocytosis. Since primary neutrophils have a short life span, we will differentiate PLB-985 human myeloid cell lines into neutrophil like-cells (NLCs) that are still able to trogocytose Tv, in order to test our hypothesis. For trogocytosis to occur, we hypothesize that the parasite must be opsonized, or "made tasty," so that neutrophils can establish contact with the parasite. Previous studies have shown that complement protein iC3b, an opsonin of the innate immune system, coats Tv, suggesting that iC3b is necessary for the contact dependent interaction between the parasite and neutrophils. Since complement receptors 1, 3, and 4 are expressed on neutrophils and can bind to iC3b, we hypothesize that these receptors play a role in initializing the contact between NLCs and Tv for trogocytosis. To test this hypothesis, we will individually knock out CD35 (CR1), CD11b (CR3), and CD11c (CR4), using the CRISPR/Cas9 RNP- homology directed repair method. Here, we show that complement is required for efficient neutrophil killing of T. vaginalis, that our NLC cell model expresses CR1, CR3 and CR4, and that we have successfully targeted all 3 iC3b receptors for knockout. Future experiments include clonal dilution of our knockout populations and testing their phenotype in parasite killing and trogocytosis assays. The results from this study can help determine which receptor(s) play a role in this novel contact-dependent method of killing T. vaginalis. A more complete understanding of the molecular mechanisms of immunity to trichomoniasis can hopefully be used to create new treatment plans or even preventative treatment options for trichomoniasis.

Poster abstract #8

Role of neutrophil FcγRI and FcγRIII against Trichomonas vaginalis

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Trichomonas vaginalis is the causative agent for the most common nonviral sexually transmitted infection called trichomoniasis. Although most infections are asymptomatic, acutely symptomatic incidence is higher in women. Persistent trichomoniasis is associated with various complications such as increased HIV susceptibility, low infant birth weight, and increased risk for developing cervical cancer. In vitro studies identified neutrophils as the primary immune cells recruited to the site of infection and combat *T. vaginalis*. Neutrophils surround the *T. vaginalis* parasite and utilize a novel process called trogocytosis to take small pieces off *T. vaginalis*' cell membrane until the parasite dies. However, the molecular mechanisms that facilitate neutrophil contact, trogocytosis, and cytotoxicity of *T. vaginalis* parasites remain unknown. In this presentation, we report our research progress involving two neutrophil surface receptors, FcγRI and FcγRIII, and serum antibodies that may potentially be involved in bridging the contact between neutrophils and *T. vaginalis*. Using an in vitro cell line model of neutrophils derived from either HL-60 or PLB-985 cells, we identified only FcγRI, not FcγRIII, to be present on the cell surface of terminally differentiated neutrophil-like cells (NLCs). We determined human IgG1 and IgG2 as serum components that can bind to *T. vaginalis* surface. We are currently conducting experiments to determine whether human IgG3 and IgG4 can also coat *T. vaginalis*. In addition, future experiments will examine the role of FcγRI in trogocytic killing by testing NLCs deficient in FcγRI against *T. vaginalis*.

Poster abstract #9

Verification of Hits of Genetic Screen for EP1 Expression in Trypanosoma Brucei

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Our lab is interested in gene expression changes that allow adaptation of the parasite that causes African sleeping sickness in mammals, Trypanosoma brucei, to its mammalian and tsetse fly host. Once it enters the tsetse fly, the parasite sheds the varying protein coat it employs to evade the mammalian immune system and instead expresses an invariant procyclin coat. We are piloting a high-throughput overexpression screen to identify genes that increase the expression of the procyclin gene EP1. Preliminary results from the pilot showed that increases in expression of the genes UBP1, RBP21, Tb927.5.2910, and Tb927.11.8740 may upregulate EP1. In order to validate these hits, we cloned these genes into an overexpression vector and transfected it into the genome of the parasite reporter line SM EP1/GFP 8-D2, in which one EP1 allele is replaced with green fluorescent protein (GFP). We found that overexpression of these genes did not increase expression of EP1, revealing that we need to refine our threshold of what constitutes a significant increase in GFP expression for the high-throughput screen. Overall, these studies will provide insight into what genomic factors influence differentiation.

Poster abstract #10

Characterization of novel monoclonal antibodies in *Toxoplasma gondii* and *Neospora caninum*

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Toxoplasma gondii is an obligate intracellular parasite in the phylum Apicomplexa that causes serious disease in immunocompromised patients and congenitally infected neonates. *T. gondii* can infect every mammal and infection is widespread geographically, thus it is considered to be one of the most successful parasites on the planet. In addition, *Neospora caninum* is a closely related pathogen that causes abortion in cattle and neurological disease in dogs. *T. gondii* and *N. caninum* serve as model systems for the study of less amenable apicomplexans, including *Plasmodium spp.*, the causative agent of malaria, and *Cryptosporidium spp*., which causes diarrheal disease in children. We have utilized an organelle isolation and monoclonal antibody approach to develop a wide array of probes for subcellular localization. We have determined localizations that include the unique organelles of apicomplexans (e.g. inner membrane complex, rhoptries, and apicoplast) and standard eukaryotic organelles (e.g. the mitochondrion). A significant number of these probes cross-react between both *T. gondii* and *N. caninum*. We additionally characterized many of these antibodies by western blot analysis using both reducing and non-reducing conditions to determine the approximate size of the proteins they detect. We then used immunoprecipitation and mass spectrometry to identify the protein targets of a subset of these antibodies. Characterization of these antibodies will establish new molecular tools for the field and identify new putative drug targets in these important pathogens.

Poster Abstract #11

Investigating Neutrophil killing of *Tritrichomonas foetus*

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Tritrichomonas foetus is an extracellular protozoan parasite that causes trichomoniasis in cattle, a sexually transmitted disease. Bovine trichomoniasis has a global distribution, and the most recent prevalence data was in 2004, with 15.8% of California and 53% of Florida large beef producers reporting an outbreak. The venereal disease affects the breeding and calving seasons, reducing calf crop by 40% and revenue by 20% on infected farms. Although unlikely, the parasite can also be transmitted by artificial insemination. Bovine trichomoniasis symptoms are not usually obvious but may include inflammation throughout the female reproductive tract. Male cattle are typically asymptomatic and can become lifelong carriers, but females exhibit reduced fertility and abortions. These symptoms may be attributed to a strong immunological response to the parasite. There is a vaccine available against bovine trichomoniasis, that enhances fertility in infected herds by 32%, and decreases pathogen shedding. Antibodies are generated in response to the vaccine, however, immunoprophylaxis and long-term immunity are not established. A greater understanding of the immunological pathways involved in T. foetus killing would help to inform future design of a more effective vaccine. At the site of infection, neutrophils are the predominant immune cell type. T. vaginalis, a human parasite that also causes trichomoniasis, was recently shown to be killed by neutrophils via a dose-dependent and contact-dependent process known as trogocytosis, which means "to nibble." However, it is not yet known how effective bovine neutrophils are in killing T. foetus and what mechanism the neutrophils use in the killing. Furthermore, how T. foetus dies following neutrophil attack is not known. Here, we show that neutrophils can be isolated from bovine whole blood with high purity. We also show that upon co-culture with two strains of T. foetus, bovine neutrophils demonstrate moderate killing activity. Future experiments will determine whether antisera containing T. foetus antibodies can enhance neutrophil killing of T. foetus, and whether neutrophils kill T. foetus using trogocytosis. In the future, we will also examine the mode of parasite death (necrosis or apoptosis) following neutrophil attack, and examine downstream immunological consequences of neutrophil attack of T. foetus. The findings of this study will aid in understanding the cellular immune response to T. foetus and might assist in the development of new preventative treatments or the success of vaccination for bovine trichomoniasis.

Poster abstract #12

Toxoplasma gondii as a unique tool in the study of microglial activation and amyloid beta clearance in Alzheimer's disease

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Neuroinflammation is a common feature in many types of neurodegenerative diseases including Alzheimer's Disease (AD). In AD, neuroinflammation is thought to result from the accumulation of amyloid beta in plaques. However, the role of brain immune cells, such as microglia, in these pathologies remains unclear. Toxoplasma gondii is a foodborne parasite that induces inflammatory monocyte recruitment and microglial activation during infection of the brain. In mouse AD models, infection with T. gondii results in fewer amyloid beta aggregates in the brain, increased microglia activation, and decreased severity of AD symptoms. We are testing the hypothesis that T. gondii infection activates and enhances the phagocytic ability of microglia to clear amyloid beta. By confocal microscopy of brain sections from infected or uninfected 5xFAD mice, we have found increased IBA-1 area, and decreased amyloid area in infected brains. By conducting two-photon imaging of T. gondii-infected mice at baseline and 2 weeks post-infection, we have observed the progression of plaques over time. We are also investigating brain-wide effects of T. gondii on microglia and amyloid density using optical clearing and light sheet imaging of intact brains. To determine human microglial responses to this parasite, we have also infected microglia from human AD patient induced pluripotent stem cells. Within these experiments, we have seen that human microglia can become infected with T. gondii and display increased activation markers such as CD86. By probing the interaction between T. gondii, microglia, and amyloid beta, we hope to elucidate novel pathways mediating neuroinflammation and neuroprotection in AD.

Poster abstract #13

UNRAVELING THE GENOME WIDE DISTRIBUTION OF PLASMODIUM FALCIPARUM MORC PROTEIN

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Plasmodium falciparum has a complex life cycle that involve transition of various stages during the progression in human host and a mosquito vector. Understanding how the parasite regulates gene expression in different life cycle stages is a paramount goal in malaria research. Microrchidia (MORC) proteins have been implicated in the compaction of DNA, gene silencing and transcription regulation in plants and animals. In toxoplasma gondii, MORC has been demonstrated to act as transcriptional switch to control Toxoplasma development and sexual commitment. Here, we used a wide range of molecular and genome wide approaches including CRISPR-cas9 genome editing technology to confirm that in Plasmodium falciparum, MORC localize in the nucleus and may interact with the parasite chromatin. Using protein immunoprecipitation assays followed by mass spectrometry, our data suggest that PfMORC interacts with key development proteins including Histone Deacetylase 1(HDAC1) and Ap2 transcription factors (e.g. Pfap2G2). Finally, using ChIP-Seq technology, we successfully analyzed the genome wide distribution of PfMorc protein onto the chromatin in all asexual stages of the parasite intra-erythrocytic developmental cycle (IDC). Our results suggest that PfMorc strongly binds to sub telomeric region, var genes promoter and var gene bodies at the ring, trophozoite and schizont stages of the parasite IDC. Furthermore, differential peak analysis of ChIP-seq data at the trophozoite and schizont stages show differential binding of PfMORC at var genes and their promoter regions. Our chromatin immunoprecipitation studies also suggest that PfMORC interacts with the promotor of key development genes including several Ap2 transcription factors, rRNAs and invasion genes. Together these data pointing towards important role of PfMORC in var gene silencing and transcriptional regulation in the parasite.

Poster abstract #14

A Multi-omics Approach to Understand the Mode of Action of a Kalihinol Analogue, a Potent New Antimalarial against Plasmodium falciparum.

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The Kalihinol Med6-189 is a member of the family of Isocyanoterpenes (ICT), which have been shown to have potent activity against multiple microbial pathogens including the human malaria parasite Plasmodium falciparum. The compound is effective against drug-sensitive and -resistant parasites with IC50 values in the low nM range and therapeutic indices >500. Phenotypic analyses and cell biological assays using fluorescent probes demonstrated that the apicoplast is one of the sites of action of the drug in the P. falciparum-infected red blood cell. Drug-drug interaction studies as well as metabolite loading using isopentenyl pyrophosphate (IPP) confirmed that Med6-189 exerts its antimalarial activity through alteration of apicoplast metabolic processes. Metabolomic profiling and drug-protein interactions by thermal shift assays showed that Med6-189 inhibits membrane biogenesis and lipid trafficking. Consistent with this mode of action, drug resistance selection -albeit very slow to achieve- and whole genome sequencing implicated genes involved in vesicular trafficking in the reduced susceptibility to Med6-189 in resistant parasites. Altogether our studies unraveled a novel of mode of action of the antimalarial Kalihinol Med6-189 through inhibition of lipid trafficking and apicoplast biogenesis. The unique properties of this class of antimalarials, their high potency and excellent therapeutic profile, and the limited capacity of the parasite to mount resistance against them make them ideal compounds to further develop as a next generation of drugs for malaria treatment and elimination.

Poster abstract #15

Identifying chronic inflammation signatures of reactive astrocytes during T. gondii infection

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Neuroinflammation is common to neurodegenerative diseases and brain injuries, resulting in immune responses characterized in part by changes in astrocytes. Astrocytes ordinarily provide critical support for neurons but become reactive in response to brain disease, injury, or infection. Reactive astrocytes (RAs) are defined by their increased proliferation, enlarged cell bodies and processes, and change in function. A longstanding and unresolved issue is whether RAs contribute to or help alleviate disease progression. Toxoplasma gondii infects a third of the world's population and is asymptomatic except during times of immunocompromise when infection leads to severe neurological damage. Toxoplasma is a highly successful neurotropic parasite that causes persistent subclinical neuroinflammation due to cyst formation in neurons lasting for the lifetime of the host. During Toxoplasma infection, astrocytes demonstrate a neuroprotective function by inhibiting parasite replication via STAT1-mediated mechanisms, while simultaneously demonstrating detrimental effects by downregulating GLT-1, suggesting heterogeneity exists among RAs. Utilizing flow cytometry and integrin expression, we have characterized subsets of astrocytes found during infection. Experiments demonstrate specific chronic RA subsets are present. To further determine if there exists a defined chronic subset of RAs, single cell RNA sequencing experiments were conducted to determine if similar populations are expressed during acute compared to chronic infection. In addition, to investigate RA function we are using a novel transgenic reporter mouse, Lcn2CreERT2; Rosa26lsl-tdTomato, that will allow for the tracking and isolation of RA populations during infection.

11th Annual Southern California Eukaryotic Pathogen Symposium

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Thank you for joining us. We hope to see you next year at the 12th Annual SCEP Symposium!

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