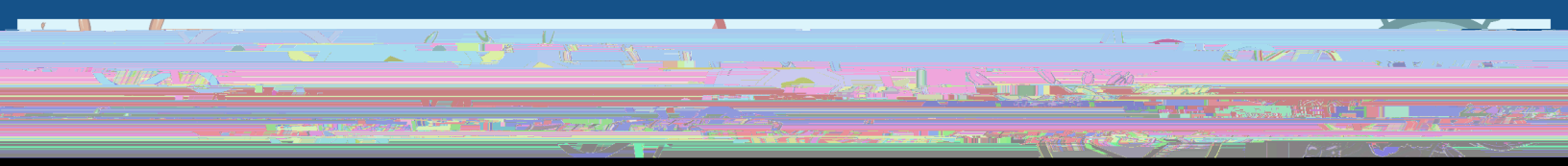

Order of Events

1:00pm	**Welcome and Opening Remarks**	**Dr. Allyson Shea**
1:05pm	**Keynote Speaker Presentation**	**Dr. Brian Fouty**
1:45pm	**Symposium Research Presentations**	
		Natthida Tongluan
		Killian Brewer
		Nam Suwanbongkot
2:30pm	**Intermission**	
2:45pm	**Symposium Research Presentations**	
		Amanda Tuckey
		Meagan Taylor
		Rachel Rodenberg
3:30pm	**Symposium Conclusion**	**Dr. Allyson Shea**

Symposium Planning Committee

Faculty Organizer—Dr. Allyson Shea

Student Members—Amanda Tuckey, Natthida Tongluan, and Rachel Rodenberg

Logistics—Meredith Moody

Roles of rickettsial outer membrane protein B (OmpB) in tick vector

Natthida Tongluan and Kevin R. Macaluso

Department of Microbiology and Immunology, University of South Alabama, Frederick P. Whiddon College of Medicine, Mobile, AL, United States

Rickettsia parkeri, a member of spotted fever group *Rickettsia*,

Role of T-cells in *Rickettsia parkeri* infection at skin interface

Chanakan Suwanbongkot, Monika Danchenko, and Kevin Macaluso

Department of Microbiology and Immunology, Frederick P. Whiddon College of Medicine, University of South Alabama, Mobile, USA

Rickettsia parkeri, an emerging bacterial pathogen, is transmitted by *Amblyomma maculatum* via infected tick saliva. During feeding, ticks secrete numerous salivary factors manipulating the host's hemostatic and immune response to promote blood feeding. With the immunomodulation property,

Chikungunya virus (CHIKV) infection in permissive mammalian cells

Meagan Taylor and Jonathan O. Rayner

Department of Microbiology and Immunology, Frederick P. Whiddon College of Medicine, University of South Alabama, Mobile, USA

Chikungunya virus (CHIKV), a mosquito-borne pathogen, is an emerging public health threat. While CHIKV tropism has been well characterized, no published studies have evaluated the replication dynamics of CHIKV in RIG-I-deficient H1-HeLa cells. To evaluate this, we infected both HeLa and H1-HeLa cells with culture-attenuated CHIKV (181/25) and evaluated titer levels at different time points. Compared to wild-type HeLa cells, CHIKV titers were consistently elevated in H1-HeLa cells. Interestingly, although the data did not show statistical significance, we expect this to be due to the use of culture-attenuated CHIKV rather than a wild-type pathogenic strain. These results suggest that a pathogenic strain (or symptomatic patient isolate) should be used to uncover the immune response associated with CHIKV infection in mammalian ho-2 (1)e pathog

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γδ T17 cells are master regulators of the acute antiviral response in the HSV-1 infected cornea

Rachel Rodenberg and Robert Barrington

Department of Microbiology and Immunology, Frederick P. Whiddon College of Medicine, University of South Alabama, Mobile, USA

Herpes Stromal Keratitis (HSK), caused by herpes simplex virus 1 (HSV-1) infecting the cornea,