Kaleb Asfaha was born in Addis Ababa, Ethiopia and grew up in the Washington D.C. metropolitan area. Before coming to UC Berkeley he studied Chemistry at the University of California Santa Barbara and San Francisco State University for a bachelor's and master's degree, respectively. He is currently a PhD student in the Vision Science graduate group housed under the School of Optometry. He works in the Gronert Lab where they study the lipid mediators involved in ocular inflammation. He is interested in conducting basic and translational research uncovering new ways to restrain undesirable inflammation in the eye, using *Chlamydia trachomatis* bacteria as a model. In the future, he hopes to open a non-profit pharmaceutical organization that addresses the needs of emerging markets.

**Fellowship Proposal**

Trachoma is a bacterial eye infection caused by *Chlamydia trachomatis* that affects an estimated 80 million people across more than 50 countries. The infection results in a roughening of the inside of the eyelid, leading to pain, breakdown of the cornea surface, and eventually to blindness, making trachoma leading cause of preventable blindness in the world. 230 million people are at risk. The bacteria spreads through personal contact or from flies contaminated with the bacteria. While it is treatable with antibiotics, infections can recur repeatedly. Surgery is often required to repair damage caused by repeated and long-term infections.

Kaleb proposes to work with UC Berkeley optometry professor Karsten Gronert, Dr. Deborah Dean of the Children’s Hospital of Oakland Research Institute (CHORI), and Dr. Hiwot Denigeh an ophthalmologist and director of the Felege Referral Hospital in Bahir Dar, Ethiopia. Trachoma is endemic in Ethiopia, and Kaleb will travel there during the summer of 2015 to collect samples for his research into key aspects of the pathology of and immune response to *C. trachomatis* eye infections. Kaleb’s goal will be analyze the detailed molecular signature of the innate immune response to infection in an attempt to confirm a hypothesis that these bacteria hi-jack aspects of this response to promote transmission throughout the eye. Previous results have shown evidence of a similar trend in a model of sexually transmitted *C. trachomatis* infection. Using the collected eye cells, Kaleb will develop a comprehensive picture of the infected cell’s immune response, including its lipid and gene expression profiles. He will then culture collected eye tissue and bacterial samples to recapitulate the infection *ex-vivo* and compare the molecular profile of these cells with that seen in the clinical samples.

With this work, Kaleb seeks to establish how intracellular sensing of microbes and the regulation of lipid molecules interface to regulate innate immune response and the *C. trachomatis* life-cycle. He sees this research as holding the promise for identifying novel targets to thwart chlamydial survival, end the cycles of infection, and thus terminate the downstream complications of visual impairment and blindness.