When I found out that I would be spending my summer at the University of Pittsburgh, I was thrilled by the thought of not only getting the chance to be trained to work in a lab conducting relevant research, but also at the chance to contribute to the scientific endeavor of understanding ourselves and the world around us (in whatever small way I could in only ten weeks). However, when I arrived in PA and started my apprenticeship with Dr. Cecelia Yates, my expectations were blown away. Not only was her own time and lab at my disposal to ensure an optimum learning environment for myself, but she also coordinated my collaboration with Dr. Binder’s lab in the Department of Immunology and Dr. Veraldi’s lab in the Department of Medicine. Furthermore, everyone who I worked with welcomed me with open arms and endearing patience in teaching me the skills and techniques crucial to conducting experiments that provide specific insights into the biochemical mechanisms of disease progression. Specifically, my time was spent investigating molecular chaperones called heat shock proteins (HSP) as therapeutic candidates for regulating pathologic fibrosis. To do this I was taught how to grow dermal human fibroblasts in vitro, treat them with growth factors and inhibitors, and run quantifiable analysis in the form of immunoblots and immunofluorescence staining to record differential protein expression between experimental groups. Additionally I used fluorescence microscopy to image sections of mouse cardiac tissue and learned how to perform a chemokine array on mouse lung tissue. By the end my internship we had gathered convincing preliminary data, suggesting that HSP70 and HSP90 (members of the HSP family) play a role in the transforming growth factor beta (TGF-β) transphosphorylation cascade, an integral component of the mechanisms by which fibroblasts take on a profibrotic phenotype, which in turn produces excessive and disorganized extracellular matrix if dysregulated. This excessive matrix deposition eventually compromises tissue function and homeostasis resulting in organ failure and death in progressive fibrosis patients. Being my first research experience, this opportunity gave me priceless insight into the career that I am pursuing. From immersion in literature from multiple overlapping fields of research, writing out, understanding, and explaining my work to others, to rigorously analyzing data, what I gained from this experience could not be replicated by any number of lectures or lab courses. The fact that we were able to generate convincing evidence of potential therapeutic candidates for treating fibrosis during my ten short weeks at Pitt was enough to make me long for more time before the start of spring semester to conduct follow-up experiments and gain a more complete picture of the molecular chaperone interactions that contribute to fibrogenesis. However, all good things must come to an end and I am nevertheless immensely grateful for the opportunity that I was given this summer in addition to the personal connections and support that I have established and have available to me through not only my mentors, but also the organizers of the program such as Dr. Mars whose endless enthusiasm and advice was and still is only an email away. As I prepare to start another academic semester, a part of me will always be tied to Dr. Yates' pathology lab at the University of Pittsburgh for seeing me through one of the most exciting milestones of my development as a scientist. To the collaborators of ASIP and the organizers of SROPP, thank you so much for investing your time, work, and resources in myself and my peers.