I am a 1st year Computer Science PhD student at Stony Brook University where I am advised by [Dr. Phillipa Gill](http://www.cs.stonybrook.edu/~phillipa/index.php).

Before coming to Stony Brook University, I completed my undergraduate degree from [LUMS SSE](http://lums.edu.pk/) where I worked with [Dr. Ihsan Ayyub Qazi](http://web.lums.edu.pk/~ihsan/) on Wireless Networks for 2 years.

I am an MS Biology student at Texas A&M University where I work in an Immunology lab under Dr. Richard Gomer. My undergraduate degree in Biology was completed at LUMS SSE where I worked with Dr. Zakir Ullah on Stem Cell Biology for 2 years.

Publications

“Loss Differentiation: Moving onto High-Speed Wireless LANs” Ruwaifa Anwar, Kamran Nishat, Mohsin Ali, Zahaib Akhtar, Haseeb Niaz and Ihsan Ayyub Qazi IEEE INFOCOM 2014, Toronto, Canada, April 2014 (To Appear)

“BLMon: A Loss Differentiation Scheme for 802.11n” [[PDF]](file:///Users/yumnazahid/Desktop/sb_page_v3%202/public_html/infocom_poster.pdf) Zahaib Akhtar, Kamran Nishat, Haseeb Niaz, Ruwaifa Anwar, Mohsin Ali, and Ihsan Ayyub Qazi IEEE INFOCOM 2013, Turin, Italy, April 2013 (poster paper)

Projects:

# Conditioned medium prepared from immortalized mouse embryonic fibroblasts supports culture of trophoblast stem (TS) cells

Trophoblast stem cells (TS) are the precursors of the differentiated cells of the placenta. TS cells are used as a model system to study placental development and function. TS cell cultures require fibroblasts conditioned medium to maintain their phenotype. Generally, primary mouse embryonic fibroblasts (PMEF) are used to prepare conditioned medium. But PMEFs are harder to derive as primary cells have slow growth and require extraction of 10-12 days old embryos from pregnant mice. In this project, we tried to eliminate these problems and tested immortalized PMEFs to prepare conditioned media for TS cell culture. Immortalized PMEFs were produced by transfecting PMEFs with a proto-oncogene SV-40 latge T antigen. They were better than PMEFs as they proliferated significantly faster, which was observed in comparative cultures of both cell lines. Immortalized PMEFs were also easier to derive because they are not primary cells and did not require dissection of pregnant mice once the cell line was established. We tested growth of TS cells on both conditioned media and showed immortalized PMEF conditioned media also supported TS cell growth in cultures. For further analysis, feeder layers of both cell types were used to show comparative growths of TS cell colonies. TS cells grown in both media were then differentiated into TG cells for comparative morphology analysis of differentiation.

# Production of induced pluripotent stem cells

Induced pluripotent stem cells (iPSCs) are a new type of pluripotent cells that can be obtained by reprogramming animal and human differentiated cells. The aim of this project was to induce pluripotency in human adult fibroblasts, derived from patient biopsies so they can be used in personalized regenerative medicine. To optimize and develop the protocols, mouse induced pluripotent stem cells were established from primary mouse embryonic fibroblasts (PMEFs), derived from 10-12 days old pregnant mouse. To produce human iPS cells, human fibroblasts were derived from a patient skin biopsy. Reprogramming factors, Oct4, Sox2, c-Myc and Klf4, were used to induce pluripotency in human fibroblasts.

**A blood protein affects multiple aspects of human lung epithelial cells**

Fibrosis involves an inappropriate and unnecessary wound healing response in a tissue, resulting in scarring and dysfunctiuon of the tissue. Fibrosis is associated with 45% of deaths in the US. In wound healing and fibrosis, immune system cells leave the blood, enter the tissue, and become activated to orchestrate the scar tissue formation. Blood contains a protein called Serum Amyloid P (SAP) which by deactivating immune system cells inhibits fibrosis in animal models and human clinical trials. SAP binds a C-type lectin receptor called DC-SIGN. DC-SIGN was intially found on monocyte-derived dendritic cells, but we previously observed that lung epithelial cells also express DC-SIGN and that activation of DC-SIGN causes the epithelial cells to express a protein called IL-10. Immunofluorescence showed DC-SIGN to be localized in the nucleus in lung epithelial cells. After exposure of lung epithelial cells to a DC-SIGN ligand, DC-SIGN becomes more prominently expressed in the plasma membrane and the cytoplasm near the membrane. In response to treatment with DC-SIGN ligands, epithelial cells also start blebbing. To determine what else SAP might affect, human lung epithelial cells were treated with SAP and its synthetic analogue, Compound 1, and subjected to proteomics. Many proteins were consistently up- or down-regulated in response to treatment with SAP and Compound 1. Some of the proteins consistently upregulated include Keratin 1 and Keratin 16, and some of the proteins consistently downregulated include Tubulin beta-6 and Calpain-2. These results indicate that epithelial cells respond in several ways to SAP (and Compound 1), and suggest new regulatory connections between the immune system and epithelial cells.