

Graduate Women in Science Travel Award Report – Spring 2011

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Thanks to support provided by a GWIS travel award, I was able to attend the Gordon Research Conference on “Epigenetics: Mechanisms, Development and Disease” in Easton, Massachusetts. The Gordon Research Conferences are recognized for presentations and discussions of the latest discoveries and novel methodologies by the best researchers in the field. I was fortunate enough to be accepted to participate and present a poster at this meeting.

There were 200 participants who covered many areas of epigenetic research using model systems and innovative techniques and applications. During the talks, I learned a lot from how specific techniques can be applied to particular model organisms to address complex questions. I incorporated some of the speakers’ ways of thinking about gene regulation and epigenetic marks during development, which is helping me to better evaluate where I am going and what experiments need to be done in my own research project.

I work with murine or human cell lines and mice, and I was able to discuss chromosome conformation capture (3C) with Nora Engel’s laboratory. Engel’s laboratory took advantage of 3C, which is a technique useful to illustrate the structural properties and spatial organization of chromosomes, to discover that imprinted regions are looped out through tissue-specific enhancer interactions in mouse. This is one of many possible directions I could take my project, and meeting people who work with 3C was of great help!

Apart from the many presentations, interactions at posters and other scientific encounters, during meals I was able to talk about my latest data on the characterization of the Δ HS-3’ β 2mice line. HS-3’ β 2 is a DNase 1 hypersensitive element that acts as an enhancer in transient reporter gene assays. The Δ HS-3’ β 2mice line is our loss-of-function model to further examine HS-3’ β 2 enhancer function. My research aims to determine if two DNase 1 hypersensitive regions in the murine β -globin gene cluster, HS-E1 and HS-3’ β 2, are responsible for hyperacetylated domain formation and/or function. Hyperacetylated domains are regions of ten of kilobases that are enriched in acetylated histone H3 and other histone modifications associated with gene activation. We were able to show that the two elements, HS-E1, located between the two embryonic genes, $\epsilon\gamma$ and β h1, and HS-3’ β 2, located downstream of the β 2 gene, act as enhancers in a gene reporter assay. I presented data from our latest publication that characterized the Δ HS-E1mice line, accompanied by our advances made toward characterizing the Δ HS-3’ β 2mice line. The latter is our current interest. Feedback from my presentations was quite useful and will make my work more complete.

Overall, this conference was a wonderful networking experience and introduction to the epigenetics field. I am truly grateful to GWIS for making it happen. This opportunity was valuable for my progress as a scientist-in-training.