Genetic Modifier Screening of Border Cells in *Drosophila*

There are many cells in humans and animals that migrate collectively, whether it be in tight or loosely gathered groups. These cells are responsible for beneficial processes such as forming organs, immune system responses, and embryonic development. Harmful process, like that of tumor metastasis, are also caused by the migration of these groups of cells. The model organism, *Drosophila*, is very helpful in observing this cell migration because of their border cells that travel in a cluster to the large oocyte during the development of the ovary. Also, many *Drosophila* genes have human homologs and therefore have correlation in the study of human's cell migrations. It was recently discovered that the Nuclear inhibitor of protein Ser/Thr phosphatase 1, or NiPP1, protein causes the border cell cluster to fall apart and travel slower during the migration to the oocyte. This is because the role of phosphatase is to keep border cells together and by adding NiPP1, it blocks or lowers phosphatase activity which leads to the falling apart of the border cell cluster. Using a genetic modifier screen, one can test the overexpression of NiPP1 to observe the Protein Phosphatase 1 (PP1) regulatory and catalytic subunits required for the adhesion and the collective migration of the border cells as well as discover the genes that are required for the correct function of PP1 in the border cells. The method of processing the genetic modifier screen is to first cross females that express NiPP1 in the border cells to males with deficiencies, which are mutant strains that have a large number of genes removed. The F1 generation is then used to collect the ovaries from the females that contain the correct genotype. These ovaries are then dissected, antibody stained, and then visualized using fluorescent microscopy. The results are then compiled and analyzed by comparing them to other deficiencies. Later, this process will move onto performing genetic screens with males with smaller deficiency mutant strains in order to determine the genes that modify NiPP1 phenotypes. Lastly, RNAi will be used to eliminate genes to determine those required for the adhesion and collective migration of the border cells. The hypothesis is that this process will result in the discovery of some genes from the mutant strains (deficiencies) that will both enhance and suppress the phenotype of border cell migration that will help to establish the relationship of certain genes to cell migration that can be used in later testing.