1)Auld, V. J., Fetter, R. D., Broadie, K. and Goodman, C. S. (1995). Gliotactin, a novel transmembrane protein on peripheral glia, is required to form the blood-nerve barrier in Drosophila. Cell 81, 757-767.

 This paper is the original paper that characterized the expression and function of Gliotactin in epithelia and peripheral glia. Researchers showed that Gli is necessary for septate junction formation in the nervous system. The evaluation of barrier permeability experiment with dye in the project is derived from the transepithelial barrier assay of this paper. The experimental design to generate knock down mutants is also inspired by this paper.

2) Bhat, M. A., Rios, J. C., Lu, Y., Garcia-Fresco, G. P., Ching, W., St Martin, M., Li, J., Einheber, S., Chesler, M., Rosenbluth, J., et al. (2001). Axon-glia interactions and the domain organization of myelinated axons requires neurexin IV/Caspr/Paranodin. Neuron 30, 369-383.

 This study investigated mice proteins involved in formation of glia. The introduction of this paper provided me an overview of the similarity and conservation or proteins present in the paranodal junction in vertebrates and the septate junction in fruit fly. Researchers in the study performed a screen for human *Neurexin* *IV* sequence utilizing the known Drosophila *neurexin* *IV* sequence. Their method inspired me to take on a similar bioinformatics approach to identify the *Drosophila* disc large interactome from the human disc large interactome.

3) Browne, K. (2009). Strategies to identify Gliotactin protein interactors in Drosophila (Unpublished master's thesis). Thesis / Dissertation at the University of British Columbia.

 This thesis is one of the two theses that formed provided the most information for my research project. The thesis compiled information and proposed a model interactome for Gli. The introduction paragraph of this thesis provided in depth comparison of junction structures and functions. Its images were particularly helpful in aiding my understanding in all the junctions and functions. This thesis also provided extensive information on Gli mutant phenotypes in the nervous system and in the epithelia. In chapter 4.4.1, Browne summarized some techniques that were unsuccessful in identifying the interactome of Gli.

4) Cox, E. A. (2004). Cell adhesion receptors in C. elegans. Journal of Cell Science, 117(10), 1867-1870. doi:10.1242/jcs.01177

The is a review article that provided an overview of all the receptors that may interact with the extracellular matrix across different species such as *C. elegans* and *Drosophila*. While this article did not contribute to the text of the research project. I used one of the figures to illustrate the structural differences between tight junctions in vertebrates and sepatate junctions in fruit flies.

5) Frese, K. K., Latorre, I. J., Chung, S., Caruana, G., Bernstein, A., Jones, S. N., Javier, R. T. (2006). Oncogenic function for the Dlg1 mammalian homolog of the Drosophila discs-large tumor suppressor. The EMBO Journal, 25(6), 1406-1417. doi:10.1038/sj.emboj.7601030

 This mammalian study investigated the tumor suppression function of human homolog of *Drosophila* disc large in mice and cell culture. While the focus of my research is not on the human disc large, this study provided information on the conservation of PDZ binding domain between the human and fruit fly homolog. It also summarized some human diseases associated with the malfunction of disc large.

6) Laprise, P., Viel, A., & Rivard, N. (2003). Human Homolog of Disc-large Is Required for Adherens Junction Assembly and Differentiation of Human Intestinal Epithelial Cells. Journal of Biological Chemistry, 279(11), 10157-10166. doi:10.1074/jbc.m309843200

 This research paper focused on the function of human disc large on junction assembly and human intestinal epithelial cell differentiation. This study provided evidence that the human disc large and the Drosophila disc large share similar function in junction assembly, further strengthening the basis for the hypothesis which assumed that the conservation of protein sequence and structure would likely be associated with conservation of protein interactome.

7) Lee, M., & Vasioukhin, V. (2008). Cell polarity and cancer - cell and tissue polarity as a non-canonical tumor suppressor. Journal of Cell Science, 121(8), 1141-1150. doi:10.1242/jcs.016634

 This paper mainly discusses how cell and tissue polarity maintenance functions to suppress the formation of tumors. In the introduction, the molecular interaction of disc large in suppressing tumors was briefly discussed. This paper contributed to my understanding of how disc large’s function in establishing polarity contributes to regulated mitosis and the suppression of undesired overgrowth of cells as observed in tumor cells.

8) Limmer, S., Weiler, A., Volkenhoff, A., Babatz, F., & Klämbt, C. (2014). The Drosophila blood-brain barrier: development and function of a glial endothelium. Frontiers in Neuroscience, 8. doi:10.3389/fnins.2014.00365

 This paper served as a background reading for the research project as it described the formation of the blood-brain barrier and glial development in depth.

9) Schulte, J., Charish, K., Que, J., Ravn, S., MacKinnon, C. and Auld, V. J. (2006). Gliotactin and Discs large form a protein complex at the tricellular junction of polarized epithelial cells in Drosophila. Journal of cell science 119, 4391-4401.

 This study described the localization of Gliotactin and Disc large at various *Drosophila* larval tissues with immunostaining. The prediction of experimental results of my proposal is deduced from the observations of Dlg and Gli mutant knock down in this study. Furthermore, this study found that while the PDZ binding motif of Gli may be important in signalling, the localization of Gli is independent of its PDZ binding motif. Moreover, researchers also found that overexpression of Gli causes a downregulation of Dlg in the wing imaginal, which suggested that Gli and Dlg localization are mediated by intermediate proteins since Dli and Dlg do not physically interact. The staining technique method in my research proposal was based on the immunostaining method described in this study.

10 ) Schulte, J., Tepass, U. and Auld, V. J. (2003). Gliotactin, a novel marker of tricellular junctions, is necessary for septate junction development in Drosophila. The Journal of cell biology 161, 991-1000.

 This study investigated the function of Gliotactin in the formation of septate junction and tricellular junction. Researchers found that Gliotactin localization at the tricellular junction is essential to the compaction and formation of the septate junction. The dye permeability assay used in this study helped me devise a method to evaluate the integrity of blood brain barrier in developing fruit fly embryos.

11) Sharif Khodaei, Z. (2017). Tricellular Junction Regulation, Signaling and Scaffolding by ( pending publish doctorate thesis). University of British Columbia.

This doctorate thesis summarized the functions of all the junctions that was discussed in my introduction, all known septate junction protein interactions, and all the core components of the tricellular junction in the first chapter. This thesis provided vital background information for me to understand all the other primary literary on septate junction protein interactions.