Vector dissemination (brackney, 2017). this study has been

Vector virus interactions is a field of study that has been focused on over time, especially with increased outbreaks of vector borne diseases that have lead to significant mortality across the world. The anatomy of the aedes aegypti mosquito is crucial in looking at virus dissemination by going directly to the root of the problem. The spread of diseases commences by a mosquito taking an infectious blood meal, which then travels through the salivary glands and into the midgut, in which it replicates itself. The midgut is surrounded by a virus impermeable basal lamina which refers to a surrounding protective layer.

Entry and exiting of viruses from the mosquito constitutes the main function of the mosquito midgut. Studies have been done to show that an additional blood meal contributes to increased efficacy of virus dissemination (Brackney, 2017). This study has been separated into two phases. For phase one, it was hypothesized that if multiple blood meals are administered, then the integrity of the basal lamina will be compromised. Other studies have shown the effect of different feeding on the basal lamina.

The basal lamina of sugarfed mosquitoes was not affected, while blood meals compromised the integrity of the basal lamina with the chikungunya virus. (Dong, 2017). Using uninfected blood in this experiment, the independent variable is defined as the different feeding backgrounds while the dependent variable is the integrity of the basal lamina. In this study apoptosis is defined as programmed cell death of the mosquito. When looking at the role of apoptosis, if the mosquito is able to trigger an antiviral response and increase programmed cell death this should logically result in decreased virus emission since there is less replication of the infected cells.

The restriction is that many viruses are encoded with proteins that inhibit apoptosis which is crucial in the spread of diseases. (Clem, 2016). Other research has demonstrated that inhibiting the AeDronc (initiator of apoptosis) gene leads to increased virus infection of dengue in aedes aegypti mosquitoes (Ocampo, 2013).

Studies have shown that when apoptosis is inhibited, mortality rates increase to about 60-70% in virus infected mosquitos (Wang, 2008). The link between apoptosis and integrity of the basal lamina has currently not been researched and will constitute phase two of this experiment. It is hypothesized for phase two that if apoptosis is inhibited then the integrity of the basal lamina will be compromised.

The independent variable has been defined as the induction/inhibition of apoptosis and the dependent variable is the integrity of the basal lamina. Other studies have revealed the link between increased virus prevalence and inhibiting apoptosis. Therefore, it can be assumed that the increased virus prevalence has a link between the integrity of the basal lamina as well. A connection can be proposed between the basal lamina integrity and apoptosis. This can be supported by the fact that previous studies have shown that when there are holes in the basal lamina it becomes inherently easier for viruses to enter and escape the midgut (Dong, 2017). Other research has shown that the maximum that the basal lamina can allow is about 5-8nm in diameter. Since virions are actually five times the maximum allowance and are able to pass through it gives further clues that the basal lamina is not a rigid structure but rather a dynamic one that adapts to

different conditions and creates these “ tears” (Houk, 1981). The actual structure of the basal lamina can also hint to the proposed results.

The basal lamina has been imaged to find that it is a grid like structure and when parallel to the epithelial cells it forms a beaded membrane (Terzakis, 1967). This grid like structure can also support how the basal lamina is “ flexible” and different factors can cause physiological or biological stress causing it to expand and create holes in the process.