# Causes of Increased Rates of Transvection at the 96C Site in Drosophila Melanogaster

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The main question behind my research is: why are we seeing high rates of transvection at the 96C chromosome site in Drosophila Melanogaster? Before diving into the specifics of my research, some background knowledge is necessary.

Since we are seeing increased rates of transvection, we want to think about gene regulation, the process that controls how much of a gene is expressed, and may be affecting this specific site. Enhancers and promoters are important in this regulation, where enhancers increase transcription and promoters beginning transcription. These two factors work in unison to transcribe a designated gene and allow expression. There are two types of expression, cis expression and trans expression. Cis expression regulates gene expression on the same chromosome, while trans expression is the occurrence of one chromosome regulating the transcription of its structurally similar chromosome. Trans expression will occur when one chromosome is missing an enhancer and the other is missing a promoter, allowing expression to still occur without a chromosome having both expression factors. This study focuses on trans expression, which is likely causing us to see these high rates of transvection.

In a paper by King et. al they focused on enhancer action in cis and trans at specific chromosomal locations by implementing a transgene encoding the P-element transposase. This employs a strong synthetic eye-specific enhancer, GMR. Using this eye tissue-specific enhancer and a green fluorescent protein (GFP) reporter, the eyes can be dissected and quantified using a confocal microscope to determine fluorescence rates. These fluorescent rates allow us to quantify the amount of transvection occurring at the 96C site.

Using the information in the King et. al paper we were able to produce two possible explanations, the first being that the 96C location could have unique features causing the high rates of transvection. The second being the changes to the P-element structure; it seems that the W U D Q W U D Q V S R V D V H O H P H Q W was damaged and repaired through an insertion. P-elements can be moved through transposases, but in this case, it seems to have been deleted. When the P-element was deleted, it was repaired with a mini-white promoter and two Zeste protein binding sites.

To narrow down which possibility is more likely we ly we lch possich possich possich possich >qch poss

-element at another chromosome location, by introducing a transposable element. If the change in location of the P-element affects the rates of transvection at a location other than the 96C location, we can conclude that this may be causing the high rates of transvection.

To test the rates of transvection we use PCR amplification along with dissecting the eye discs of third instar larvae. Few dissections were completed before we ran into technical difficulties. Although no conclusive results were found, another member of the Bateman lab will pick up where we left off. We are