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Characterization of the FoxP[X43] deficiency in *Drosophila*

CG1699 is the Drosophila homolog of the human FoxP genes, which are important in vocal learning and speech. This is evident in a mutation of human FoxP2 causing a dominant developmental dysphasia (a speech disorder). A transposable P-element, RS-5-3955, was inserted into the last exon of Drosophila FoxP. An imprecise P-element excision resulted in a deficiency, termed FoxP[X43], which has a recessive lethal and a dominant brain dsymorphic phenotype. The aim of this project was to determine the extent of the deficiency in FoxP[X43] and to verify that it affects only the FoxP locus. To do this, crosses of FoxP[X43] and nearby P-elements were made, and then Polymerase Chain Reaction (PCR) was used to verify whether or not the FoxP[X43] deficiency contains DNA on both sides of the insertion. The logic is that PCR cannot easily amplify fragments greater than 3 kb whereas the P-element insertion is over 8 kb in length. Therefore, if we obtain a product using primers on both sides of the element this must have come from the FoxP[X43] chromosome. We can then move one primer further down the chromosome towards the FoxP locus until PCR fails in FoxP[X43] / P flies but not in wild type / P flies. The strategy is then applied to the other side using different P-elements and primers. When candidate deficiency endpoints have been found, PCR across the deficiency will identify the two deficiency ends. Once characterized, this will provide integral information for studying the FoxP genes in both Drosophila and humans.

This project was completed to fulfill a Capstone requirement.