A Drosophila Glioma Model to Investigate Therapy Resistance and Repopulation

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Glioblastoma is the primary cause of cancer related fatalities in pediatric and adult populations. The current standards for treatment have been unsuccessful in preventing tumor relapse due to the highly invasive and proliferating nature of the tumor. Glioblastoma is caused by DNA alterations resulting in over-expression of oncogenes or inactivation of tumor-suppressor genes. When mutated, the PI-3 Kinase pathway is significant to cancer development. My hypothesis is that we can further study how tumor repopulation occurs following irradiation in Glioblastoma by creating a simulation in a Drosophila model by inducing this mutated pathway. The aims of my study were to create a Glioblastoma model in Drosophila and identify idea levels of radiation that a tumor-induced larvae could be exposed to, in order to study tumor repopulation following irradiation. The PI3K and EGFR pathways were induced into the glial cells of Drosophila models. Radiation was tested on wild type and tumor induced Drosophila and 3.5 gy was established as the ideal level of radiation for tumor-induced larvae. After radiation, the brain samples were dissected and stained with antibodies ELAV, Prospero, and Repo to identify different areas of the brain lobes and how they were affected. Brain lobe samples were examined through confocal microscopy and brain size was compared by calculating sample image pixelation. It was concluded that the radiation reduced tumor load and caused a delay in the developmental cycle of the tumor induced larvae.

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