

Title: Analyzing the Ability of CDK Inhibitors to Enhance Response to Treatment in Recurrent Glioblastomas

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Glioblastomas (GBM) are the most common and aggressive type of primary brain tumors found in adults with a median survival of 15 months for newly diagnosed patients. The current most standard form of chemotherapy is with Temozolomide (TMZ). However, one cause for the low survival of GBM patients is due to their ability to resist the programmed cell death pathways caused by TMZ. The aim of this study is to analyze how cyclin-dependent kinase (CDK) inhibitors, specifically CYC065: a CDK 2/9 inhibitor, can potentially enhance the response to treatment with TMZ in patient-derived GBM cell lines, MZ256 and MZ304. Each cell line was cultured and then treated with a control of DMSO, TMZ (150 μM), CYC065 (5 μM) alone and in combination for 24, 48, and 72 hours. In order to analyze the cells' responses to treatment, researchers used Western Blots to detect the levels of Caspase-3, a caspase protein involved in apoptosis execution and MCL1, an anti-apoptotic protein, with Beta-Actin serving as a control for the analytic method. Researchers also conducted MTT assays to assess cell metabolic activity. Results obtained via western blots how a decreased level of Caspase-3 expression in the cells treated with CYC065 and the CYC065-TMZ combination treatment and show that CYC065 was able to successfully target the MCL1 protein in the cell lines. Both the western blots and the MTT assays also suggest that CYC065 not only affects cell viability, but also sensitizes glioblastoma cells to TMZ.

Biography

Nikita Bhatia is a fourth-year student at the University of California, Berkeley studying bioengineering with an emphasis on cell and tissue engineering. She was previously a summer student through the StAR Research Program at the Royal College of Surgeons, where she conducted research on GBMs. Currently, Nikita Bhatia does at the University of California, San Francisco on tumorigenesis and signaling pathways in cancer.

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