OFFICIAL ABSTRACT and CERTIFICATION

Examining P53 Mutant Triple Negative Breast Cancer Cell Viability and Sphingosine Kinase 1 in Response to CHK1 Inhibitor and Doxorubicin

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Triple Negative Breast Cancer [TNBC] is breast cancer that lacks abnormal expression of the progesterone receptor, estrogen receptor, or human epidermal growth factor receptor 2. Without this abnormal expression, targeted hormone therapy cannot be used for TNBC. In addition, 44% of TNBCs have nonfunctional p53 genes. Since p53 responds to DNA damage by halting cell cycle progression and inducing apoptosis, TNBC without functional p53 is also more resilient to genotoxic chemotherapy agents. In this investigation, check kinase 1 [CHK1], which functions by blocking cell cycle progression to allow DNA repair, was inhibited in combination with genotoxic stress. It was hypothesized that genotoxic stress with the CHK1 inhibition would damage the genome of a cell and then prevent the cell from repairing such damage, resulting in cell death. Cell viability and sphingosine kinase 1 [SK1], an enzyme associated with cell proliferation, levels were observed to assess the effectiveness of the combination treatment as compared to a single treatment of each drug and no treatment. No statistically significant differences in cell viability were observed among the treatment groups. However, there was a trend in the data suggesting that CHK1 inhibition alone and CHK1 inhibition combined with genotoxic stress are equally effective at reducing cell viability compared to genotoxic stress alone or no treatment. If supported by more data, this would suggest the potential of using CHK1 inhibition alone as a TNBC cancer treatment. This would be beneficial as this could avoid the side effect of cardiotoxicity from using genotoxic agents.

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