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Differential Expression Of BCL-2 proteins In U2OS: A Look At What Really Kills Cancer

The benefits of developing a cancer drug that can specifically target and kill tumor cells are many. Finding the missing links between tumor-suppressor activation and cell death was the goal of this study in the osteosarcoma line U2OS. Samples were grown and split into a control and two test treatments of which one was treated with a chemotherapeutic drug "5Flourauracil" and the other with "Nutlin3a." 5FU has been shown to kill U2OS bone cancer cells, yet it kills rather imprecisely and destroys properly-functioning cells simultaneously. Nutlin, however, was designed to induce cell-death pathways in cancer cells, yet it fails to induce apoptosis. After treatment, western blots were taken to observe expression of key proteins to understand which may be the "turning-points" between cancer cell life and death. p53, a tumor suppressor gene, was activated in both treatments and Puma, a BCL-2 protein, was expressed equally in both treatments. Follow up blots indicated that Caspases 3 and 8 were activated in the 5FU treatment and not Nutlin. Equal Puma expression implies that apoptosis is not Puma-dependent in U2OS. Caspase 8, which is only activated by external factors, implies that 5FU induces cell death via extrinsic factors.