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Exploration of Antagonist Efficacy for LPrA2 Like Peptide

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ABSTRACT

Leptin is a small protein hormone that controls satiety and is produced by adipocytes. Obese people have increased levels of circulating leptin. Excessive leptin levels cause a break down in the control of leptin signaling pathways leading to increased angiogenesis, proliferation, cell migration, invasion, and anti-apoptotic events. Obesity and leptin signaling have been linked to cancer progression. Literature shows that LPrA2 is an effective leptin antagonist as it decreases proliferation of breast cancer cells in vitro.

This project compares the effectiveness of LPrA2 like compounds in breast cancer cell-line MDA-MB-468 (M-468 BCs). We hypothesize that the new peptide antagonists have shorter sequences than LPrA2 and could be more advantageous inhibitors of leptin signaling in BCs than the original LPrA2. During this study, we first tested toxicity of LPrA2 like peptides in MCF10-A cell-line. Then we tested the effectiveness to inhibit leptin induce S-phase in BCs and compared them to LPrA2. M-468 BCs were treated with various compounds for 48 hours. Cell cycle staging was determined using a cellometer.

Our results show that two of the new peptide antagonists were more effective in S-phase inhibition than LPrA2. We also expect that a combination of the new peptide antagonist and chemotherapy drug will inhibit proliferation in vitro.

This study could lead to the development of future studies that test the efficacy of C6 and C8 using an in vivo mouse model.

Key words: Leptin, Obesity, Breast Cancer, Leptin Antagonist (LPrA2)

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