Examining the Cell Cycle Effects of 3-Beta-hydroxybutyrate on MCF-7 Breast Cancer Cells
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The Ketogenic diet (KD) has recently rose to popularity in the past few years, but it is not a new dietary intervention. It was first developed in the 1920s as a nutritional intervention for patients with epilepsy and treatment for associated seizures. The diet comprises approximately 90% of dietary calories from fat, 8% from protein, and 2% from carbohydrates. It is currently being studied as a potential nutritional approach in cancer patients. The reason being is built on the important differences in metabolism between healthy cells and cancer cells. Under normal conditions, healthy cells in “resting” conditions will rely mostly on fat for energy production via specifically aerobic metabolism. Cancer cells possess a unique metabolism, a reliance on glucose and glycolysis for energy, which is known as the Warburg shift and represents one of the hallmarks of cancer. Given that the KD severely limits carbohydrate availability it represents a potential therapy with an increase in circulating ketones, the most abundant being beta-hydroxybutyrate (βHB), serving as metabolites for energy production in the mitochondria. These ketone bodies may reverse the Warburg effect by forcing the cell to utilize aerobic metabolism for energy production. My studies utilize MCF7 cells, a representative breast cancer cell line, and preliminary data shows that when insulin levels are decreased, there are concomitant alterations in proteins that regulate cell signalling pathways and proliferation. In the presence of high glucose, βHB had little effect on cellular phenotype, as expected. The next steps will examine the effects of βHB when insulin and glucose levels are reduced, a condition that mimics the effects of the KD on circulating blood levels of these 2 variables.