Synovial Sarcoma, a soft tissue sarcoma, has limited effective chemotherapy treatments with approximately half of synovial sarcoma patients developing metastasis by year 5. The presence of the t(X;18) translocation forms the fusion oncogene SS18-SSX, a characteristic trait of synovial sarcomas. Along with the detection of the fusion gene SS18-SSX, BCL2 has been seen to be upregulated in synovial sarcoma. BCL2 family members regulate apoptosis via feedforward mechanisms, and it then becomes a balancing act between BCL2 family member genes and pro-apoptotic genes. The initial thought was that targeting BCL2 specifically would shift the balance in favor of the pro-apoptotic genes which would then lead to cell death and tumor reduction. While disrupting BCL2 genetically and pharmacologically did not produce the desired results, targeting BCL-XL with the small molecule inhibitor BXI-72 showed a significant decrease in tumor size and an increase in apoptosis. Thus, we believe that BCL-XL is a better target for the treatment of synovial sarcomagenesis by shifting the balance to favor apoptosis.