Pancreatic cancer is the fourth leading cause of cancer-related death in the United States with an estimated 37,390 deaths expected to occur in 2012. The prognosis is very poor due to the recurrence and metastasis of the cancer with a 6% five-year survival rate for all stages combined. This study examined the effectiveness of dimethylamino-parthenolide (DMAPT) as a radiosensitizer to the human pancreatic cancer PaCa2 cell line. It is hypothesized that DMAPT, a bioavailable drug derived from parthenolide, will inhibit the activation of NF-κB and enhance radiation-induced cell killing of PaCa2 cells. NF-κB is a transcription factor that promotes cell survival, tumor progression, and angiogenesis and reduces susceptibility to apoptosis. The results show that DMAPT was toxic to the PaCa2 cell line. As a result, DMAPT suppressed cell growth and increased the doubling time of PaCa2 cells. The combination of 4μM DMAPT and radiation decreased cell survival. The PaCa2 cell line is radiosensitized by DMAPT but further investigation is required to determine the mechanism through which DMAPT works.