Angiogenesis is a mechanism necessary for the expansion and progression of tumors. Understanding the mechanism of angiogenesis in PMP allows us to select therapeutic targets that may be used in place of or with existing anti-angiogenic therapy (Bevacizumab). We have shown that CEA, a common glycoprotein produced by PMP, interacts with immune cells (macrophages) in the peritoneal cavity and produces specific cytokines (IL-6, IL-8) that are pro-angiogenic. The interaction of CEA with these cells may be an alternate target to Vascular Endothelial Cell Growth Factor (VEGF) and may be effective in reducing angiogenesis. We have also developed patient-derived xenograft (PDX) models of PMP in mice and have shown they behave similarly to that seen in humans. These will be essential for pre-clinical studies of novel anti-angiogenic therapies.