Overview: Of the many lifestyle habits that are associated with cancer development, various epidemiological observations have provided a strong link between increased alcohol consumption and breast cancer progression. Specifically, this observation was made in women on estrogen replacement therapy thus suggesting a possible synergistic link between alcohol and estrogen.

Method: For our current topic, Tg-1 murine mammary carcinoma cells were treated with varying concentrations of alcohol +/- estrogen and analyzed for angiogenesis markers, such as VEGF and eNOS, and the pro-proliferation marker, MEK. To further characterize neovascularization, we performed a scratch wound assay using the murine endothelial cell line, SVEC4-10, which was cultured in Tg1-1 conditioned media. SVEC4-10, treated with Tg-1-1 conditioned media were processed for Western blot analyses for MEK expression.

Result: After the treatment with alcohol and estrogen for 24 hour, VEGF and eNOS were upregulated in response to both estrogen and alcohol compared to untreated cells and cells treated with either alcohol or estrogen alone. MEK levels remained constant through the conditions with a marginal upregulation in presence of alcohol and estrogen when compared to untreated cells. The scratch wound assay results indicated enhanced migration of endothelial cells in response to conditioned media from Tg1-1 cells cultured in the presence of both alcohol and estrogen. MEK was upregulated in SVEC4-10 cells cultured in the media obtained from cells cultured with both alcohol and estrogen, indicating enhanced proliferation. The western blot results indicate a pro-neovasculargenic effect elicited by alcohol and estrogen, as determined by enhanced proliferation and migration of endothelial as well as increased expression of the pro-vasculogenic markers, VEGF and eNOS.

Conclusion: The identification of these cellular and metabolic markers will establish a biochemical link between estrogen activity and alcohol, could signal transduction induced cellular effects.