

P037 The effect of estrogen receptor status and *RAD51* overexpression on tumor progression

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RAD51 maintains genomic integrity by repairing DNA double-strand breaks (DSBs) via homologous recombination (HR). Its expression is tightly regulated and a base line level is required for proficient DNA repair. Surprisingly, overexpression of *RAD51* leads to induction of alternative DSB repair pathways, such as HR by gene conversion and single-strand annealing of complementary DNA strands, which has been correlated with chemo-/radio-resistance. *RAD51* overexpression—found in a wide-variety of tumors—induces hyper-HR which leads to genomic instability. We study the effects of *RAD51* overexpression on the development and progression of breast carcinomas. We hypothesize that overexpression of *RAD51* in breast carcinomas may be as much of a determining factor in tumor progression and chemo-/radio-sensitivity as estrogen receptor (ER) status. For our studies, we will employ established breast carcinoma cells that are either ER+ or ER-, and express normal or elevated *RAD51*. The cells will be used to develop xenograft tumor models to compare the rate of tumor growth and chemo-/radio-sensitivity between ER+ and ER- tumors which express normal or elevated *RAD51*. Our studies can provide vital information to enable the *RAD51* DNA repair pathway to be used as a target to improve the efficacy of cancer treatment regimens for resistant forms of breast cancer.