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HuR overexpression in MB231 breast cancer cells

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Cancer cells share acquired capabilities necessary for their malignant transformation. These "hallmarks of cancer" include increased proliferation, self-sufficiency in growth signals, insensitivity to antigrowth signals, evasion of apoptosis, angiogenesis and metastasis (Hanahan and Weinberg 2000). HuR is a RNA-binding protein which has been implicated in regulating mRNAs involved in each of these characteristics. We hypothesize that HuR maintains the growth characteristics of malignant cancer cells through the stabilization and increased translation of cancer relevant genes. If HuR does enhance malignancy then the overexpression of HuR would amplify the capabilites of malignant cancer cells and increase cell proliferation. This hypothesis was tested by creating a breast cancer cell line that stably overexpresses HuR. A vector overexpressing HuR was created by ligating a PCR amplified insert containing HuR and a HA hemagluttin tag into a Zeocin resistant episomal plasmid. Cells normally express HuR, so the tag was used to distinguish the overexpressed HuR from endogenous HuR. This plasmid was used to transfect MB-231 estrogen receptor-negative breast cancer cells. After transfection, Zeocin selected against the cells that did not incorporate the plasmid. Western Blots for the surviving cells revealed that HA HuR was expressed, implying that the cells were overexpressing HuR. Proliferation assays of heterogenous populations of both HA HuR-containing and normal MB231 cells yield no difference in cell division. Further experiments will use homogenous populations that highly overexpress HuR to see if HuR overexpression alters the proliferation and cell cycle capabilities of these cells. References: "Hallmarks of Cancer" Hanahan, Douglas and Weinberg, Robert A. Cell. Vol. 100, 57-70. 2000