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| **Poster Presentations - Tumor Vascular Biology** |

**Abstract 3490: Identification and characterization of novel galectin-9 splice variants in endothelial cells**

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Evidence is accumulating that endothelial galectins play an important role in tumor angiogenesis. Recently, we reported altered galectin-9 expression during endothelial cell activation. Here, we further assessed the expression and function of galectin-9 in endothelial cells. Using cDNA flanking PCR and subsequent cloning we identified 5 different galectin-9 splice variants in endothelial cells, 2 of which result in truncation of the C-terminal carbohydrate recognition domain and one of which has not been described previously i.e. galectin-95/6/10. By real-time PCR analysis we confirmed the expression of these 5 splice variants in endothelial cells and found that galectin-95 is the dominant splice variant in endothelial cells, as has been described for most other cell types. Endothelial cell activation resulted in a decreased expression of total galectin-9 levels which was mainly due to a decrease in galectin-95 expression. The expression of 2 other splice variants, galectin-95/6 and galectin-95/6/10 was retained after cell activation. To study the functional consequences of alternative splicing patterns on endothelial cell function, we cloned the 5 endothelial galectin-9 splice variants into expression constructs. We were able to demonstrate efficient transcription/translation of galectin-9 splice variants by real time PCR and Western blot respectively, following transfection of cell lines from non-endothelial (HEK293T) and endothelial (HMEC) origin. These data confirmed that exclusion of exon 10 causes a frameshift and premature stopcodon, resulting in the generation of protein that lacks the C-terminal CRD. Next, we assessed the function of gal-9 splice variants in endothelial cells by determining the effects on endothelial cell proliferation, migration and sprouting. Finally, we explored whether these splice variants have diverging roles in regulating the interaction between endothelial cells and immune cells. In conclusion, we report that endothelial cells express 5 splice variants of gal-9, one of which has not been described previously, and we show that these splice variants have diverging roles in endothelial cell biology. We hypothesize that interfering with the function of endothelial gal-9 splice variants might be an interesting approach for novel anti-cancer therapies.

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