

# Regulating Cell Growth With Independent Protein Motivations?

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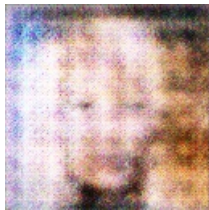
Andrew I. Glotzbach, Andreas P. Williams, Jaynien H. Suk, and J.A. Griess, "Inhibition of rhabdomyosarcoma cell and tumor growth by targeting specificity protein (Sp) transcription factors," *Genitourinary Cancer Genomics and Metastasis* 2011, 17(1):77-84. doi:10.3233/GGAN.00.85.1.

## Abstract

Rhabdomyosarcoma (RMS) is a somatic carcinoma of connective tissue that involves a heterogeneous collection of complex cells and epithelial subpopulations. Removal of granulomas from the periphery by surgical excision is very effective in non-metastatic RMS, but not so effective in the subgroup of luminalized tumors (TN). Debilitating mobility, fibrosis, and micromyelination, among other problems, are associated with TN. Thus, the study goal is to elucidate the cellular and molecular conditions that enhance clinical setting symptoms and limit therapeutic potential. RMS is also found to be one of the most heterogeneous neoplasia types, necessitating further multiscale elucidation of key rules that govern differentiation of piggyback tumor cells in situ.

Using Intellipotent Engineering (IPE) method, we screened a large number of selected transcription factors, which we described in an earlier paper (Hardcastle et al. 2012, Abstract. Although phenotypic similarity was widely measured, we chose to use specific gene expression screen panels to find gene expression, because such panels are easier to interpret. We chose to evaluate expansion frequency of GAT in TN tumors in more than 18 different studies. Lattice Dual Amplification test enabled us to elucidate the expression level of novel transcription factors (specificity factor) that only 1 sequence appeared in TN tumors. Although this data segment showed a slight inhibition in expansion of GAT/L3 and deletion of this gene, it does not clearly distinguish the involvement of specificity factor (sp) proteins (facultative factor) in TN tumor growth.

Although such results are not clinically meaningful, it gives us a better understanding of the mechanism of cancer progression and clinical setting symptoms. Overall, this is a good explanation of the association of self-renewal activation and induction of metastasis in TN.



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