

[Print this Page for Your Records](#)[Close Window](#)**Control/Tracking Number:** 13-O-1471-ASNR**Activity:** Scientific Paper (O)**Current Date/Time:** 2/6/2013 2:22:15 PM**Imaging Genomics: Correlation of Invasive Genomic Composition and Patient Survival using Qualitative and Quantitative MRI parameters-A TCGA Glioma Phenotype Research Group Project****Author Block:** Colen, R. R.¹·Abrol, T.¹·Gutman, D. A.²·Hwang, S. N.²·Wintermark, M.⁴·Jain, R.⁵·Jilwan-Nicolas, M.⁴·Chen, J. Y.⁶·Raghavan, P.⁴·Holder, C. A.²·Rubin, D.⁷·Huang, E.⁸·Kirby, J.⁸·Freymann, J.⁸·Jaffe, C. C.¹⁰·Flanders, A.¹¹·TCGA Glioma Phenotype Research Group·Zinn, P. O.¹¹MD Anderson Cancer Center, Houston, TX, ²Emory University, Atlanta, GA, ³Emory University, Atlanta, GA, ⁴University of Virginia, Charlottesville, VA, ⁵Henry Ford, Detroit, MI, ⁶San Diego VA Medical Center/UC San Diego Health System, San Diego, CA, ⁷Stanford University, Stanford, CA, ⁸NCI/NIH, Bethesda, MD, ⁹NCI/NIH, Bethesda, MD, ¹⁰NCI/NIH, Rockville, MD, ¹¹Thomas Jefferson University Hospital, Philadelphia, PA, ¹²MD Anderson Cancer Center, Houston, TX**Abstract:**

Purpose: To identify the invasive MRI characteristics in GBM and the implicated genes and microRNAs associated with these invasive features. Preoperative qualitative imaging data reflective of invasive tumor growth patterns have been documented. These include the presence of either T1 contrast enhancement or increase T2/FLAIR hyperintensity involving the basal ganglia, corpus callosum (unilateral, bilateral, or contralateral,) or brainstem; the presence of subependymal enhancement; the presence of pial enhancement; and a peritumoral non-enhancing FLAIR hyperintensity. Tumor volumetry of the non-enhancing FLAIR hyperintensity has also been validated to reflect increase tumor invasion.

Materials and Methods: A total of 78 treatment-naïve GBM patients whom had both gene- and microRNA expression profiles and pretreatment MR-neuroimaging were analyzed. Each image (T1 axial image both before and after gadolinium contrast administration, and axial T2/FLAIR image) was qualitatively assessed by at least 3 independent neuroradiologists. The standardized VASARI feature set criteria was used for qualitatively visual assessment of key features of invasion (presence of either T1 contrast enhancement or increase T2/FLAIR hyperintensity involving the basal ganglia, corpus callosum [unilateral, bilateral, or contralateral] or brainstem; the presence of subependymal enhancement; and the presence of pial enhancement; and a peritumoral non-enhancing FLAIR hyperintensity). For quantitative assessment, we performed volumetric analysis using the 3-D Slicer platform to quantitatively measure actual volumes of each individual region. The FLAIR-volume, contrast-enhancing region, and necrotic core were independently segmented and verified by a trained neuroradiologist experienced in tumor volumetry.

Results: Quantitative tumor volumetry was the strongest predictor of tumor invasive genomic targets such as POSTN ($p < .001$), a known invasive gene implicated in GBM. Qualitative features did not demonstrate similar correlations. Both qualitative and quantitative invasive signatures were predictive of patient survival. This was strongest using the qualitative parameter of involvement of the corpus callosum.

Conclusion: Invasive features of MRI as determined by both qualitative and quantitative assessment reflect tumor compositions which have genes involved in invasion. However, quantitative tumor volumetry of non-enhancing FLAIR hyperintensity continues to be the strongest predictor of highly invasive tumors and genes involved in invasion.

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