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[Fulltext](#) | [PDF \(2.57 M\)](#)**IDENTIFICATION OF THE DELETED IN LIVER CANCER 1 GENE, DLC1, AS A CANDIDATE MENINGIOMA TUMOR SUPPRESSOR.****CLINICOPATHOLOGICAL STUDY**

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*Hankins, Gerald R. Ph.D.; Sasaki, Tsutomu M.D.; Lieu, Ann-Shung M.D.; Saulle, Dwight B.S.; Karimi, Kambiz Ph.D.; Li, Jin Zhong D.V.M., Ph.D.; Helm, Gregory A. M.D., Ph.D.***Abstract:**

**OBJECTIVE:** Meningiomas are the second most common primary tumors of the central nervous system. Meningiomas at the cranial base pose technical challenges and result in increased morbidity. To investigate the molecular mechanisms of meningioma formation, the expression profiles of 12 000 genes from meningiomas and dural specimens were compared.

**METHODS:** Ribonucleic acid from 6 meningiomas (World Health Organization Grade I) and 4 dural specimens was profiled using U95A GeneChips (Affymetrix, Inc., Santa Clara, CA). Expression profiles of the 2 groups were compared using dChip and Data Mining Tool software packages (Affymetrix, Inc.) to identify differentially expressed genes. Down-regulation of a differentially expressed tumor suppressor gene, deleted in liver cancer 1 (DLC1), was verified by quantitative real-time reverse transcription-polymerase chain reaction and immunohistochemical staining. Function and methylation of DLC1 were assessed by ectopic expression in 5 primary cultures, demethylation assay using 5-aza-2'-deoxycytidine, and methylation-specific polymerase chain reaction in 4 meningioma samples.

**RESULTS:** Gene expression profiling revealed up-regulation of 5 genes (fibroblast growth factor 9, gibbon leukemia virus receptor 2, cyclin D1, eukaryotic translation initiation factor 5A, and 28S ribosomal ribonucleic acid) and down-regulation of 35 genes, including DLC1, in meningiomas. The down-regulation of DLC1 in meningiomas was confirmed by quantitative real-time reverse transcription-polymerase chain reaction and immunohistochemical staining. Transfection of DLC1 complementary deoxyribonucleic acid into primary cultures of 5 meningiomas resulted in decreased replication. Although demethylation decreased meningioma cell growth rates in vitro, methylation-specific polymerase chain reaction did not detect DLC1 promoter methylation.

**CONCLUSION:** The results suggest that DLC1 may function as a tumor suppressor gene in meningiomas. Furthermore, DLC1 promoter methylation does not appear to be responsible for the decreased DLC1 expression in these tumors.

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