Protein ZMYND8 tied to suppression of prostate cancer tumor metastasis

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Although it reads like European license plate number, a protein known as ZMYND8 has demonstrated its ability to block metastasis-linked genes in prostate cancer, according to a study at The University of Texas MD Anderson Cancer Center. The findings, resulting from cell line and mouse model studies, are published in the July 28 online issue of *Molecular Cell*.

"These findings are important as cancer metastasis is a complicated process and is both devastating and clinically challenging," said Min Gyu Lee, Ph.D., associate professor of Molecular and Cellular Oncology. "For metastasis, cancer cells acquire migratory and invasive abilities and so gaining new insight into how this occurs and how to stop metastasis is crucial. We believe this study opens a window into this process."

Lee's study centered on modification of proteins crucial to gene regulation, known as histones. Alterations in histone modifications, including acetylation and methylation, are frequently associated with cancer development. Lee's group looked at ZMYND8 as a histone "reader" that could possibly impact gene expression by recognizing these histone modifications known as histone "marks."

"It has been well documented that the effects of histone acetylation and methylation on gene expression can be mediated by specific binding proteins called 'readers,'" said Lee. "We identified ZMYND8 as a reader for histone marks called H3K4me1 and H3K14ac, both of which are tied to metastasis-linked genes."

The research group also noted that ZMYND8 cooperated with a type of histone mark "eraser" called JARID1D to suppress metastasis-linked genes.

"These findings are of special interest in light of our earlier study that JARID1D levels are lower in metastasized prostate tumors than in normal prostate and primary prostate tumors," said Lee. "This study revealed a previously unknown metastasis-suppressive mechanism in which ZMYND8 counteracts the expression of metastasis-linked genes by reading dual histone marks H3K4me1 and H3K14ac and cooperating with JARID1D."

Provided by University of Texas M. D. Anderson Cancer Center