97th AACR Annual Meeting
April 1-5, 2006
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Abstract Number:	2501
Presentation Title:	**DLC-1 tumor suppressor gene induces apoptosis in human non-small cell lung carcinoma cells following a unique process of cell morphological changes and protein nuclear translocation**
Presentation Start/End Time:	Monday, Apr 03, 2006, 1:00 PM - 5:00 PM
Location:	Exhibit Hall, Washington Convention Center
Poster Section:	2
Poster Board Number:	27
Author Block:	*Bao-Zhu Yuan*, *Amy M. Jefferson*, *Lyndell Millecchia*, *Steven H. Reynolds*. National Institute for Occupational Safety and Health, Morgantown, WV

The deleted in liver cancer (DLC-1) gene is a recently identified tumor suppressor gene for human non-small cell lung carcinoma (NSCLC). It can inhibit the growth and induce morphological changes of NSCLC cells in gene transfection studies. To explore the association between morphological change and DLC-1's tumor suppression function, we performed a further investigation utilizing gene transfection with a green fluorescent protein (GFP)-fused DLC-1 cDNA and laser scanning confocal microscopy. New studies revealed that DLC-1 transfection can initially induce multiple branched cytoplasmic extensions, which are followed by membrane blebbing along the cytoplasmic extensions and progressive cell shrinkage, and ended with cytoplasmic and nuclear decomposition, the morphological evidence of cell apoptosis. The process of morphological change is associated with RhoGAP-domain-related reduction of stress fibers and filopodia early in gene transfection and with collapsing of the actin cytoskeleton at the time of cell apoptosis. DLC-1's ability to induce tumor cell apoptosis is supported by the induction of caspase 3 activation in DLC-1-transfected cells following the morphological changes. Furthermore, the apoptosis is accompanied by DLC-1 protein nuclear translocation, which depends on a nuclear localization sequence (NLS). Gene deletion and NLS point mutation analyses suggest that DLC-1 can exert its tumor suppression function in both the cytoplasm and nucleus. However, the induction of cell apoptosis is more dependent on DLC-1 protein nuclear translocation, which could be retarded by active RhoA signaling. This study revealed several new features about DLC-1 which are important for understanding DLC-1's tumor suppression functions.

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