

LAPTM4B is associated with poor prognosis in NSCLC and promotes the NRF2-mediated stress response pathway in lung cancer cells

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We recently demonstrated that lysosomal protein transmembrane 4 beta (LAPTM4B) is elevated in non-small cell lung cancers (NSCLCs) and in the surrounding premalignant airway field of cancerization. In the present study, we sought to begin to understand the relevance of LAPTM4B [3] expression and signaling to NSCLC pathogenesis. In situ hybridization analysis of LAPTM4B transcript in tissue microarrays comprised of 368 NSCLCs demonstrated that LAPTM4B expression was significantly increased in smoker compared to non-smoker lung adenocarcinoma tumors ($P < 0.001$) and was significantly associated with poor overall survival ($P < 0.05$) in adenocarcinoma patients. Knockdown of LAPTM4B expression inhibited cell growth, induced cellular apoptosis and decreased cellular autophagy in serum starved lung cancer cells. Expression profiling coupled with pathways analysis revealed decreased activation of the nuclear factor erythroid 2-like 2 (NRF2) stress response pathway following LAPTM4B knockdown. Further analysis demonstrated that LAPTM4B augmented the expression and nuclear translocation of the NRF2 transcription factor following serum deprivation as well as increased the expression of NRF2 target genes such as heme oxygenase 1/HMOX1). Our study points to the relevance of LAPTM4B expression to NSCLC pathogenesis as well as to the probable role of LAPTM4B/NRF2 signaling in promoting lung cancer cell survival.

To read more, please click [here](#) [4].

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