ELEVATED LEVELS OF GITR (GLUCOCORTICOID-INDUCED TNFR-RELATED PROTEIN) AND TIM-3 (T CELL IMMUNOGLOBULIN- AND MUCIN-DOMAIN-CONTAINING MOLECULE-3) IN BRONCHOALVEOLAR LAVAGE FLUID (BALF) OF PATIENTS WITH ADVANCED NON-SMALL CELL LUNG CANCER

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GITR (Glucocorticoid-induced TNFR-related protein) is a member of the tumor necrosis factor receptor (TNF-R) and seems to interact with activated T-lymphocytes and endothelial cells and in the regulation of T-cell receptor of T-cell receptor-mediated cell death. TIM-3 (T cell immunoglobulin- and mucin-domain-containing molecule-3) is expressed on the surface of terminally differentiated Th1, regulates macrophages activation and inhibits anti-tumor immunity. There is no data about concentration of GITR and TIM-3 in BALF of NSCLC (Non-Small Cell Lung Cancer) patients. To study this issue, we measured BALF (Elisa) levels of GITR and TIM-3 in 44 NSCLC patients (before chemotherapy) and 15 healthy subjects. Both GITR and TIM-3 concentrations were elevated in BALF of NSCLC group compared with control [GITR: 53.44 (29.61-146) vs 43.12 (24.79-52.12) pg/ml, p=0.0002; TIM-3: 83.84 (38.34-228) vs 63.43 (35.86-81.32) pg/ml, p=0.0008). Receiver-operating characteristic (ROC) curves were applied to find the cut-off the BALF levels of GITR and TIM-3 (NSCLC vs Healthy: GITR =43.12 pg/ml, TIM-3 =63.43 pg/ml). We did not find any correlation between the BALF levels of GITR, TIM-3, and the stage of tumor or treatment response (prospectively). There was a positive correlation between the BALF level of GITR and time to tumor progression (r=0.518, p=0.04). We conclude that GITR and TIM-3 may play important role in carcinogenesis of NSCLC.