		Tumor							PLR	
		(vs.	Stage I BD		Differentiation			PAI	(vs. no-	PSI
Protein	Spot	normal)	(vs. III)	(vs. BA)	Poor	Well	Moderate	(vs no-PAI)	PLR)	(vs. no-PSI)
name	No.	(90 vs. 10)*	(62 vs. 28)	(72 vs. 14)	(20)	(22)	(48)	(15 vs 75)	(39 vs. 51)	(11 vs. 79)
ANXA1	1245	$+ 0.02^{\dagger}$								
GRK4	477	+ 0.002								
KRT19	1955	+ 0.016								
KRT7	1968	+ 0.049								- 0.025
ACTB/G	634	- <0.001								
FGF4	1331	- <0.001						+ 0.024	- 0.031	
MYCN	1268	- <0.001				- 0.028				
AAT	1413	- <0.001								
GFAP	2336		+ 0.007	+ 0.008	+ 0.003		- 0.003	+ 0.024		
GSTP1	1298		+ 0.027							
ANXA8	923		- 0.001							
KRT1	1888			+ 0.038						
PDIR5	513			+ 0.046						
SOD2	1328			+ 0.005						+ 0.001
KRT7	691			- 0.022					- 0.011	
RAB7	1262			- 0.031						- 0.017
EPHX1	891				- 0.031		+ 0.031			
ENO1B	627							+ 0.023	- 0.030	
PGAM1	1160							+ 0.037		+ 0.008

Table 5. Relationships between clinical-pathological variables and survival-associated protein expression: *P* value and direction of association

+, Significantly increased; -, significantly decreased. BD, bronchial-derived; BA, bronchoalveolar; PAI, presence of angiolymphatic invasion; PLR, presence of lymphocytic response; PSI, pleural surface involvement. PBP (1546#) was significantly decreased based on smoking status (vs non-smoking, P = 0.044) and presence of K-*ras* mutation (P = 0.016). CALU (1738#) was significantly increased in the presence of K-*ras* mutation (P = 0.046) and p53 accumulation (P = 0.029). Other known survival-associated proteins did not change in all these variables.

*Number in parentheses indicates number of samples used in each group.

[†]Significantly increased in tumor compared to normal lung with *P* value = 0.02 based on F test.