

Figure S1. Cell cycle analysis and miR-516b-5p overexpression. (A) FEZF1-AS1-knockdown induced G<sub>2</sub>/M arrest in both H1299 and H520 cells. (B) ITGA11-knockdown did not influence the cell cycle. (C) miR-516b-5p was overexpressed in H1299 and H520 cells using miR-516b mimics. Data are presented as the mean  $\pm$  SEM. \*\*\*P<0.001 vs. NC. ITGA11, integrin subunit  $\alpha$ 11; NC, negative control; si, small interfering; FEZF1-AS1, FEZ family zinc finger 1 antisense RNA 1; miR, microRNA; FC, fold-change.

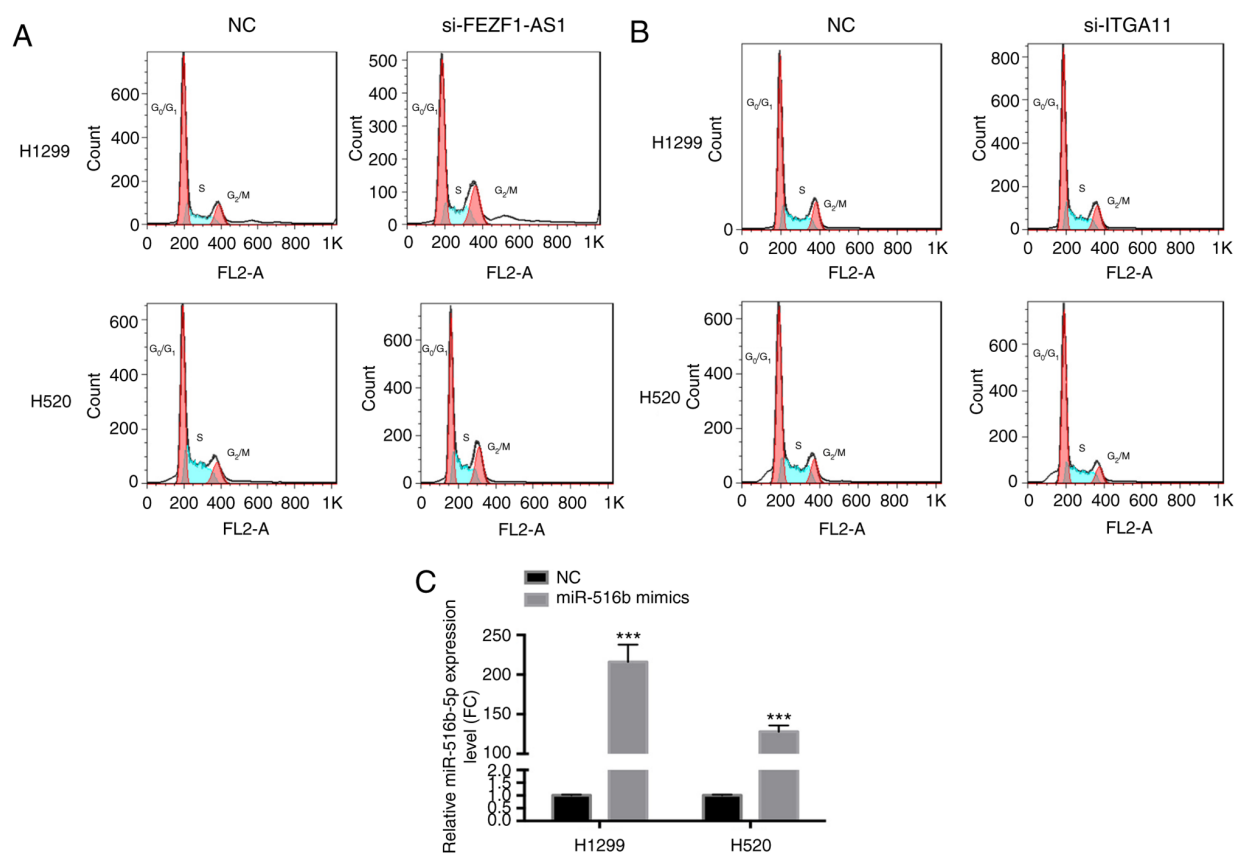




Table SI. Details of the pathology, smoking history, molecular classification (EGFR, ALK, PD-1 and PD-L1) of the 45 patients with non-small cell lung cancer.

Samples	FEZF1-AS1 expression <sup>a</sup>	Pathological diagnosis <sup>b</sup>	Smoking cigarettes/day	Smoking time, years	Smoking index <sup>c</sup>	Smoking <sup>c</sup>	Family history	EGFR mutation <sup>d</sup>	ALK <sup>d</sup>	PD-1, % <sup>e</sup>	PD-L1, % <sup>e</sup>
1	High	AD	10	30	300	1	No	/	/	/	/
2	High	AD	50	20	1,000	2	No	/	/	/	/
3	High	SCC	10	40	400	2	Yes	/	/	/	/
4	Low	AD	0	0	0	0	No	/	/	/	/
5	Low	AD	20	20	400	2	No	/	/	/	/
6	Low	SCC	0	0	0	0	No	/	/	/	/
7	High	AD	10	30	300	1	No	/	/	/	/
8	Low	AD	10	20	200	1	No	/	/	/	/
9	Low	Atypical	0	0	0	0	No	/	/	/	/
10	Low	SCC	20	40	800	2	No	/	/	/	/
11	Low	AD	20	40	800	2	No	/	/	/	/
12	High	AD	0	0	0	0	No	/	/	/	/
13	High	AD	0	0	0	0	No	/	/	/	/
14	Low	AD	0	0	0	0	No	/	/	/	/
15	Low	AD	0	0	0	0	No	/	/	/	/
16	High	AD	0	0	0	0	No	/	/	/	/
17	Low	AD	10	40	400	2	No	/	/	/	/
18	Low	SCC	20	40	800	2	No	/	/	/	/
19	Low	SCC	20	40	800	2	No	/	/	/	/
20	High	AD	40	20	800	2	No	/	/	/	/
21	High	SCC	20	30	600	2	No	/	/	/	/
22	Low	AD	40	40	1,600	2	No	-	-	0	0
23	High	SCC	0	0	0	0	No	-	-	0	0
24	High	AD	0	0	0	0	Yes	E19	-	0	0
25	Low	AD	20	30	600	2	No	/	/	/	/
26	High	SCC	60	30	1,800	2	No	-	-		
27	Low	AD	0	0	0	0	No	/	/	/	/
28	High	AD	20	30	600	2	No	/	/	/	/
29	High	SCC	40	35	1,400	2	No	-	-	0	40
30	High	AD	20	50	1,000	2	No	-	-	0	0
31	High	SCC	0	0	0	0	No	/	/	/	/
32	Low	AD	20	30	600	2	Yes	-	-	0	0
33	High	SCC	40	40	1,600	2	Yes	/	/	/	/
34	Low	SCC	60	30	1,800	2	No	/	/	/	/
35	Low	SCC	0	0	0	0	No	/	/	/	/
36	Low	AD	10	20	200	1	No	/	/	/	/
37	High	SCC	10	30	300	1	No	/	/	/	/
38	High	SCC	20	30	600	2	No	/	/	/	/
39	Low	SCC	30	30	900	2	No	/	/	/	/
40	High	SCC	20	40	800	2	Yes	/	/	/	/
41	Low	AD	10	40	400	2	No	/	/	/	/
42	Low	AD	40	30	1,200	2	No	/	/	/	/
43	High	AD	0	0	0	0	No	/	/	/	/
44	High	AD	20	20	400	2	No	/	/	/	/
45	High	AD	0	0	0	0	Yes	E21	/	/	/

<sup>a</sup>FEZF1-AS1 was divided into low and high expression groups based on the median of FEZF1-AS1 expression. <sup>b</sup>Samples included 17 SCC, 27 AD and 1 atypical carcinoid. <sup>c</sup>Smoking index=cigarettes/day x smoking time (years). Since a smoking index  $\geq 400$  indicated a high risk of lung cancer, patients were divided into never smokers (0, smoking index=0), light smokers (1, smoking index <400) and heavy smokers (2, smoking index  $\geq 400$ ). <sup>d</sup>EGFR was detected via PCR, and ALK was detected via fluorescence *in situ* hybridization by the Department of Pathology of the Fourth Hospital of Hebei Medical University. /, did not test; -, negative mutation. <sup>e</sup>Percentage of positive cells. PD-1 and PD-L1 were detected via immunohistochemistry by the Department of Pathology of the Fourth Hospital of Hebei Medical University. /, did not test; 0, no expression. These procedures were performed according to routine hospital protocols. FEZF1-AS1, FEZ family zinc finger 1 antisense RNA 1; ALK, anaplastic lymphoma kinase; PD-1, programmed cell death 1; PD-L1, programmed death-ligand 1; SCC, squamous cell carcinoma; AD, adenocarcinoma.