Supplementary Figure Legends

Supplementary Figure S1

Confirmation of the quality of tissues on tissue microarray (TMA). AE1/AE3 common cytokeratin and rabbit normal IgG were used as a positive and negative control, respectively.

Supplementary Figure S2

qRT-PCR analysis of the candidate gene in normal human organs. The relative expression level was normalized to the GAPDH level in each sample. Mean ± SEM for two experiments are shown.

Supplementary Figure S3

The 5-year survival rate for lung cancer patients with all histology and subsets of patients with adenocarcinoma (ADC). There is no significance between KIF11-H and KIF11-L in all histology (*P*=0.7693). Although it is not statistically significant, there is a tendency that patients with KIF11-H shows favorable survival compared with patients with KIF11-L (*P*=0.1104). Squares, high-level KIF11 expression (KIF11-H); Circles, low-level KIF11 expression (KIF11-L).

Supplementary Figure S4

The confirmation of the knockdown effect via HPPS-chol-siRNA-KIF11 in H460SM by quantitative RT-PCR (qRT-PCR) analysis *in vitro*. (left) HPPS-chol-siRNA-KIF11 reduced the KIF11 mRNA expression in 48 hours after transfection. The relative KIF11 expression level was normalized to the ACTB level in each sample. Mean ±SEM for two experiments are shown. (right) Effect of HPPS-chol-siRNA-KIF11 on lung cancer cell proliferation *in vitro*: H460SM cells were treated with or without HPPS-chol-siRNA-KIF11 and HPPS-chol-siRNA-scramble (600 nM) for 96 h, and cell viability was determined using a cell proliferation assay. Results shown are mean ± SD (bars) of three experiments.

Supplementary Figure S5

The relative changes in tumor volume after the last injection was significantly reduced in HPPS-chol-siRNA-KIF11 treatment group compared to control saline or HPPS-chol-siRNA-scramble groups (*P*<0.0001 by repeated measure one-way ANOVA).

Supplementary Figure S6

Synergetic effects of *KIF11* and *PLK1* siRNAs on H460 (upper) and SBC5 (lower) cancer cell proliferation *in vitro.* Cells were treated with each siRNAs for 96 h, and cell viability was determined using Cell Proliferation Assay. The maximum 60% (H460) and 91% (SBC5) synergetic growth inhibition effect of these combination when we compare them with Negative Control groups at different siRNA concentrations.  Results shown are mean ± SD (bars) of three experiments.

Supplementary Figure S7

Our proposed ‘patient-specific nanoparticle-based lung cancer therapy’. Based on the gene expression pattern from biopsy sampling of metastatic LNs using EBUS-TBNA, customized multiple patient-specific siRNAs will be conjugated to the nanoparticle.