**Supplementary Figure S1.** Flow cytometric histograms for PD-L1 expression at the surface of lung cancer cell lines positive (H2228, H3122) or negative for the *EML4-ALK* fusion gene.

**Supplementary Figure S2.** Effects of inhibitors of PI3K-AKT and MEK-ERK signaling on PD-L1 expression in Ba/F3 cells expressing *EML4-ALK*. Ba/F3 cells were transfected with an expression plasmid for EML4-ALK or the corresponding empty vector (mock), cultured for 6 h, and then incubated in the absence or presence of LY294002 (10 µM), U0126 (20 µM), or DMSO vehicle for 18 h. The abundance of PD-L1 mRNA in the cells was then determined by quantitative RT-PCR analysis. Normalized data are expressed relative to the value for mock-transfected cells, are means ± SD of triplicates, and are representative of three independent experiments.

**Supplementary Figure S3.** Effects of AKT and ERK depletion on PD-L1 expression in *EML4-ALK* fusion–positive (H3122) cells. *A*, H3122 cells were transfected with a nontargeting (NT) siRNA or siRNAs specific for AKT or ERK mRNAs, after which cell lysates were subjected to immunoblot analysis with antibodies to the indicated proteins. *B*, RT and real-time PCR analysis of PD-L1 mRNA in cells treated as in *A*. Normalized data are expressed relative to the value for NT siRNA–transfected cells, are means ± SD of triplicates, and are representative of three independent experiments. The siRNAs specific for AKT mRNA (AKT-1, 5ʹ-CCAGGUAUUUUGAUGAGGA-3ʹ; AKT-2, 5ʹ-CAACCGCCAUCCAGACUGU-3ʹ) or ERK mRNA (ERK-1, 5ʹ-CAAGAGGAUUGAAGUAGAA-3ʹ; ERK-2, 5ʹ-UCAGCCCCUUUGAGCACCA-3ʹ) were obtained from Nippon EGT. The data presented were obtained with AKT-1 and ERK-1 siRNAs, but similar results were obtained with AKT-2 and ERK-2 siRNAs.