Supplementary Figure Legends

Supplementary Figure S1. *In vitro* cell proliferation in galectin-1 knockdown of NSCLC. A, IHC staining was performed using an anti-galectin-1 antibody on a tissue microarray containing samples from 47 patients with different stages of adenocarcinoma. B, IHC staining was performed using an anti-galectin-1 antibody on a tissue microarray containing samples from 17 patients with different stages of squamous cell carcinomas. Galectni-1 expression levels were verified according to the score of tumor staining. C, Galectin-1 was knockdown in LLC-1 and A549 cell lines by lentiviral shGal-1 infection. Luciferase shRNA lentivirus (shLuc) was used as a control. The expression levels of galectin-1 were detected by RT-PCR. D, The effect of galectin-1 knockdown on cell proliferation *in vitro* was examined by MTS assay.

Supplementary Figure S2. Intracellular galectin-1 knockdown repressed COX-2 expression. A, mRNA levels of tumorigenic-associated genes in shLuc- or shGal-1-infected LLC-1 were detected using RT-PCR (left panel). The expression levels of galectin-1, COX-2 and VEGF were confirmed by RT-qPCR (right panel). B, The protein levels of COX-2 and PGE2 in shLuc- or shGal-1-infected LLC-1 cells were determined by Western blot and PGE2 EIA assay, respectively. C, A549/shGal-1 cells were transfected with pcDNA3 empty vector

(pc) or galectin-1 (Gal-1) plasmids. The RNA and protein levels of transfectants were analyzed by RT-PCR and Western blot. D, shLacZ- or shGal-1-infected A549 cells were cotransfected with pXC918-COX-2-luc and pRL-SV40. 24 hours later, these cells were treated with or without TGF-β1 for 48 hours. COX-2 promoter activity (RLU) was calculated as firefly luciferase activity/renilla luciferase activity.

Supplementary Figure S3. Effects of galectin-1 overexpression on tumor progression of H522 cells. A, H522 cells were transfected with galectin-1 (Gal-1) or pcDNA3 empty vector (pc). The expression levels of galectin-1 and COX-2 were detected using RT-PCR and Western blot. B, H522/pc or H522/Gal-1 cells (5×10^4) were seeded onto upper side of transwell inserts and incubated for 24 hours at 37°C to determine the migratory and invasive abilities. C, H522/pc or H522/Gal-1 cells (2000 cells/6-cm) were seeded in soft agar for evaluating anchorage-independent tumor growth.

Supplementary Figure S4. Effects of galectin-1 knockdown on tumor progression of PC-9 cells. A, Galectin-1 was knockdown in PC-9 cell line by lentiviral shGal-1 infection. The expression levels of galectin-1 and COX-2 were detected using RT-qPCR. B, The expression levels of galectin-1, COX-2, p-p38 MAPK, p-ERK and NF-κB-p65 were detected

in shLuc- or shGal-1-infected PC-9 cells using Western blot. C, shLuc- or shGal-1-infected PC-9 cells (5×10^4) were seeded onto upper side of transwell inserts and incubated for 24 hours at 37°C to determine the migratory and invasive abilities (top panels). shLuc- or shGal-1-infected PC-9 cells (2000 cells/6-well) were seeded in soft agar for evaluating anchorage-independent tumor growth (bottom panel).

Supplementary Figure S5. Clinical relevance between galectin-1 and COX-2 in lung cancer. A, Statistic analysis of galectin-1 expression was compared with COX-2 in lung cancer tissues (n = 82). B, IHC staining was performed using an antibody to COX-2 on tissue microarray containing samples from 47 patients with stage I (n = 17), stage II (n = 13) and stage III (n = 17) lung adenocarcinoma. C, Statistic analysis of galectin-1 expression was compared with COX-2 in squamous cell carcinoma tissues (n = 17). D, Statistic analysis of galectin-1 expression was performed in different stages of squamous cell carcinoma tissues (n = 17). E, Proposed model was showed how galectin-1 regulated progression of lung cancer. TGF-β1 induces H-Ras and galectin-1 association to activate both p38 MAPK and ERK1/2 pathways. Then, NF-κB, the common mediator of p38 MAPK and ERK1/2 pathway, would translocate to the nucleus and bind to COX-2 promoter to activate COX-2 transcription.