

### **Supplemental Figure legends.**

**Suppl. Fig. S1: Characterization of CSPG4 in Malignant Mesothelioma.** **A.** Lysates (50  $\mu$ g protein) from MM REN, Con, Phi, Rob, Gard, Hmeso, Gor and PPM-Mill cells and from HM cells were immunoblotted with CSPG4-specific mAb 763.74 or with GAPDH-specific mAb and detected by ECL. The CSPG4-positive Colo38 melanoma cells and the CSPG4-negative Burkitt's lymphoma cells Raji were used as positive and negative controls, respectively. **B.** Lysates (50  $\mu$ g protein) of HM cells, obtained from pleural effusions of different individuals were immunoblotted as in **A.**

### **Suppl. Fig. S2: Silencing of CSPG4 expression in MM cells.**

**A.** PPM-Mill and Hmeso cells were transfected with CSPG4-specific siRNAs or with scramble siRNA. The down-regulation of CSPG4 was monitored by testing the cell lysates with CSPG4-specific mAbs 763.74 in Western blotting. GAPDH was used as a loading control. The density of the bands corresponding to the CSPG4 components and to GAPDH was measured using densitometry (mean  $\pm$  SEM), PPM-Mill ( $1.039 \pm 0.02742$  n=3) and Hmeso ( $0.06872 \pm 0.008025$  n=3). The results are expressed as the ratio between CSPG4 and GAPDH band intensities. \* $P < 0.0005$ . **B.** CSPG4-positive MM cells, PPM-Mill and Hmeso, and CSPG4-negative MM cells, REN transfected with CSPG4-specific siRNA or with scramble siRNA and were cultured for 48 hours. Cell growth was determined by MTS assay (mean  $\pm$  SEM), PPM-Mill ( $82.62 \pm 1.359$ , n=6), Hmeso ( $84.12 \pm 1.055$  n=6) and REN ( $97.95 \pm 0.7352$  n=6). Asterisks indicate  $P < 0.05$  significance.

**Suppl. Fig. S3: The interaction between CSPG4 and the extracellular matrix (ECM)**

**promotes MM cell adhesion.**

**A.** MM cells were plated in serum-free medium on uncoated tissue culture dishes, or pre-coated with different ECM components such as CI, CIV and FN (at a concentration of 10 $\mu$ g/ml of ECM in PBS) for 48 hours. Microphotographs show monolayer culture morphology for the different treatments (magnification, 100X). ECM components enhance CSPG4 expression. The cellular proteins were extracted and analyzed by immunoblotting for CSPG4 expression; GAPDH was used as loading control. **B.** CSPG4-positive PPM-Mill and CSPG4-negative REN MM cells were treated daily with mAb TP41.2 or IgG control, seeded on CI- or FN-coated dishes and cultured for 72 hours. Then cell morphology was observed under the contrast-phase microscope (magnification 100X) and cell viability was determined by MTS assay. Asterisks indicate  $p < 0.005$  significance. All data represent the mean  $\pm$  SD of 3 independent experiments, performed in triplicate. (PPM-Mill  $P \leq 0.0001$ ; REN  $P = 0.746$ ).

**Suppl. Fig. S4: CSPG4 knockdown reduces MM cell migration.**

PPM-Mill and REN MM cells were transfected with CSPG4-specific siRNAs or with scramble siRNA. Cells were then plated on the membrane of the upper chamber of a Transwell® plate and grown for 24 hours in the presence of 10% FBS. The cells migrated to the lower surface of the membrane were stained with Giemsa, solubilized and the absorbance was measured at 560nm to determine the extent of cell migration. Asterisk indicates  $P = 0.0002$  significance.

**Suppl. Fig. S5: CSPG4-specific mAb TP41.2 reduces MM wound healing.**

Wound closure of PPM-Mill, Hmeso and REN, treated with mAb TP41.2 or with IgG control, was assessed following a 24 hour and 48 hour incubation. The wound closure extent (Image J software) is shown in the bar graph on the right. Asterisk indicates  $P \leq 0.05$ ).

**Suppl. Fig. S6: CSPG4 knockdown decreases the rate of wound healing in MM cells.**

Wound closure of PPM-Mill, Hmeso and REN, transfected with CSPG4-specific siRNA (siRNA) or with scramble control siRNA (scramble), was assessed 24 hours and 48 hours later. The wound closure extent (Image J software) is shown in the bar graph on the right. Asterisk indicates  $P \leq 0.05$ ).