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## **Supplementary figure legends**

Fig. S1. OPN provides cells an increased resistance to UV-induced apoptosis dependent on CD44 variant isoform. Subconfluent AZ521 clones harboring control plasmid or plasmids encoding specific CD44 isoforms were cultured in serum-free medium for 24 h, and incubated with or without added OPN (10  $\mu$ g/ml) for 4 h. Cells were then UV-irradiated at 90 J/m<sup>2</sup>, and harvested for apoptosis assay by flow cytometric analyses of sub-G1 fractions at designated time.

8 Fig. S2. Engagement of CD44 induces lipid raft coalescence. AZ521/CD44<sub>V6-10</sub> cells 9 were treated with or without OPN or Hermes-3 (H-3). The cells were lysed in chilled 1% 10 cold Triton X-100 buffer, and lysates were subjected to sucrose gradient fractionation as 11 described in the Materials and methods. A total of 10 fractions were collected from top to 12 bottom. The 2-4 fractions represent the lipid rafts. The relative protein distribution of the 13 low-density fractions (as pooled from fractions 2-4) isolated from AZ521/Mock and 14 AZ521/CD44<sub>V6-10</sub> cells treated with H-3 (as filled bars), OPN-treated (as hatched bar) and 15 untreated or control (as empty bars) cells was shown in a bar graph. Data from three separate experiments are collected and presented as mean  $\pm$  SD. \*\*, P < 0.01 by *t* test. 16

Fig. S3. Engagement of CD44 mutants that are defective in associating with lipid rafts fails to induce lipid raft reorganization. Individual AZ521/CD44 cell clones were treated with IgG or H-3 mAb as described. The cells were lysed in chilled 1% cold Triton X-100 buffer, and lysates were subjected to sucrose gradient fractionation as described. A total of 10 fractions were collected from top to bottom. The relative protein distribution of the low-density fractions (as pooled from fractions 2-4) was shown in a bar graph. Data from three separate experiments are presented as mean  $\pm$  SD. \*\*, P < 0.01 by *t* test.





