Supplemental figure legends

Supplemental figure 1. LSECtin is induced to be expressed by murine melanoma tissues. A, quantitative PCR analysis of LSECtin expression using B16 cells and melanoma cells isolated from tumor-bearing mice. *, \( P<0.05 \). Data are represented as the ratio normalized to GAPDH expression. B, flow cytometry analysis of LSECtin surface expression using B16 cells and melanoma cells isolated from tumor-bearing mice. White histograms demonstrated LSECtin surface expression on gated 7-AAD negative cells. Negative controls were shown as grey histograms.

Supplemental figure 2. Determination of concentration of IL-6 and IL-10 within tumor. Non-necrotic fresh tumor tissues isolated from B16-inoculated mice were cut into small pieces, homogenized in extraction buffer on ice, passed through a sieve. Then the homogenized solution was centrifuged and supernatant were evaluated for the local production of IL-6 and IL-10 as pg/g.

Supplemental figure 3. Confirmation of the surface expression of LSECtin on stable B16-mock and B16-LSECtin transfectants by flow cytometry. White histograms demonstrated LSECtin surface expression on gated 7-AAD negative cells. Negative controls were shown as grey histograms.

Supplemental figure 4. Validation of the specificity of anti-LSECtin. A, anti-LSECtin could bind with coated recombinant LSECtin protein in a dose-dependent way. B, binding activity of anti-LSECtin with LSECtin protein was measured by ELISA in the presence of competition protein at indicated concentrations. C. binding activity of anti-LSECtin with LSECtin expressed by B16-LSECtin and fresh melanoma cells isolated from B16-inoculated mice was measured by flow cytometry in the absence (black line) or presence (grey dotted line) of competition protein. Negative controls were shown as grey histograms.

Supplemental figure 5. Anti-LSECtin ameliorates B16 melanoma growth. WT
C57BL/6 mice were inoculated with $5 \times 10^4$ B16-LSECtin cells on day 0 and treated with 250 $\mu$g/ml of the indicated or control antibody intraperitoneally (i.p.) on day 3, 6, and 9. The LSECtin antibody significantly inhibited B16 tumor progression. **, $P<0.01$. Data represent the mean ± SEM of two different experiments.

Supplemental figure 6. Tumor-derived LSECtin stimulates MDSC to express iNOS and promotes MDSC accumulation in spleen, but not in tumor site. A, MDSC, sorted from splenocytes, were cocultured with irradiated B16-mock or B16-LSECtin cells. The level of iNOS production were determined using RT-PC. *, $P<0.05$. These results are representative of three independent experiments. B, C57BL/6 mice were inoculated with $2 \times 10^5$ B16-mock and B16-LSECtin cells. After two weeks, the percentages of CD11b$^+$Gr-1$^+$ cells of CD45$^+$ cells in spleen (left, *, $P<0.05$) and tumor (right) were determined by flow cytometry.

Supplemental figure 7. Confirmation of LAG-3 expression on T cells. CD3$^+$ T cells after sorted from C57BL/6 mice (black line) or after stimulated by anti-CD3 and anti-CD28 (heavy black line) were stained with APC-conjugated anti-LAG-3. TIL or TDLN cells (black line) isolated from tumor-bearing C57BL/6 mice were stained with APC-conjugated anti-LAG-3.