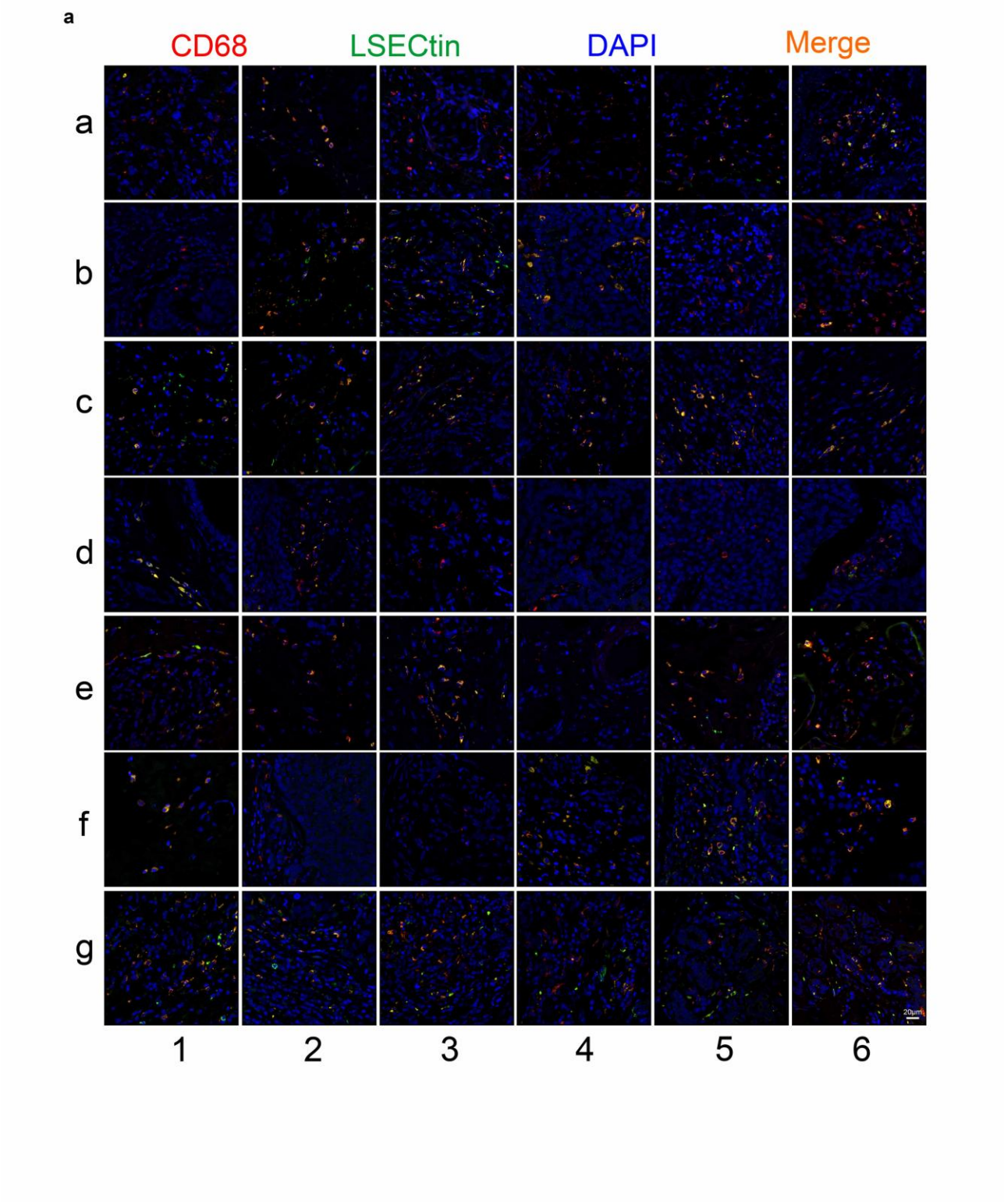
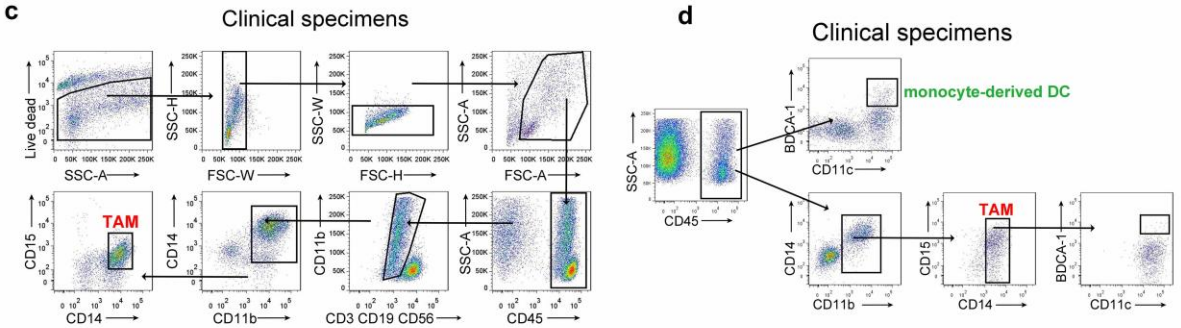
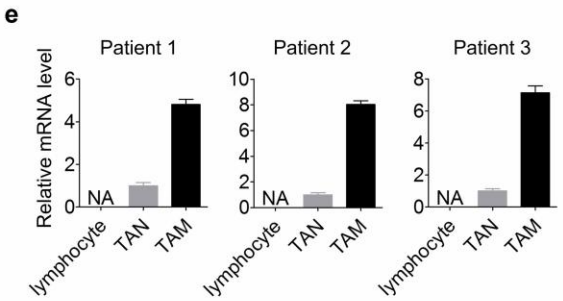
Supplementary information, Fig. S1

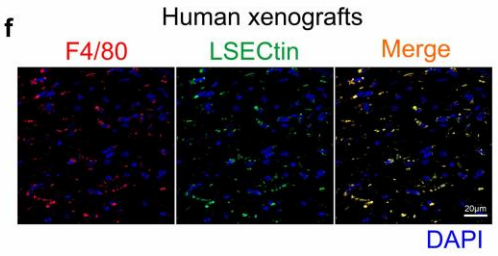


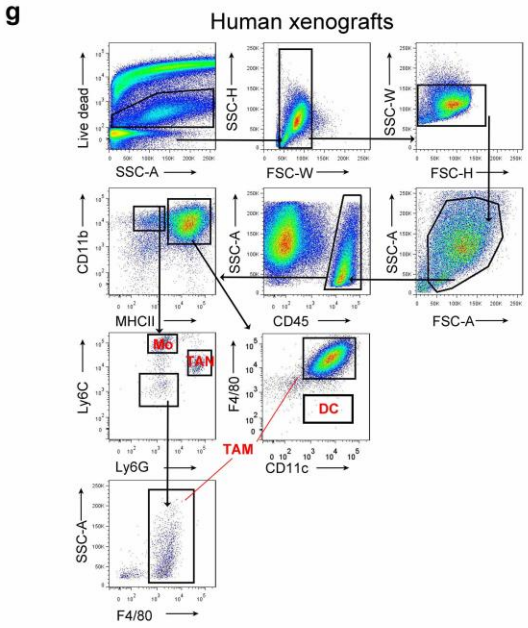
b

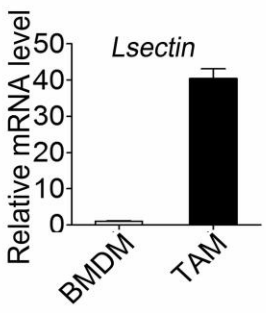
	LSECtin-positive samples	LSECtin/CD68 merge samples
Luminal A	6/10 (60%)	5/10 (50%)
Luminal B	10/14 (71%)	10/14 (71%)
HER2+	7/10 (70%)	7/10 (70%)
TNBC	8/8 (100%)	8/8 (100%)
Total	31/42 (74%)	30/42 (71%)

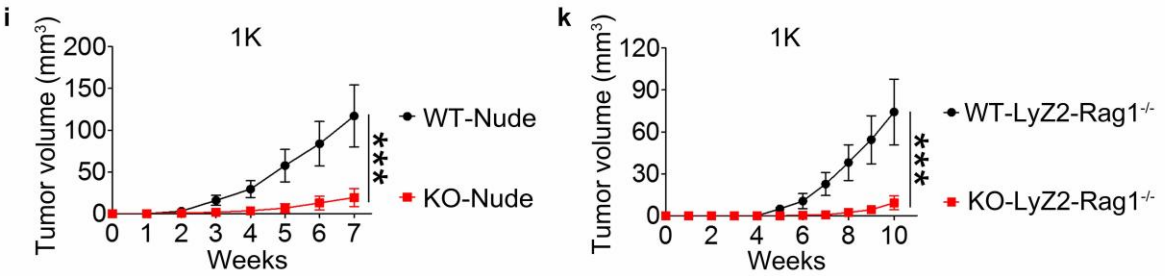


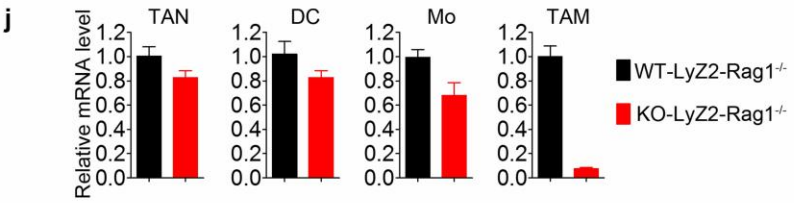


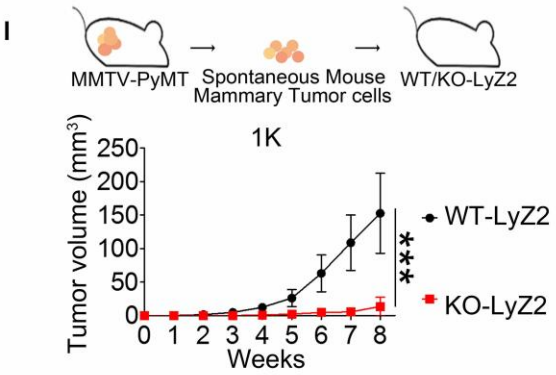




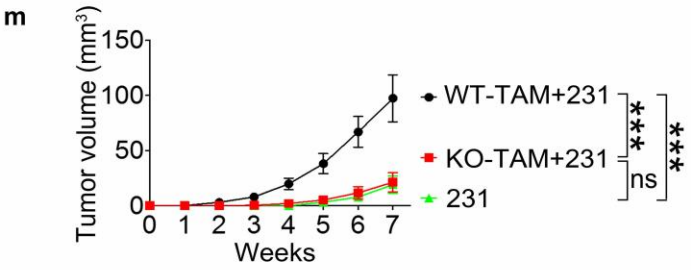


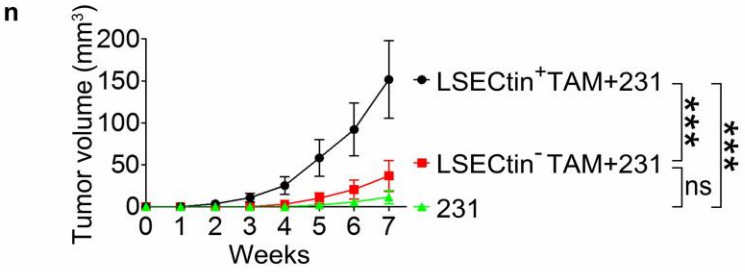






Group	100K	1K
WT-LyZ2	8/9 (89%)	5/10 (50%)
KO-LyZ2	10/11 (91%)	1/10 (10%)





Supplementary information, Fig. S1. LSECtin expressed on macrophages drives breast cancer growth

(a) Immunofluorescent staining of tumor tissues representing different types of breast cancer for CD68 (red) and LSECtin (green). A high proportion of CD68-positive cells are LSECtin-positive (scale bar=20 μm). Details regarding the tumor pathology are presented in Supplementary information, Table S1.

(b) The number and percentage of samples containing LSECtin and CD68 co-expressing cells in tumor sections from 42 breast cancer patients.

(c) Gating strategy for the identification of TAMs in the tumor tissues of different patients.

(d) Gating strategy for the identification of TAMs and monocyte-derived DCs. TAM subpopulation scarcely contains monocyte-derived DCs.

(e) Real-time PCR analysis showing the expression of *Lsectin* relative to that of the housekeeping gene *β-actin* in TAMs from breast cancer patients (3 pieces of tumor tissues for each patient). One of three experiments is shown.

(f) Immunofluorescent staining of human xenograft tumors for F4/80 (red) or LSECtin (green). A high proportion of F4/80-positive cells are LSECtin-positive (scale bar = 20 μm). One of three experiments is shown.

(g) Flow cytometric analysis on different myeloid cell populations showing the gating strategy for the identification of tumor-associated macrophages (TAM), tumor-associated neutrophils (TAN), monocytes (Mo) and dendritic cells (DC) in human xenograft tumors.

(h) Mouse Bone marrow-derived macrophages (BMDMs) or tumor extract solution educated BMDMs (induced TAMs) were used for real-time PCR analysis. *β-actin* served as a control. The levels of *Lsectin* were up-regulated in BMDMs after stimulation by tumor extract solution. One of three experiments is shown.

(i,k) 1×10^3 231 cells were injected into KO-Nude mice or littermate controls (n=15 each) (i); into KO-LyZ2-Rag1$^{-/-}$ mice (n=20) or littermate controls (n=23) (k). The tumor volume was monitored weekly.

(j) Real-time PCR analysis showing the expression of *Lsectin* relative to that of the housekeeping gene *β-actin* in TAN, DC, Mo or TAM from WT- LyZ2-Rag1$^{-/-}$ or KO-LyZ2-Rag1$^{-/-}$ models. The data from three mice were pooled.

(l) LSECtin specific knockout of TAMs delays tumor growth in the immunocompetent mouse allograft models. Primary mouse tumor cells were isolated from MMTV-PyMT mice and injected into macrophage-specific LSECtin knockout mice (LyZ2-Cre; LSECtin$^{fl/fl}$; KO-LyZ2 in short) or littermate controls (LSECtin$^{fl/fl}$; WT-LyZ2 in short). The tumor volume at 7 weeks after injection of 10^3 tumor cells is shown (left) (n=10 each). The table indicates the number of tumors initiated (right).

(m,n) 1×10^3 231 cells were injected with admixed 1×10^4 WT-TAMs or with admixed KO-TAMs (m); with admixed human primary LSECtin$^-$TAMs or with admixed LSECtin$^+$TAMs from surgical breast cancer specimens (n) into KO-Nude mice. The tumor volume of KO-nude mice injected with 1×10^3 231 cells without admixed TAMs served as controls. The tumor volume was monitored weekly. One of three experiments is shown.

Data are presented as the mean ± SEM. *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$ (two-way ANOVA test for i, k, h, l, m, n).