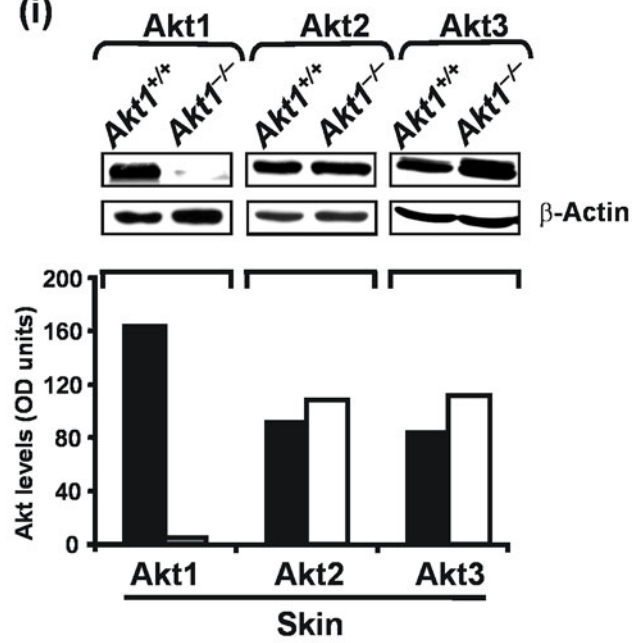
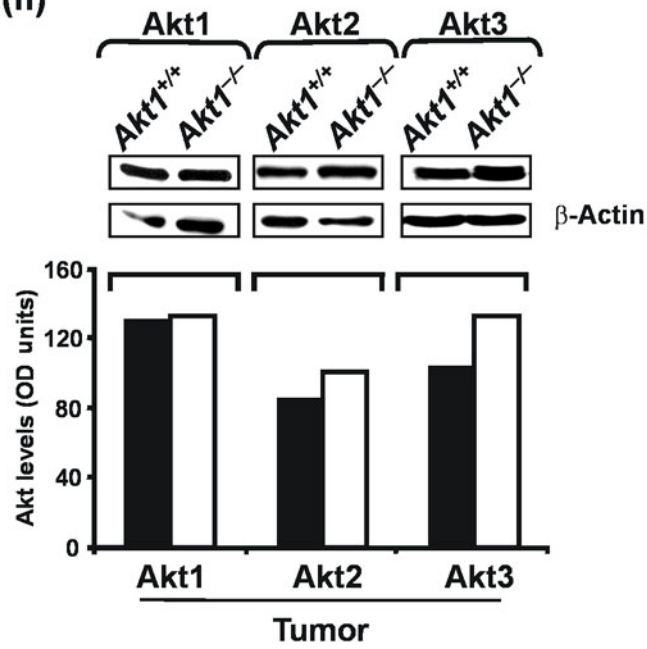
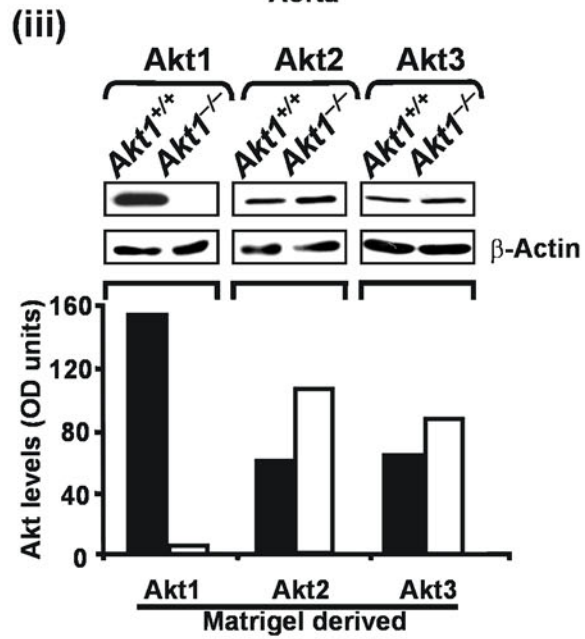
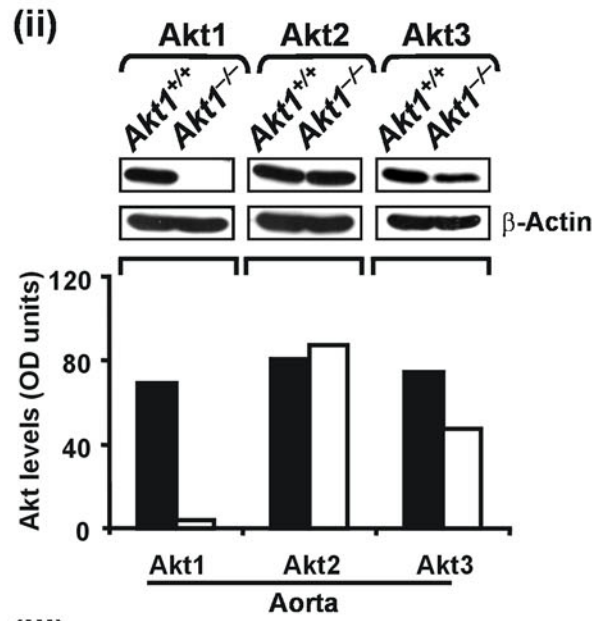
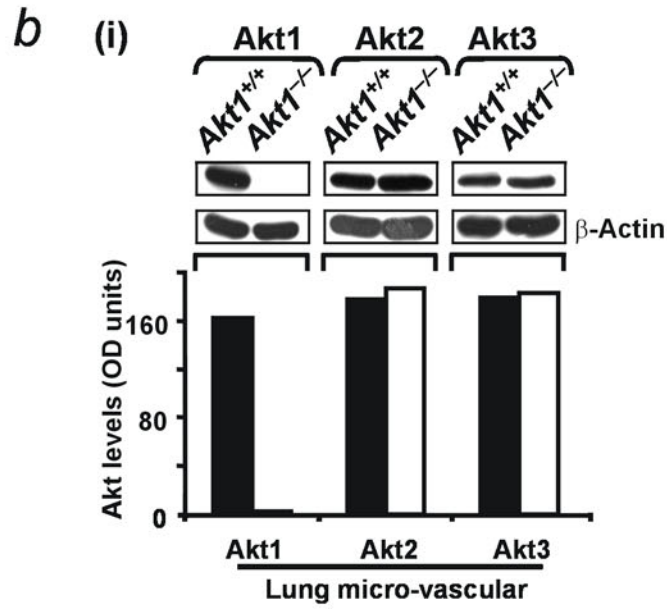


a (i)

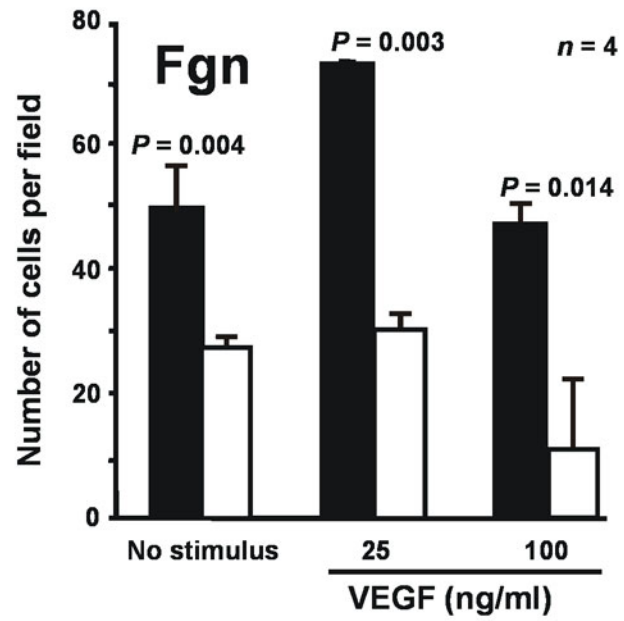


(ii)

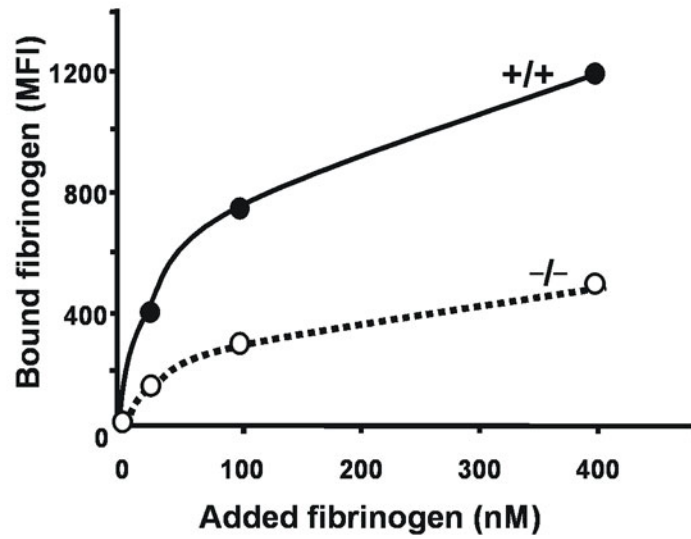




*C*



*d*



**Supplementary Fig. 1** *a*, Western blot analysis and band densitometry of different isoforms of Akt (Akt1, Akt2 and Akt3) in skin (i) and tumor (ii) of WT and Akt1<sup>-/-</sup> mice *b*, Western blot analysis of Akt isoforms expression in EC from lung (i), aorta (ii) and matrigel implants (iii) from WT and Akt1<sup>-/-</sup> mice *c*, Migration of WT (black bars) and Akt1<sup>-/-</sup> (white bars) EC toward fibrinogen was stimulated by 25 and 100 ng/ml of VEGF as indicated. *d*, Analysis of soluble ligand binding by WT (filled circles) and Akt1<sup>-/-</sup> (open circles) aortic EC was performed using FITC-fibrinogen. Isolated EC were incubated in suspension with increasing concentrations of FITC-fibrinogen in the presence of 0.5 mM of MnCl<sub>2</sub> and Mean of Fluorescence Intensity (MFI) was determined by FACS. Note an impaired binding of extracellular matrix to Akt1<sup>-/-</sup> EC.