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Supplemental Figure 1

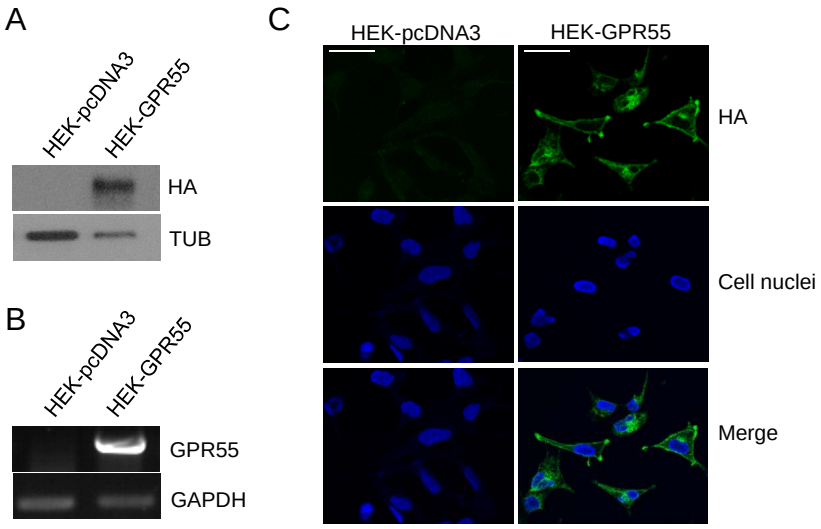

Figure S1. Characterization of GPR55-overexpressing HEK293 cells. HEK293 cells stably expressing 3xHA-GPR55 were previously generated (Henstridge *et al.*, 2009). (A) Western blot analysis of HA-tagged GPR55. Antibodies were: anti-HA (Cell Signaling Technology, Danvers, MA) and anti-α-tubulin (used as loading control, Sigma-Aldrich, St. Louis, MO). (B) GPR55 mRNA expression as determined by RT-PCR. Primers were: sense 5'- GTCCCCCTTCCCGTCCCTGTG-3' and antisense 5'- GCTGGCTGCGATGCTGTAGATGC-3'. GAPDH was used as internal control. (C) Immunofluorescence analysis of GPR55 expression by anti-HA staining (in green). Cell nuclei (in blue) were stained with Hoechst 33342 (Invitrogen, Carlsbad, CA). Scale bar, 4µm.

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Supplemental Figure 2

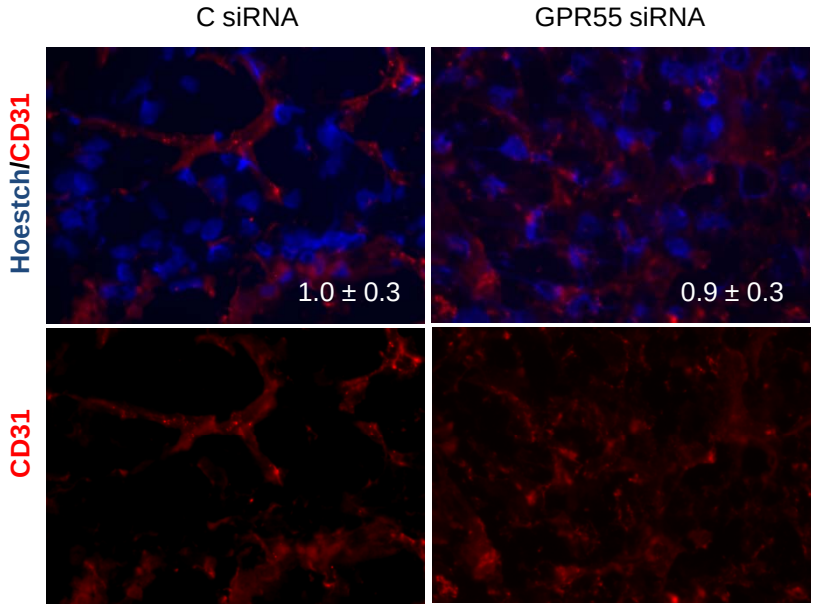

Figure S2. Immunofluorescence analysis of CD31 staining in T98 cells-derived xenografts. The potential antiangiogenic effect of GPR55 silencing *in vivo* was determined by immunofluorescence analysis of the vascular endothelial marker CD31. Tissue-tek embedded paraformaldehyde fixed tumor sections were incubated with anti-CD31 antibody (Pharmingen/BD Biosciences, San Jose, CA). The secondary anti-rabbit Alexa Fluor 594 antibody was from Invitrogen (Carlsbad, CA). Cell nuclei were stained with Hoechst 33342 (Invitrogen) and are shown in blue. Confocal fluorescence images were acquired using Laser Sharp 2000 software (Bio-Rad) and CD31 staining intensity (expressed in arbitrary units, mean ± s.e.m.) was calculated with ImageJ software (n= 4 animals per group, 8 sections per animal).

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Supplemental Figure 3

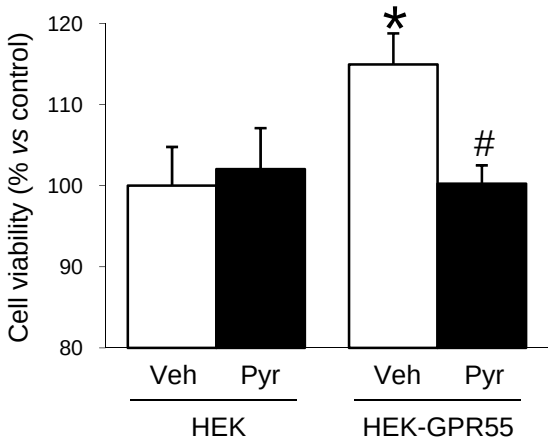

Figure S3. Inhibition of LPI synthesis prevents GPR55-induced increase in cell proliferation. HEK293 cells stably expressing 3xHA-GPR55 (HEK-GPR55) or the corresponding empty vector (HEK) were serum-starved overnight and incubated with the PLA_2 inhibitor pyrrophenone (Pyr, 1 μM, generously donated by Dr. Balsinde, Instituto de Biología y Genética Molecular, Valladolid, Spain) or vehicle (Veh) for 48 h. Cell viability was determined by the MTT test. Results are expressed as % *vs* the cell viability of vehicle-treated HEK293 cells, set at 100%. *, $p<0.05$ *vs* vehicle-treated HEK293 cells; #, $p<0.05$ *vs* vehicle-treated HEK293-GPR55 cells; n=3.