**Generation of genetically modified cells**

A431-CD47KO cell lines were generated by lentiviral transduction of pLentiCrispR-v2 – CD47KO (pLentiCrispR-v2 was a gift from Feng Zhang (Addgene plasmid #52961)); A431 cells expressing HER2/neu (A431HER2/neu) were generated by retroviral transduction, followed by positive selection based on puromycin resistance, as previously described (8). SKBR3-CD47KD cells were generated by lentiviral transduction of pLKO.1-puro – CD47KD. Transduced and afterwards puromycin selected cells showed a CD47 expression of 10-15% of normal. As control cell line a scrambled shRNA was used. Knockdown and knockout of antigens on cell lines was routinely verified by flow cytometry. Ba/F3 cells expressing EGFR were transfected with WT EGFR (Upstate) and EGFR expressing clones were selected using neomycin. Ba/F3 cells expressing HER2/neu were generated by retroviral transduction, followed by positive selection using puromycin resistance and limiting dilution.

**Antibodies and reagents**

SIRPα or CD47 were blocked using human SIRPα mAb 12C4 or F(ab’)2 fragments of human CD47 antibody B6H12 (both at 10 µg/mL), respectively (16). IgG trastuzumab (Roche), IgG cetuximab (Merck KGaA), anti-HER2-IgA2and anti-EGFR-IgA2) were generated as described (12) and used at a final concentration of 5 μg/mL, unless stated otherwise. To block mouse SIRPα we used the rat IgG2 antibody MY-1, generously gifted to us by the group of prof. dr. Takashi Matozaki (University of Kobe, Japan), at a final concentration of 10 μg/mL in our *in vitro* studies (31).

**Beads binding assay**

The SIRPα expressing murine macrophage cell line RAW 264.7 was used to show the blocking capabilities of the mouse anti-SIRPα antibody MY-1, using a beads binding assay. In short: 105 RAW 264.7 cells were seeded in a V shape 96 wells plate, and washed with PBS containing 0.1% BSA. Afterwards cells were incubated in PBS containing 0.1% BSA and 10 μg/mL anti-SIRPα blocking antibody (MY-1) and kept on ice for 30 minutes. Another vial containing 1 μg/mL mouse CD47-Fc together with 20μg/mL goat anti-human Alexa647 and 1% normal rat serum was incubated on ice for 30 minutes. Cells were washed with PBS containing 0.1% BSA, after which 50 μL mouse CD47 goat anti-human Alexa647-NRS complex was added to cells and kept on ice for 30 minutes. Finally cells were washed and binding was determined using flow cytometry.