V90lec13 were evaluated with a 4-hours Calcein-AM assay or with a 40/60-hours real-time cell assay against HER-2+ breast cancer cell lines. The concentration of cytokines produced upon the 20-hours co-culture of effector with target cells was assessed with a multiplex assay by flow cytometry analysis. The in vivo biodistribution of the fluorophore-conjugated HER2xCD3 bsAb was monitored in tumor bearing NSG mouse model.

**Results**
HER2xCD3 binds efficiently to CIK cells with the ScFv of CD3 and to cancer cells with the ScFv of HER-2 in a dose-dependent manner. The specific combination of HER2xCD3, TRS or TRS V90lec13 with CIK cells significantly enhances their anti-tumor activity against several breast cancer cell lines, compared to CIK cells alone, even at a very low effector/target ratio (0.1:1). Interestingly, TRS-resistant tumor cell lines show to be sensitive instead to HER2xCD3-directed CIK cell lytic activity. The increase of CIK cell killing is correlated to the dose of bsAb and it is functional even at very low concentrations. Moreover, redirected-CK cells presents a proinflammatory and not a toxic cytokines profile. The bsAb HER2xCD3 arrives efficiently at the tumor site where reaches the maximum concentration after 8 hours of injection into mice.

**Conclusions**
These results highlight the potentiality of using clinical grade mAbs or recombinant immunotools to improve the cytotoxic activity of CIK cells against HER-2+ tumor cells, opening new perspectives for adoptive immunotherapy to treat solid tumors.

**REFERENCES**

**Disclosure Information**