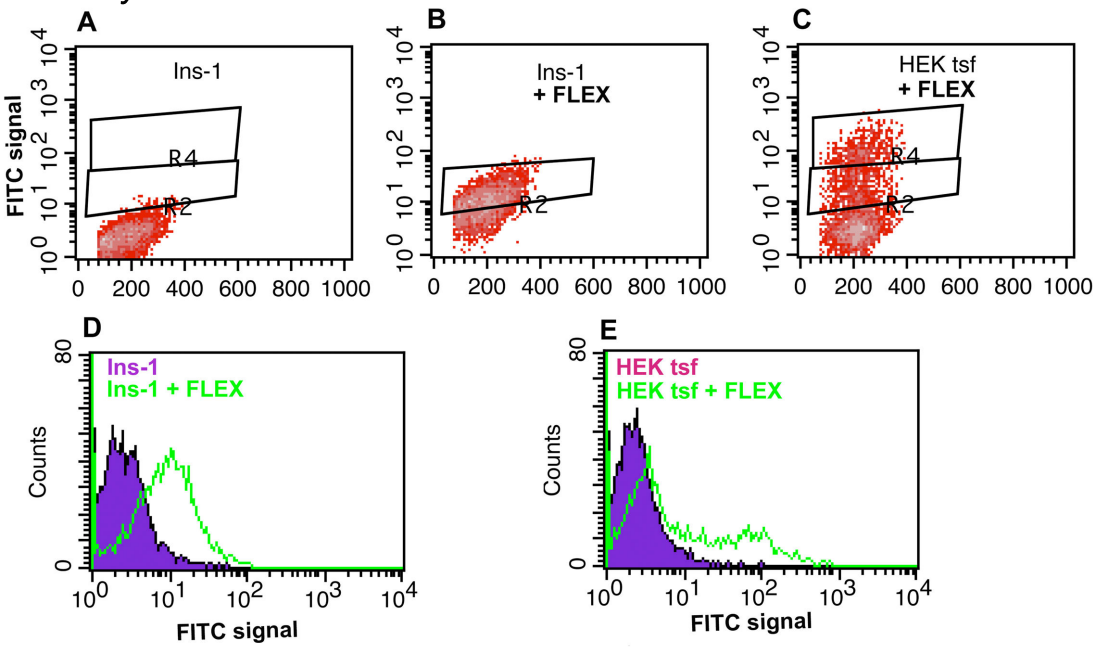
Schelshorn D, Joly F, Mutel S, Hampe C, Breton B, Mutel V, Lütjens R - *Lateral allosterism in the glucagon receptor family: GLP-1 induces GPCR heteromer formation* - Molecular Pharmacology



Supplementary Figure 1: GLP-1 binding by FACS. (A) Binding of FITC-labelled 1 µM Exendin-4 (FLEX) to INS-1E cells shifted the fluorescence of the whole population, resulting in a fluorescent labelling over base-line of 67.4 % of the cells (B). All positive cells were grouped into gate R2. Likewise, HEK cells over-expressing GLP-1R-RLuc8 + GIPR (same amounts of DNA as used in the BRET experiments) were labelled. The transfected cells population displayed a very heterogenous binding profile compared to INS-1E (D,E); many cells showed no labelling (non-transfected) and 37.1 % of the cells were labeled above base-line (C). Applying the gate R2 from INS-1E cells, 16.6% of all HEK cells (44.7% of transfected) express the GLP-1R at levels comparable to INS-1e (C) and 55.3% express the receptor at higher levels (gate R4). On average, on overexpression of 17.2 fold of the GLP-1R can be determined under the condtions used for BRET. INS-1E Kd= 2.7 nM/ Bmax = 3.89 (RLU); HEK: Kd= 5.5 nM/ Bmax = 64.72 (RLU).