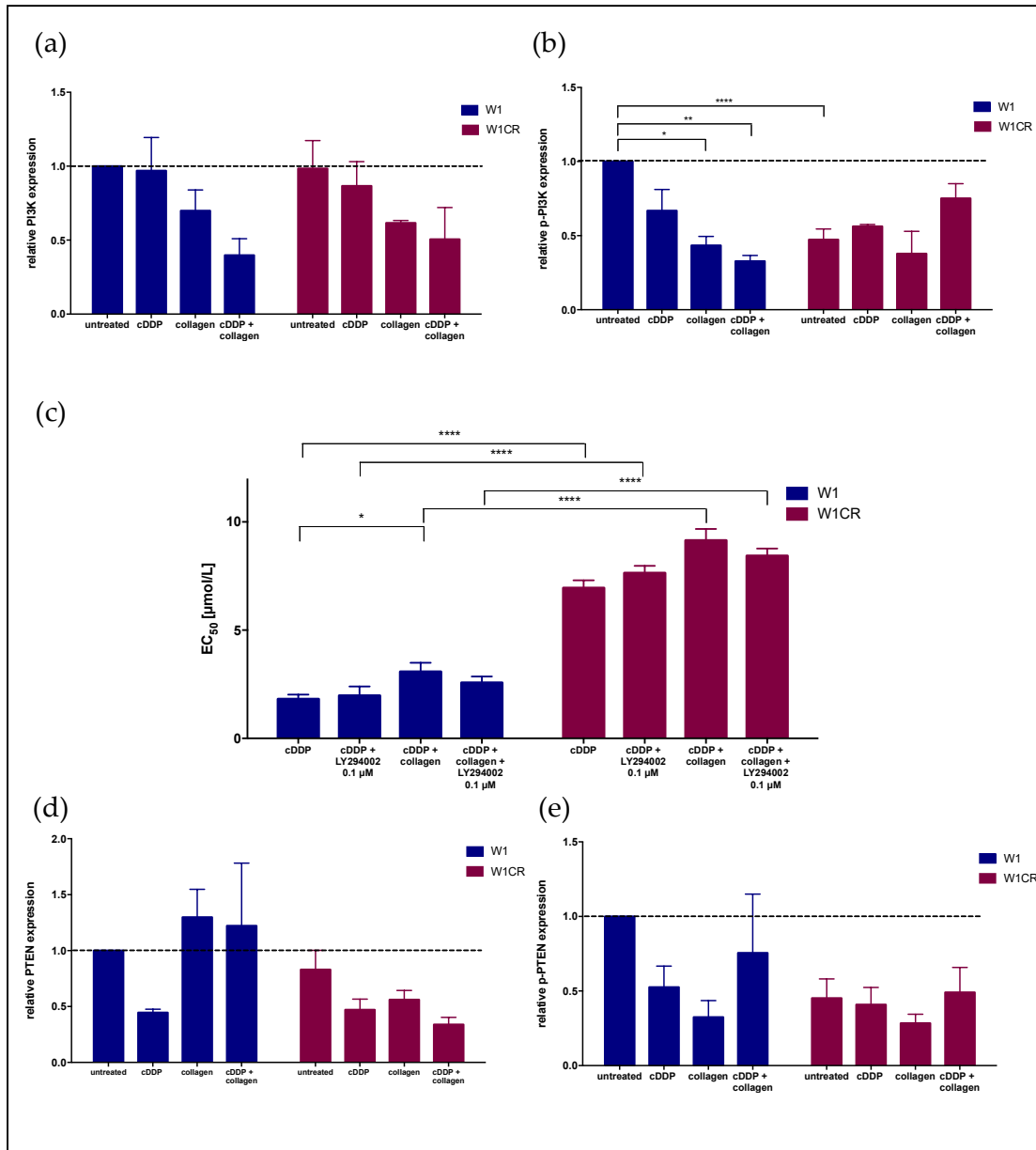
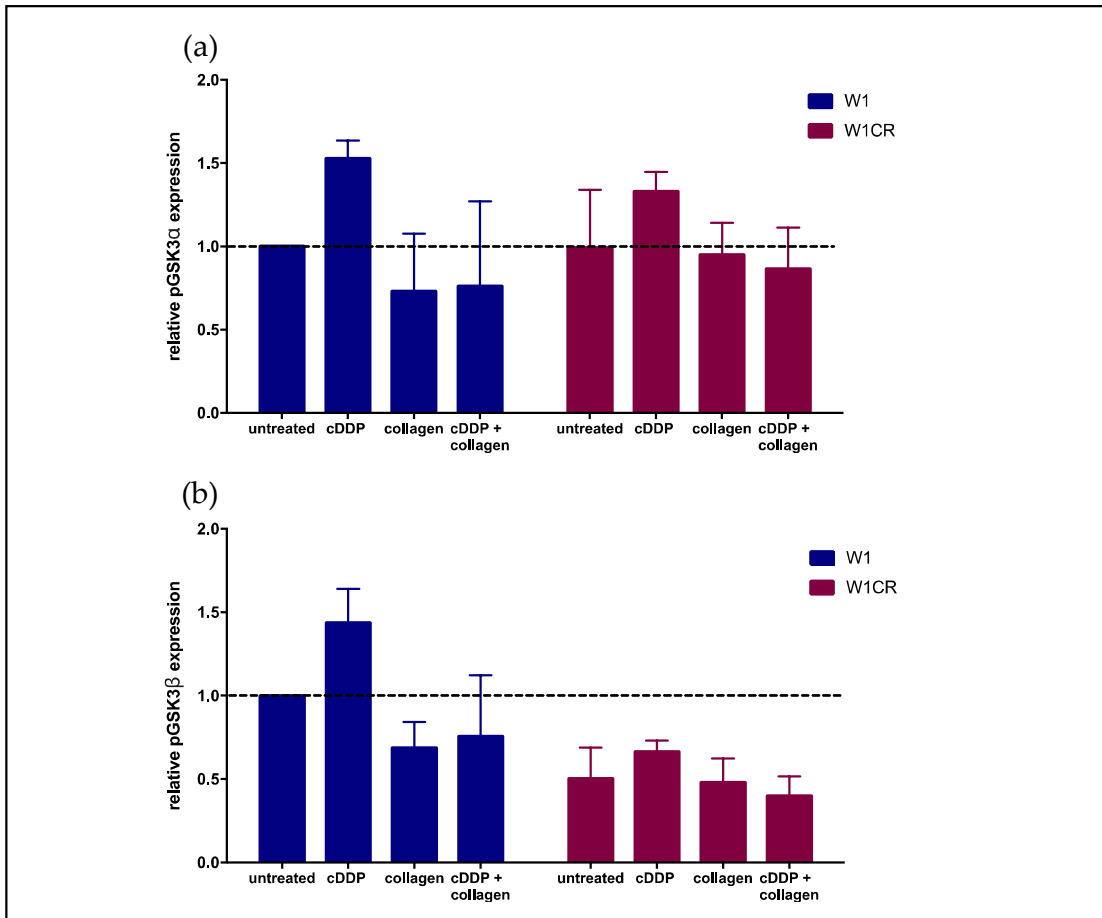


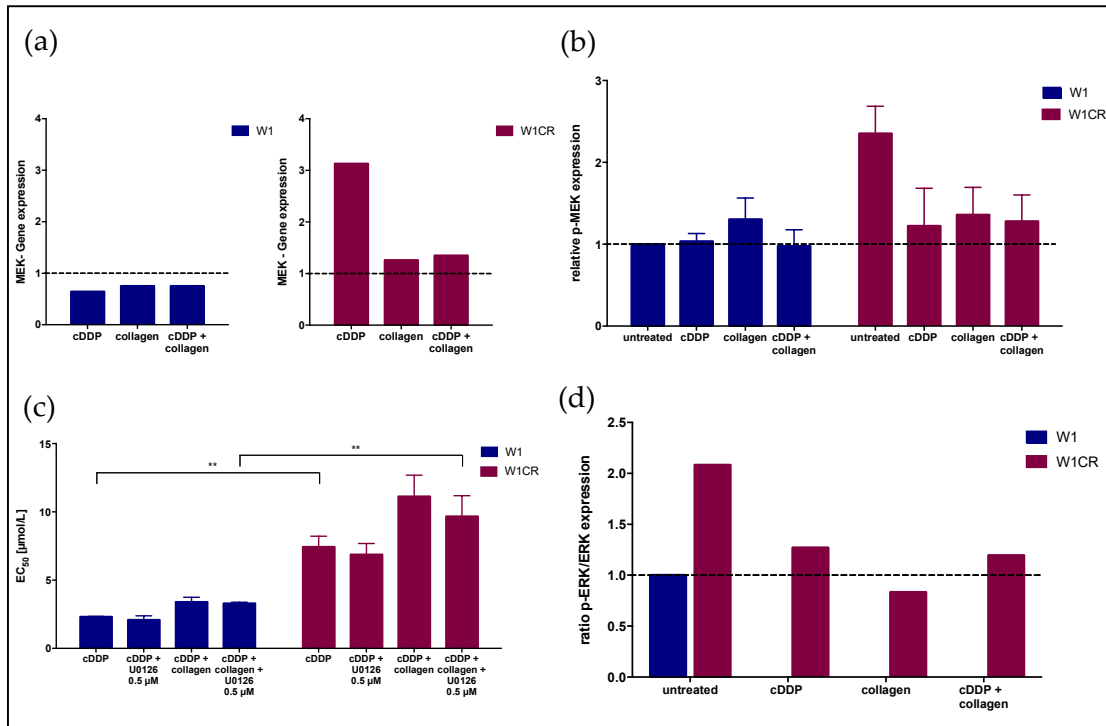
**Figure S1:** Knockdown of ITGB1 in W1 and W1CR cells. Shown is a representative Western blot of ITGB1 knockdown in W1 and W1CR cells, confirming the almost complete deletion of ITGB1 in the knockdown cells compared to scrambled control.



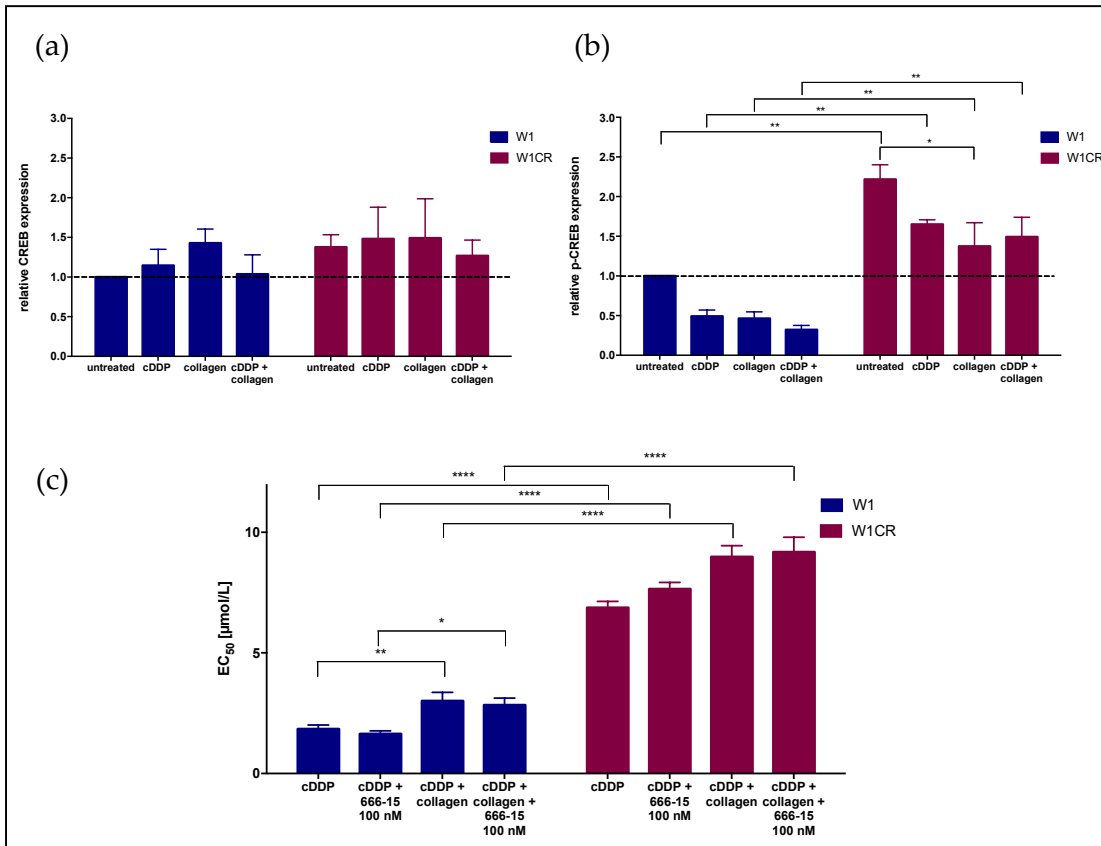
**Figure S2:** The impact of PI3K and PTEN pathway on the resistance of W1 and W1CR cells. Western blot data of PI3K (a) and phosphorylated fraction of PI3K (b). EC<sub>50</sub> levels under PI3K inhibition with LY294002 (0.1 μM) (c). Protein levels of PTEN (d) and p-PTEN (e). Data are means of at least n=3 (±SEM), asterisks indicate statistical significance: \*p<0.05; \*\*p<0.01; \*\*\*p<0.001; \*\*\*\*p<0.0001.



**Figure S3:** Western blot data of the wnt signaling related p-GSK3 $\alpha$  (a) and p-GSK3 $\beta$  (b) proteins in W1 and W1CR cells and their deregulation by cisplatin and/or collagen treatment.



**Figure S4:** The influence on MAPK pathway in W1 and W1CR cells. Deregulation of gene expression (fold change) of MEK (a) in W1 and W1CR cells upon the indicated treatment. Expression of p-MEK (b) at protein level and inhibition of p-MEK by U0126 at 0.5 μM (c). (d) Protein expression of ERK given as the p-ERK/ERK ratio as an indicator of ERK signaling activity. Protein data are means of at least n=3 (±SEM), asterisks indicate statistical significance: \*p<0.05; \*\*p<0.01.



**Figure S5:** The role of CREB, a MAPK pathway related downstream component in W1 and W1CR cells to regulate the sensitivity to cisplatin. Western blot data of CREB (a) and p-CREB (b) in W1 and W1CR cells upon the indicated treatments. (c) Impact of the CREB inhibitor 666-15 (0.1 μM) to affect the cisplatin sensitivity in both cell lines. Data are means of at least n=3 (±SEM), asterisks indicate statistical significance: \*p<0.05; \*\*p<0.01; \*\*\*p<0.001; \*\*\*\*p<0.0001.