FLOW CYTOMETRIC ANALYSIS MONITORING THE EFFECT OF PAN-CASPASE INHIBITOR (QvD) ON MARINE COMPOUND-INDUCED APOPTOSIS IN HCT116 COLON CANCER CELL LINE. CELLS WERE PRE-INCUBATED FOR 30 MIN WITH AND WITHOUT PAN-CASPASE INHIBITOR QvD (10 µM) THEN TREATED WITH MARINE COMPOUNDS (50 µM) OR TRAIL (0.25 AND 1 µg/mL) FOR 24 H. FOLLOWING INCUBATION, CELLS WERE STAINED WITH 7AAD AND Annexin V AND FLUORESCENCE WAS ANALYZED BY FLOW CYTOMETRY.
Figure S2: Autofluorescence screening and monitoring apoptosis-induced by marine compounds in HCT116 and Jurkat cells using flow cytometry. (A & B) HCT116 cells were incubated with the depicted compounds for 24h and stained (A) or not (B) with 7AAD and Annexin V and fluorescence was analyzed by flow cytometry. (C) Jurkat cells were treated as above and compound-induced autofluorescence was assessed by flow cytometry at 450, 530, 610 and 660 nm.
Figure S3: Detection of apoptosis induced by marine compounds in HCT116 and Jurkat isogenic cells. (A) HCT116 (WT), Cas8-deficient HCT116-Cas8−/−, and TRAIL receptors deficient HCT116-TRAIL-R1/TRAIR2−/− (B) Jurkat WT, Jurkat-Caspase-8−/−, and Jurkat-FADD−/−. Isogenic cells were incubated with the compounds for 24 h and then subjected to dual staining with annexin V/7AAD/. Apoptosis rate was determined by flow cytometry.
Figure S4: Raw data of the Western blots