Figure S1. Semi-quantitative analysis of TrkB, TrkC, MEK and ERK expression in chemoresistant colon cancer cells. The intensity of each band was normalized by corresponding β -actin and/or total protein expressions, respectively. Data are presented as the mean \pm SD and analyzed using one-way ANOVA. (A) Comparison of each protein level with that of internal control (β -actin). (B) Comparison of phosphorylated protein level with that of total protein. *P<0.05, **P<0.01, #P<0.005 and ##P<0.001 as indicated. Trk, tropomyosin receptor kinase; p, phosphorylated.

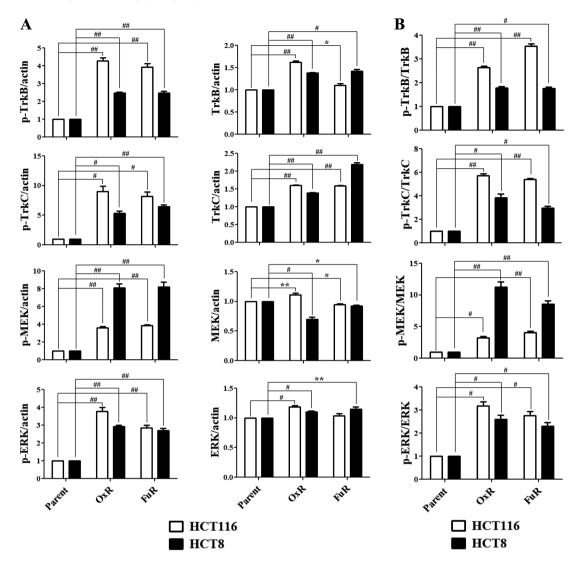


Figure S2. Effect of Trk inhibitor or si-HOXC6 on the viability of chemoresistant colon cancer cells. Cells were treated with the 2 μ M CH7057288 (Trk inhibitor) and 200 nM HOXC6-siRNA. At 24 h after treatment, cell viability was measured using a Cell Counting Kit-8 assay. Absorbance at 450 nm is presented. Data are presented as the mean ± SD of three independent experi ments. OxR and FuR (A) HCT116 and (B) HCT8 cells. Trk, tropomyosin receptor kinase; si or siRNA; small interfering RNA; Ctrl, control; OD, optical density; OxR, oxaliplatin resistant; FuR, 5-Fu-resistant.

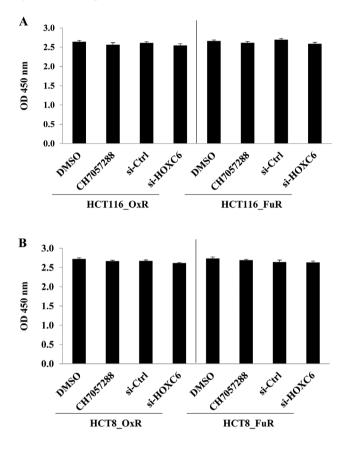


Figure S3. Semi-quantitative analysis of p-TrkB, TrkB, p-TrkC and TrkC expression in chemoresistant colon cancer cells after treatment with the Trk inhibitor. The intensity of each band was normalized by corresponding β -actin and/or total protein expressions, respectively. Data are presented as the mean \pm SD and analyzed using one-way ANOVA. (A) The effect of CH7057288 on the expression of p-TrkB, TrkB, p-TrkC and TrkC with comparisons made to that of (A) the internal control and (B) total protein as appropriate. #P<0.005 and ##P<0.001 as indicated. p, phosphorylated; Trk, tropomyosin receptor kinase; OxR, oxaliplatin resistant; FuR, 5-Fu-resistant.

