

Figure S1: Dose response for resveratrol activation of AMPK. L β T2 cells were starved overnight before resveratrol (1-100 μ M) was added to cells for 4 h. Cell lysates were isolated with RIPA buffer, protein concentrations were measured and 30 μ g protein was separated by SDS-PAGE and immunoblotted for pAMPK then stripped and reblotted for tubulin

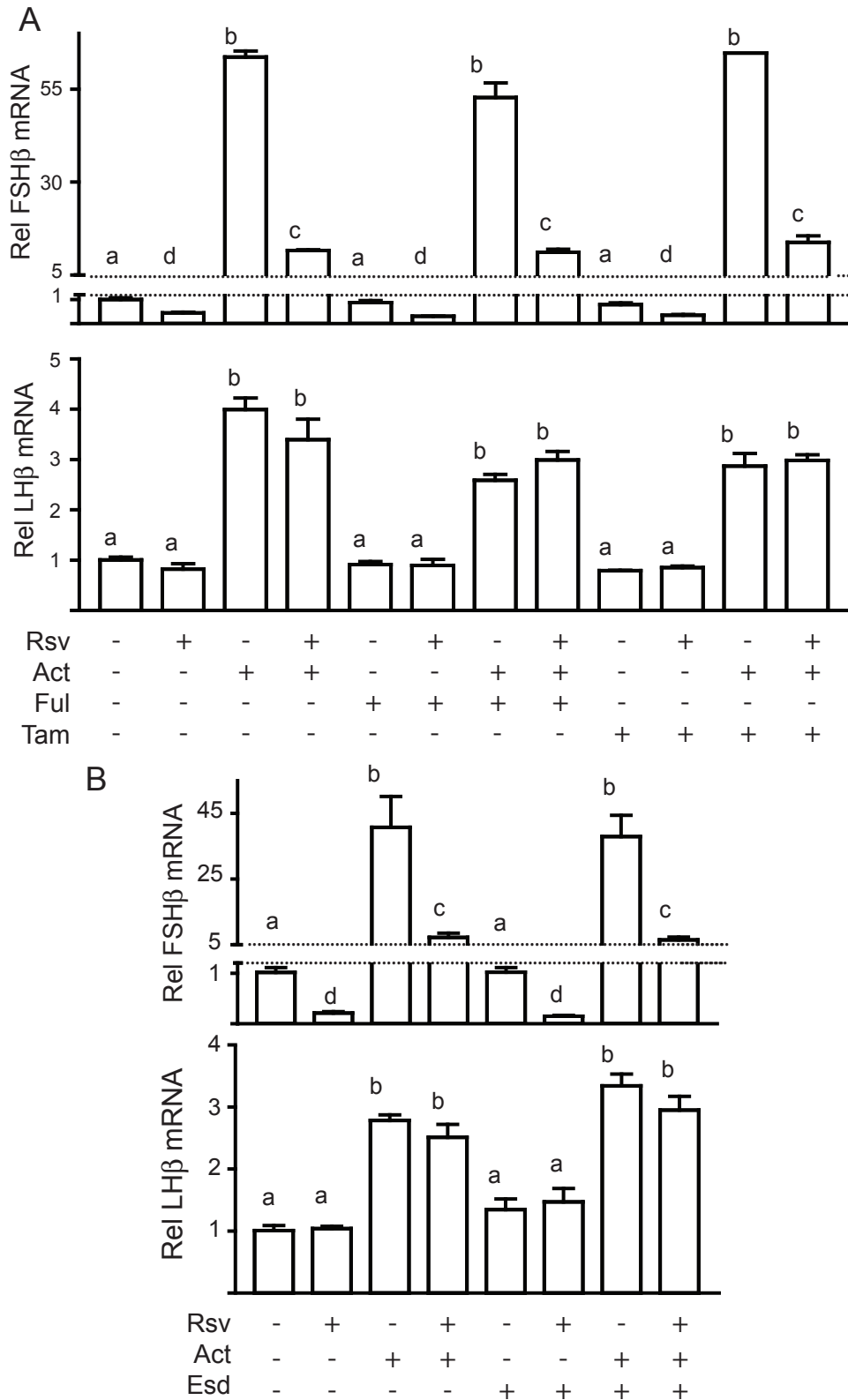


Figure S2: Estrogen receptor agonists and antagonists do not reverse resveratrol effects
 L β T2 cells were starved overnight with or without 12.5 ng/ml activin (Act), then 100 μ M resveratrol (Rsv) or the indicated agonists or antagonists or both were added for the last 4 h. Total RNA was isolated and FSH β and LH β mRNA levels measured by QPCR. Panel A: FSH β and LH β mRNA levels in response to fulvestrant and tamoxifen. The estrogen receptor (ER) antagonists fulvestrant (Ful, 100 nM) and tamoxifen (Tam, 100 nM) were added for the last 4 h of treatment. Panel B: FSH β and LH β mRNA levels in response to estradiol. The ER agonist 17 β -estradiol 100 nM was added for the last 4 h of treatment. Data are presented as relative mRNA level compared to basal untreated cells normalized to the RPS3 protein mRNA. Graphs show the mean and standard deviation from three experiments, n = 6. Letters indicate statistical significance (p<0.05), bars with the same letter are not significantly different.

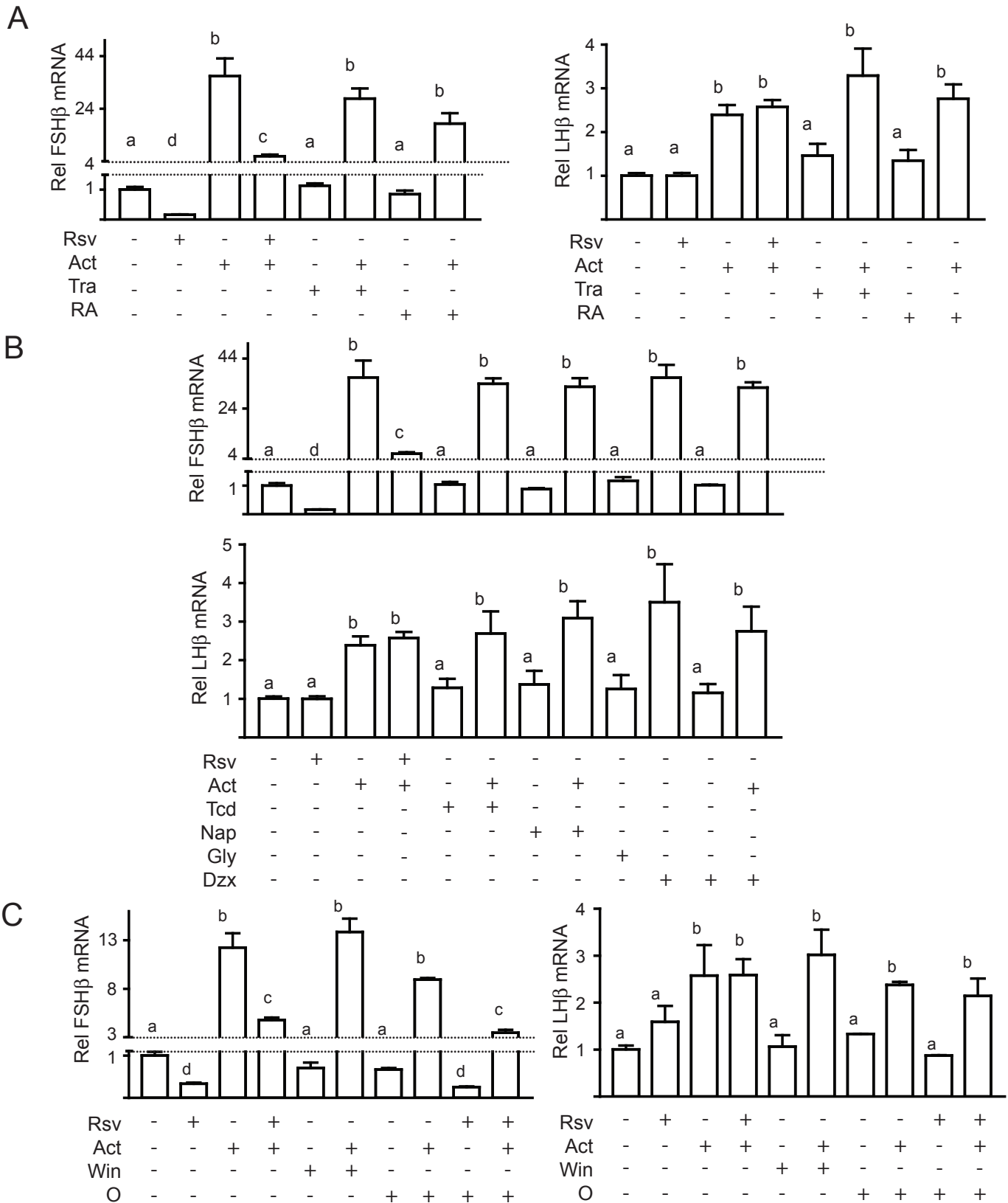


Figure S3: Nuclear receptor agonists and antagonists do not mimic resveratrol effects. L β T2 cells were starved overnight with or without 12.5 ng/ml activin (Act), then 100 μ M resveratrol (Rsv) or the indicated agonists or antagonists or both were added for the last 4 h. Total RNA was isolated and FSH β and LH β mRNA levels measured by QPCR. Data are presented as relative mRNA level compared to basal untreated cells normalized to the RPS3 protein mRNA. Graphs show the mean and standard deviation from three experiments, n = 6. Letters indicate statistical significance (p < 0.05), bars with the same letter are not significantly different. Panel A: RAR agonist all-trans-retinoic acid (Tra, 10 μ M) or RXR agonists 9-cis-retinoic acid and 13-cis-retinoic acid (RA, both at 10 μ M) were added for the last 4 h. Panel B: AhR agonist 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCD, 10 nM) or antagonist β -naphthoflavone (Nap, 30 μ M), SUR1 antagonist glybenclamide (Gly, 10 μ M) or diazoxide (Dzx, 10 μ M) were added for the last 4 h. Panel C: CB1 agonist WIN 55212-2 mesylate (Win, 100 nM) or antagonist O-2050 (O, 10 μ M) were added for the last 4 h.