Supplementary material

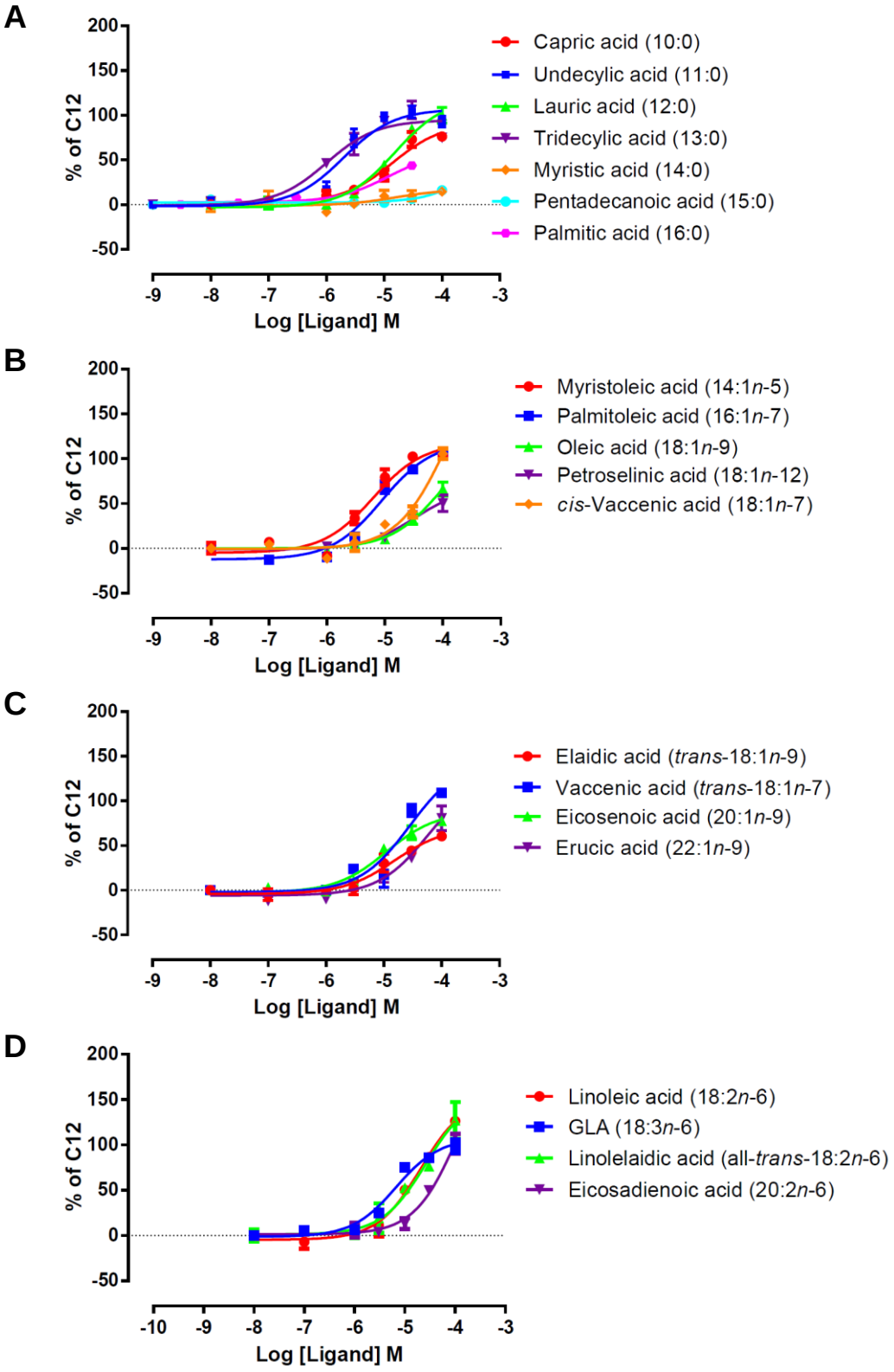

Fig. S1A-D. Concentration-response curves for NEFAs on hFFA1. Data points as mean ± SEM, n = 2-4.

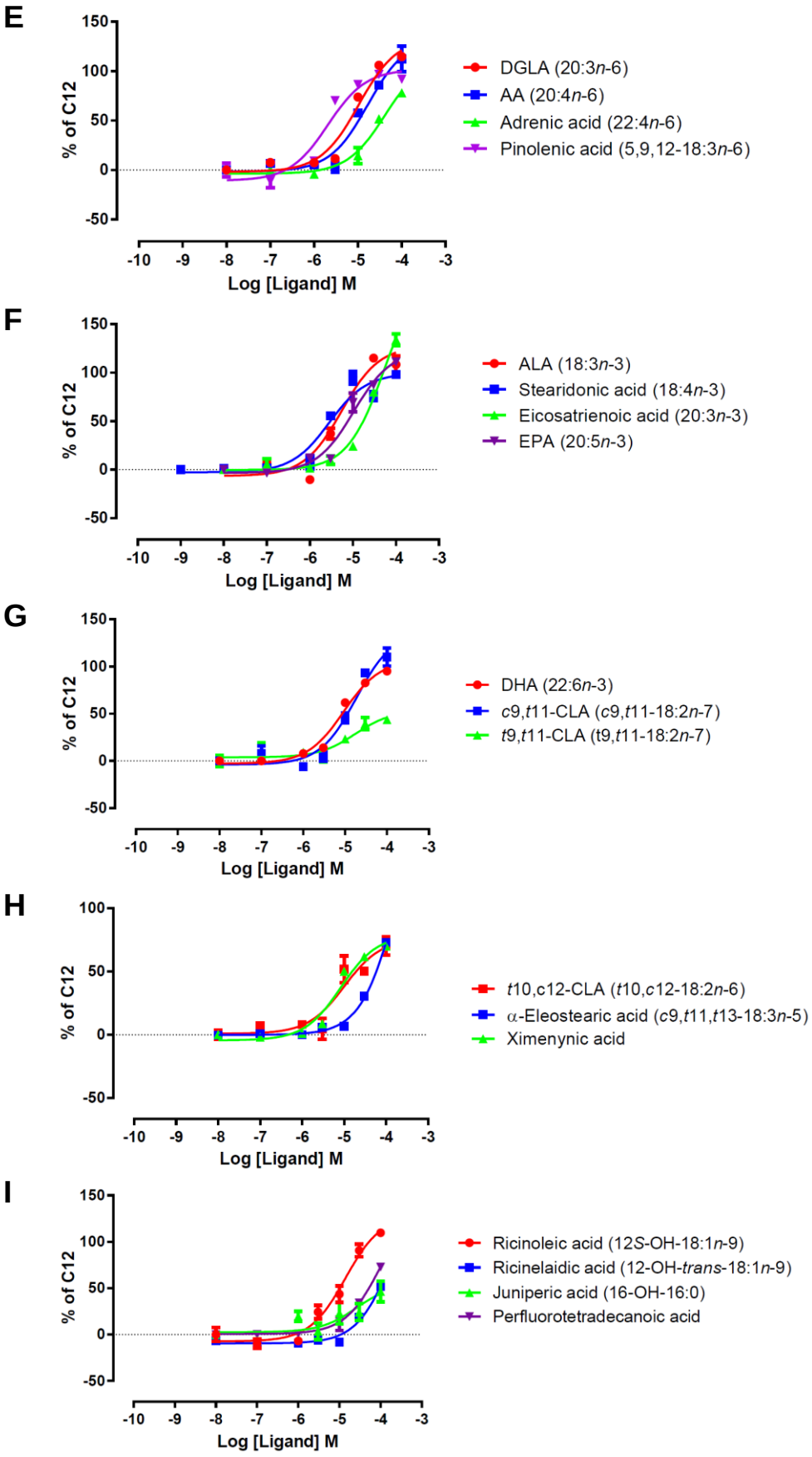

Fig. S1E-I. Concentration-response curves for NEFAs on hFFA1. Data points as mean ± SEM, n = 2-4.

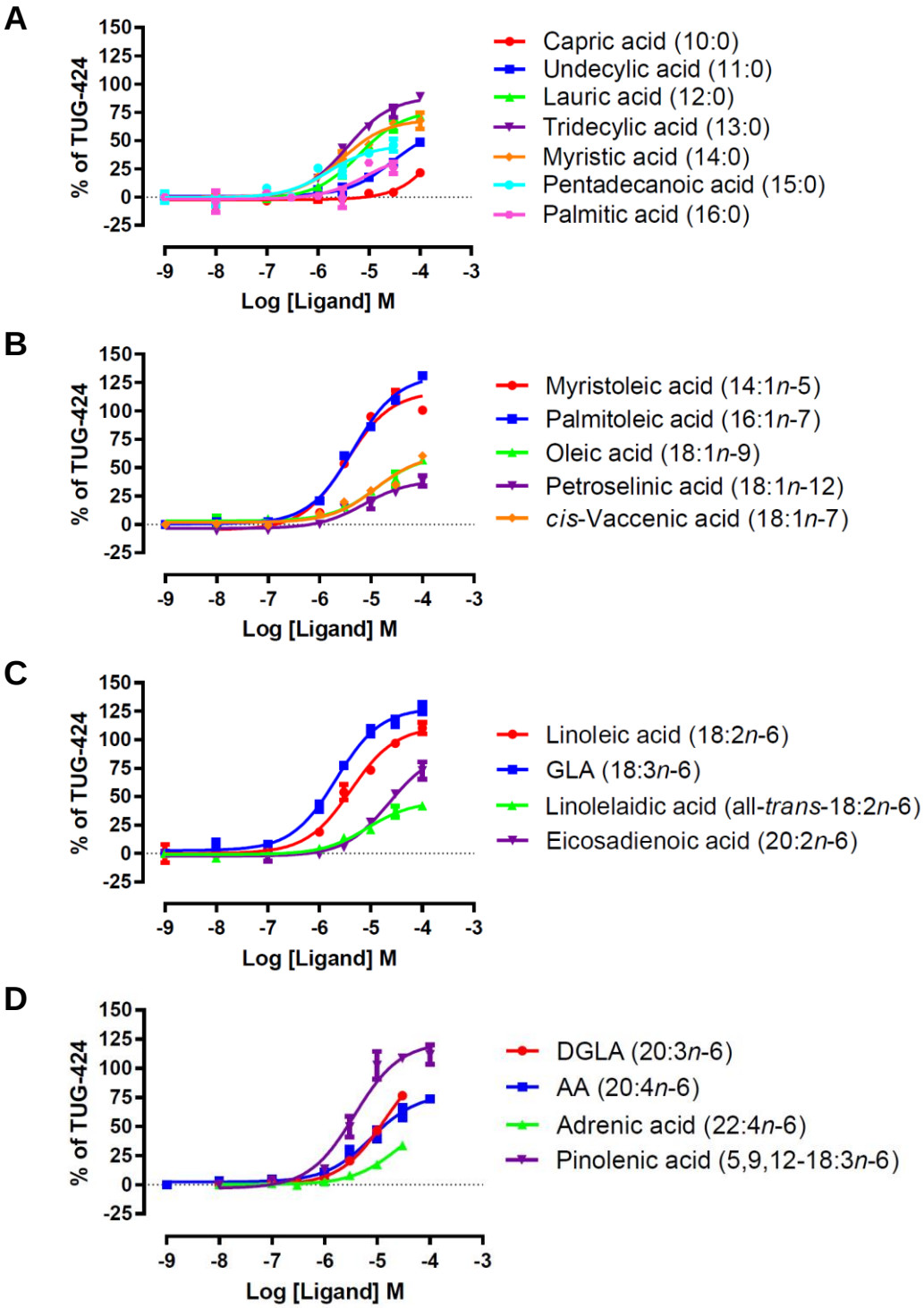

Fig. S2A-D. Concentration-response curves for NEFAs on hFFA4. Data points as mean ± SEM, n = 2-4.

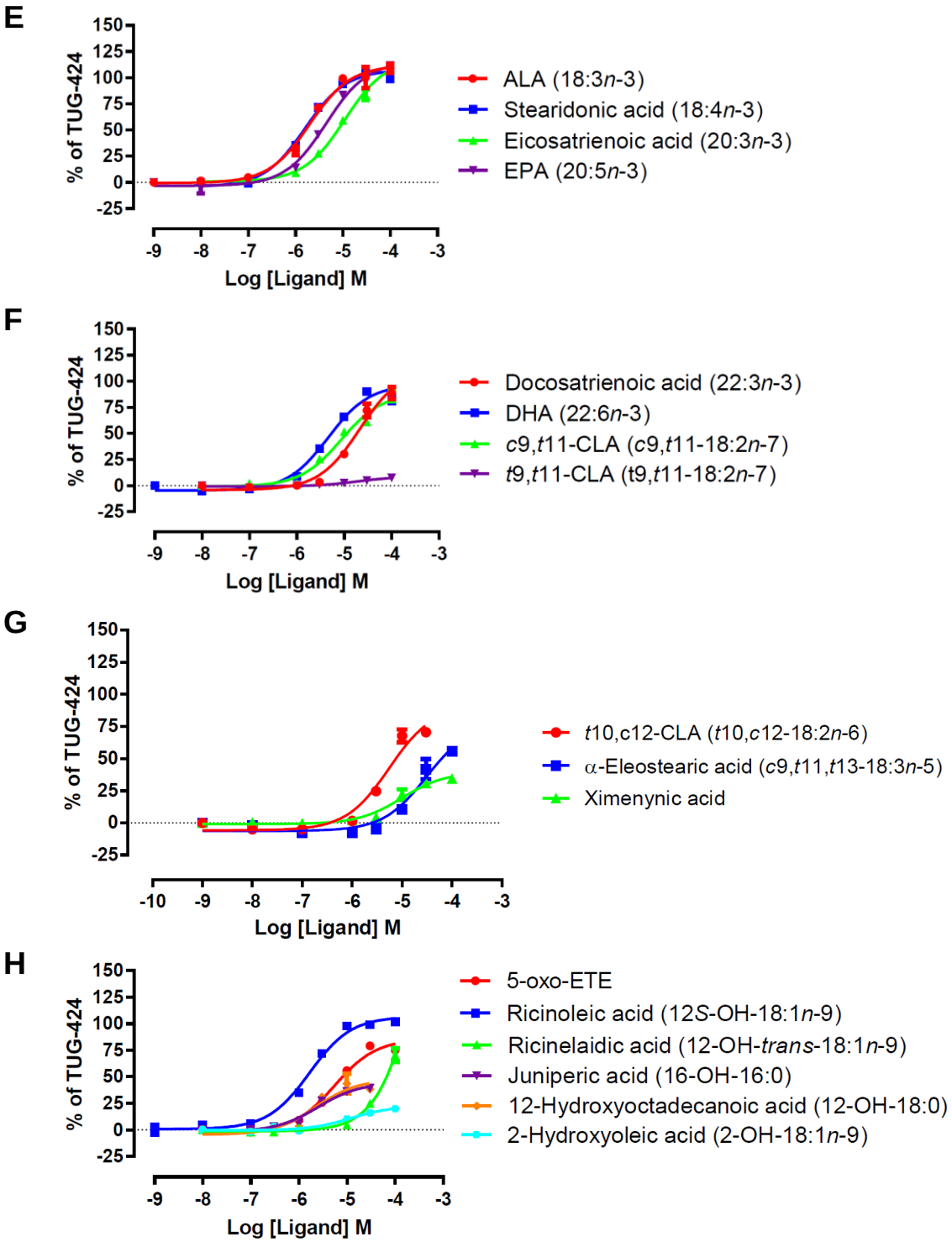

Fig. S2E-H. Concentration-response curves for NEFAs on hFFA4. Data points as mean ± SEM, n = 2-4.

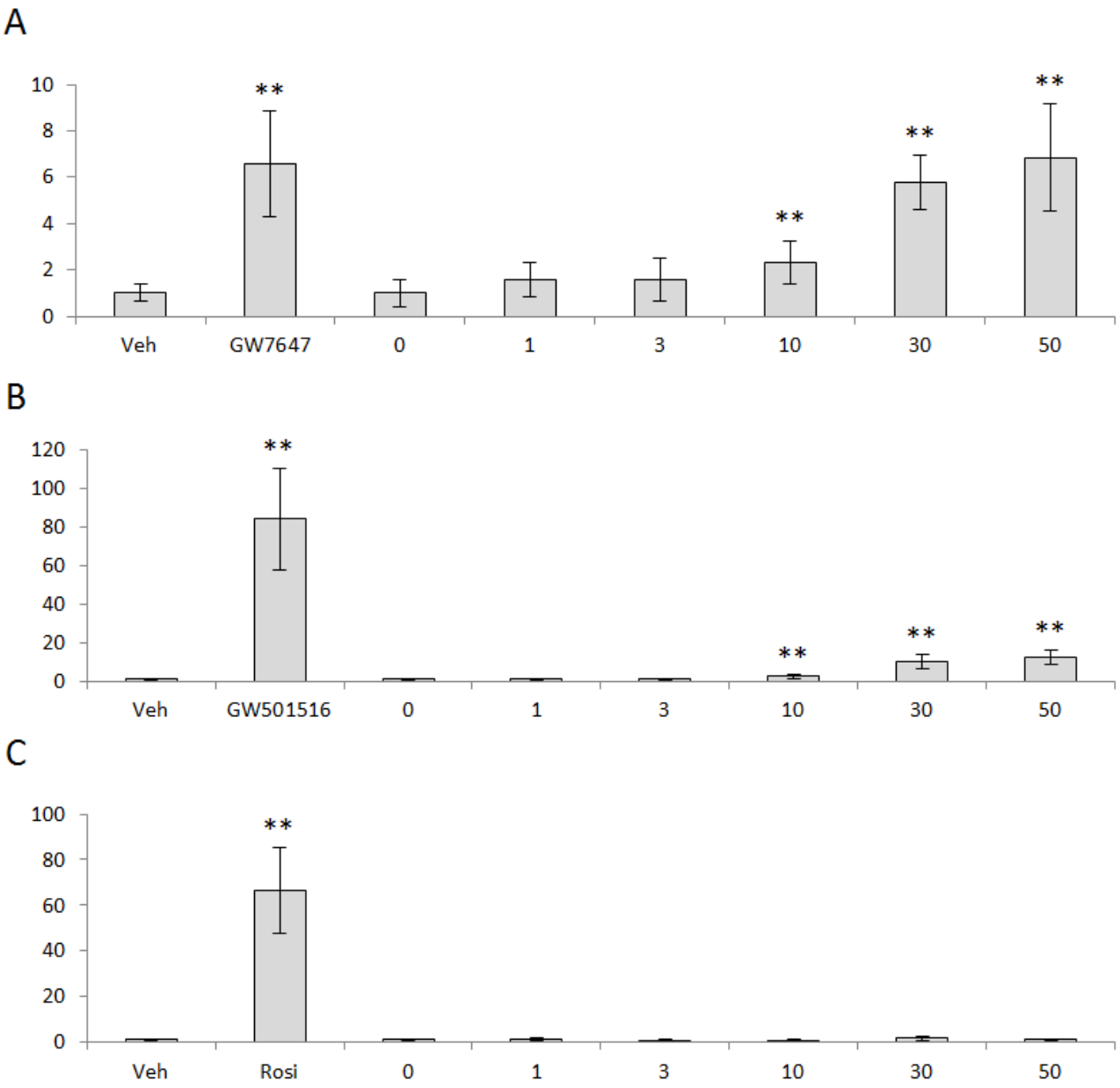

Fig. S3. Activity of pinolenic acid on PPARα (A), PPARδ (B) or PPARγ (C) at 0-50 μM using GW7647 (30 nM), GW501516 (100 nM) and rosiglitazone (Rosi, 1 μM) as positive controls. The y-axis indicates fold activation over vehicle (Veh). Error bars represent standard deviation. *, $p<0.05$; **, $p<0.01$, n = 6-8.

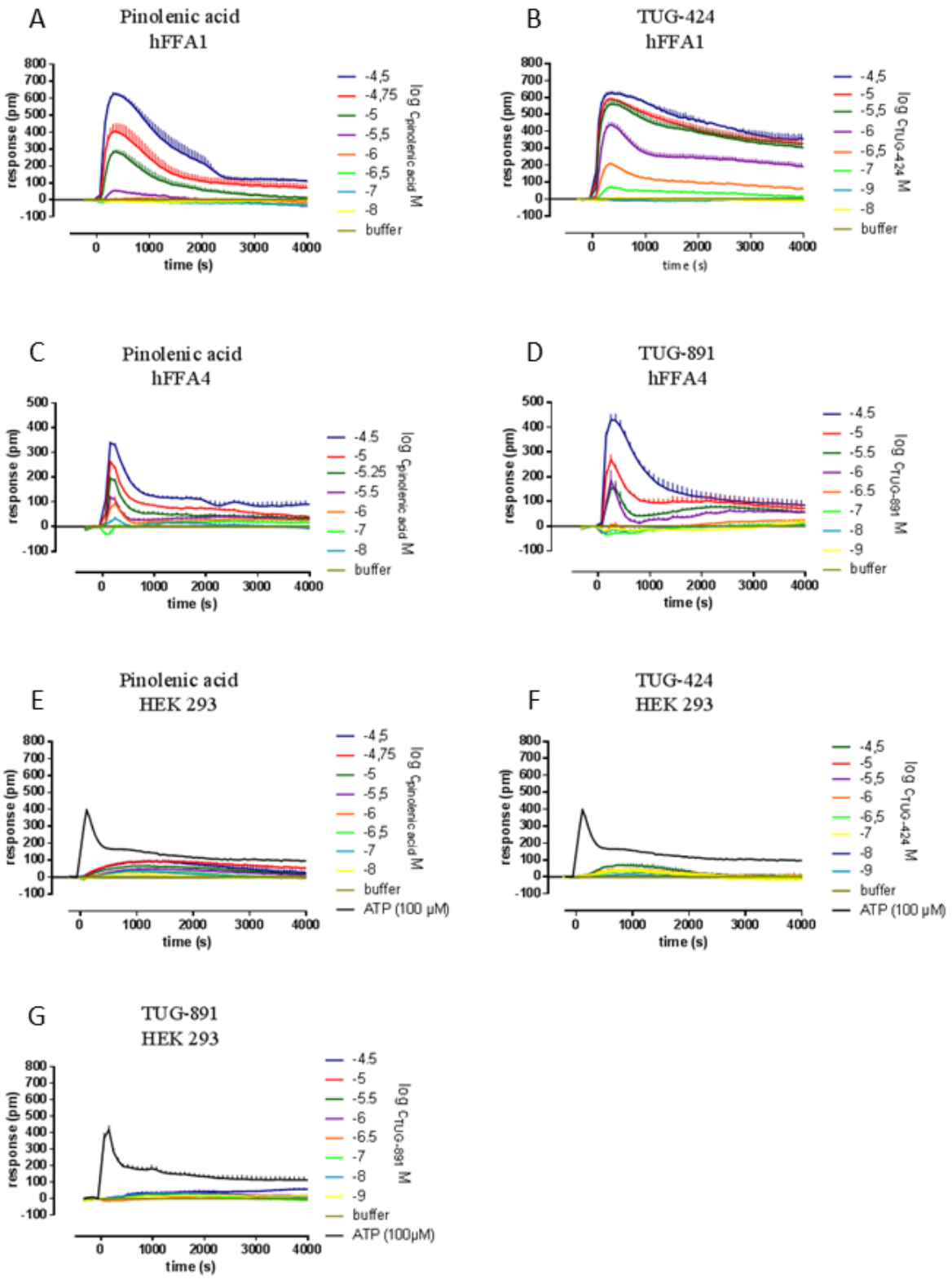

Fig. S4. Optical fingerprints of pinolenic acid (A, C, E), the selective FFA1 agonist TUG-424 (B, F) and the selective FFA4 agonist TUG-891 (D, G) determined by label-free DMR assays in HEK cells overexpressing hFFA1 (A, B), hFFA4 (C, D) or native HEK cells (E-G). Depicted are representative traces as mean values ± S.E.M. measured in triplicates.