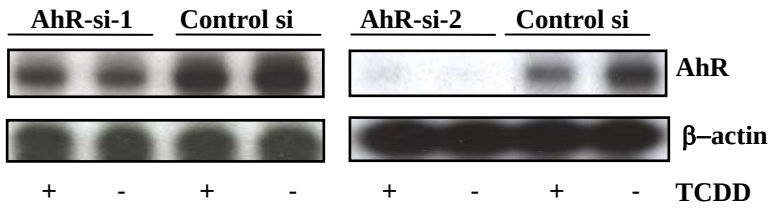
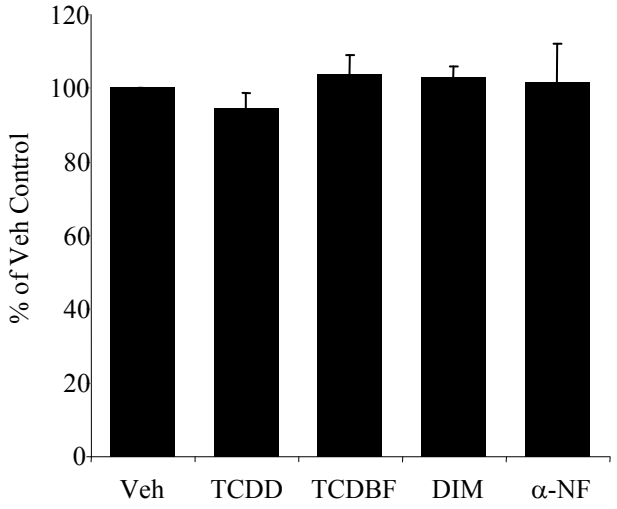
Supplemental Figure 1



Suppl. Fig. 1. Expression of endogenous AhR protein in human breast cancer cells. SKBR3 cells were transfected with either a nontargeting siRNA duplex (Control si) or one of two independent AhR siRNAs (AhR-si-1, AhR-si-2). After 24 h cells were treated with Veh or TCDD (10 nM) for 24 h. 48 h post-transfection cells were harvested for total cellular protein. Cell extracts (20 μg/sample) were separated by electrophoresis and proteins were transferred to a PVDF membrane. Western blotting of SKBR3 cell extracts was performed with an anti-AhR antibody. Membranes were stripped and rehybridized with a β-actin antibody for normalization for protein content.

Supplemental Figure 2



Suppl. Fig. 2. AhR Agonists Do Not Inhibit Colonization of HeLa Cells. Results from the soft agar colony formation assay. Anchorage-independent growth of HeLa cells (which lack detectable AhR activity) following continuous exposure to multiple AhR ligands (TCDD, 10 nM; TCBDF, 10 nM; and DIM, 20 μM) and the AhR antagonist α-NF (10 μM) for 21 days. Cells were plated in 6 well plates in soft agar containing AhR ligand and overlayed with media containing ligand. Graphical data are represented as % of Vehicle (Veh) control (set at 100%). (n = 3 independent assays; no statistically significant differences exist between treatments).