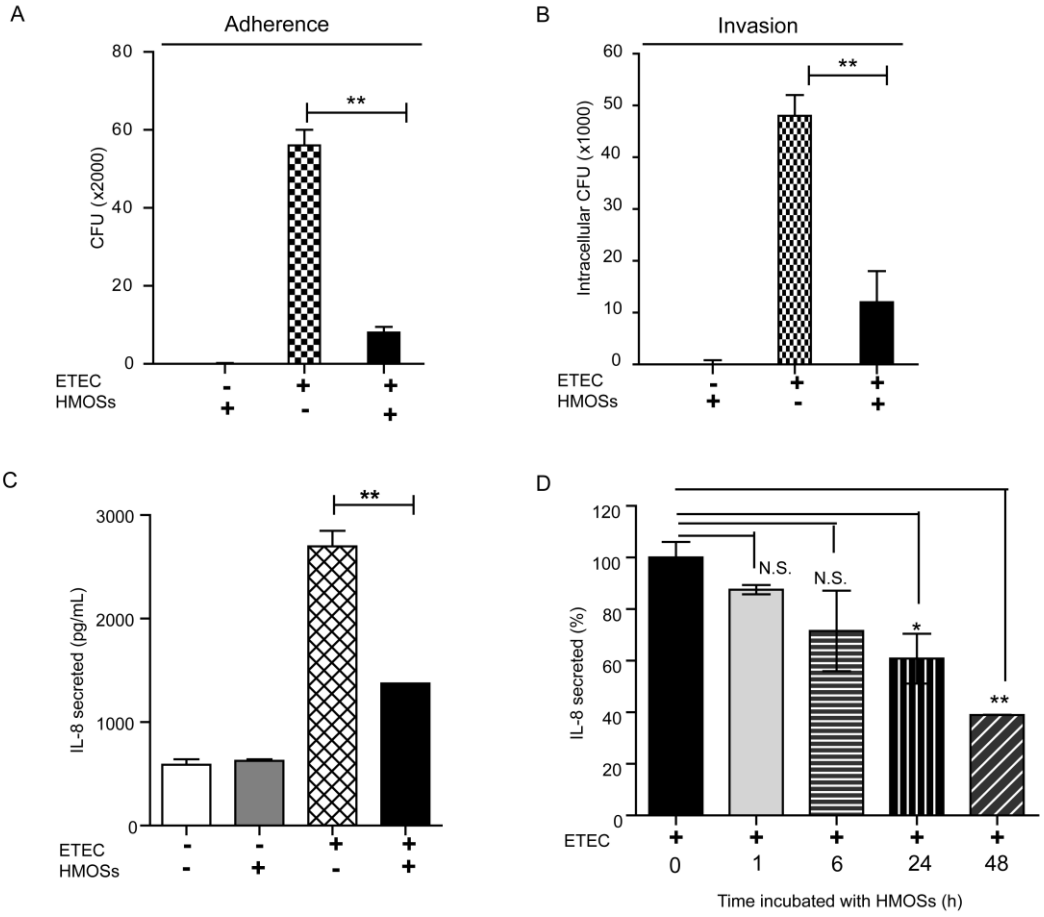
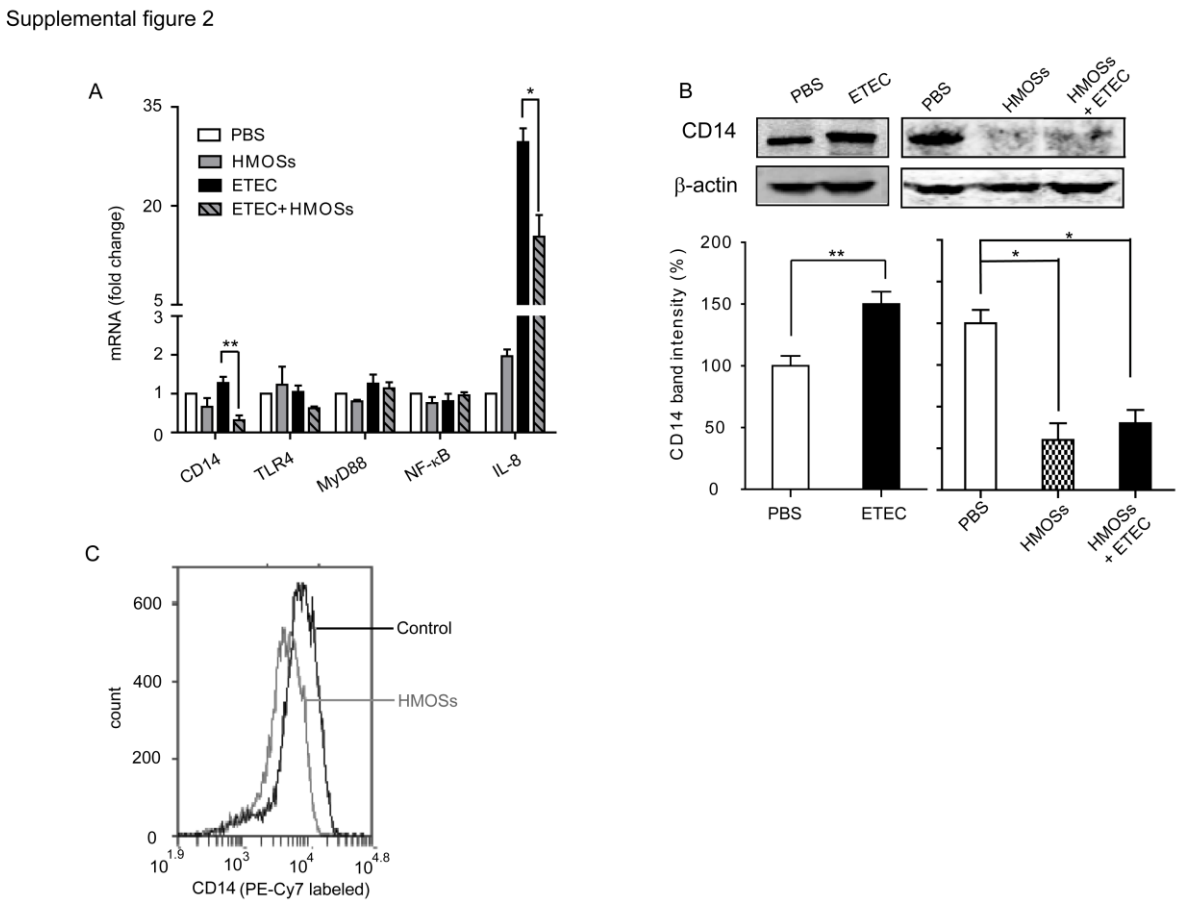
**Supplemental figure 1**. **T84 response to ETEC is inhibited by HMOSs. T84 were treated with 5 mg/mL HMOSs for 48 hours before exposure to ETEC (MOI 20). A)** Adherence was reduced to 12%**. B)** ETEC invasion was reduced to 22% **C)** IL-8 secretion was reduced to 40%. **D)** IL-8 reduction was time dependent. Means ± SEM, n=12. *, p≤0.05; **, p≤0.01 by ANOVA.

Supplemental figure 1

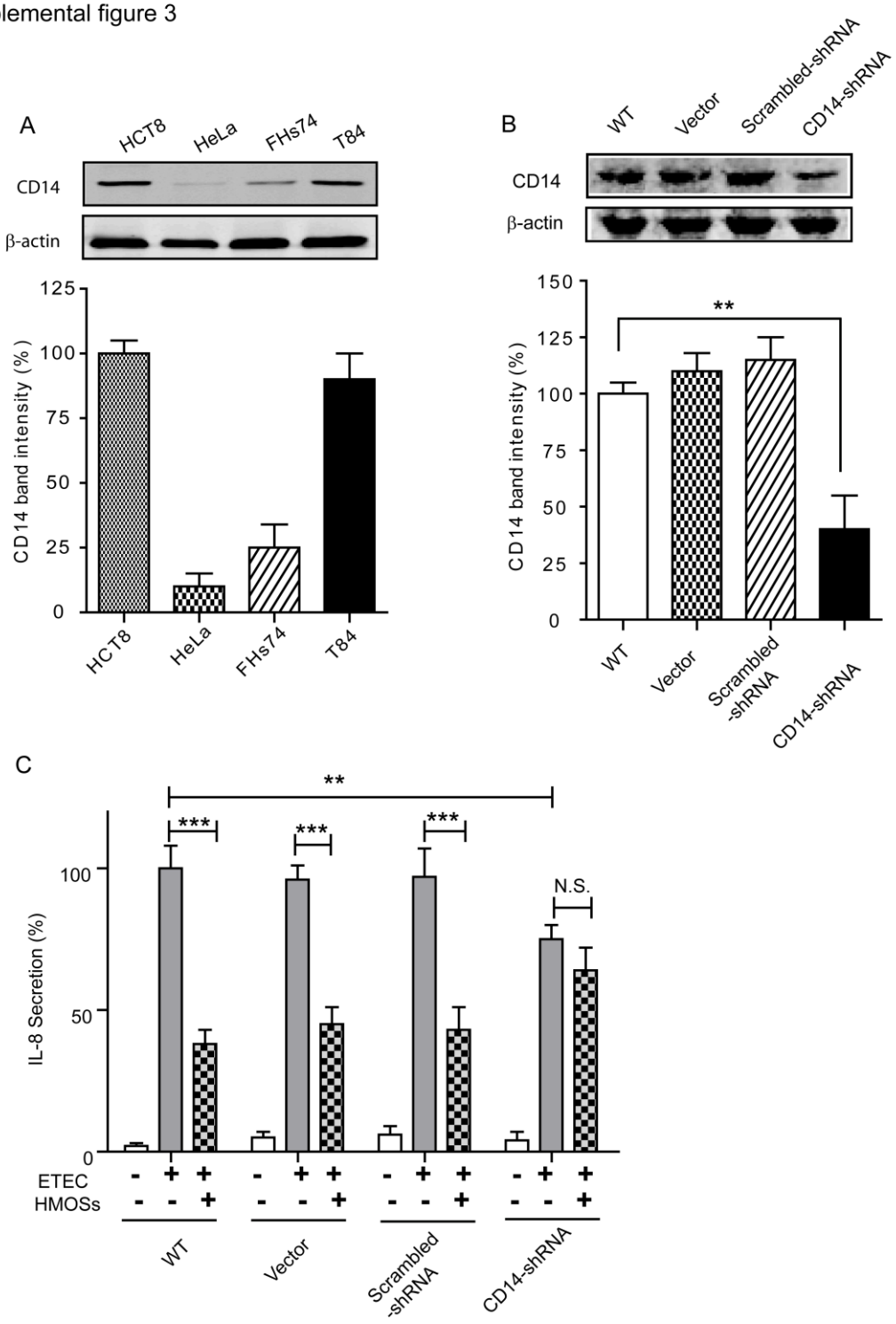


**Supplemental figure 2**. **CD14 expression in ETEC-infected T84 cells is inhibited by HMOSs. T84 were treated with 5 mg/mL HMOS for 48 hours before exposure to ETEC (MOI 20). A)** HMOSs attenuate CD14 mRNA and IL-8 mRNA induction by ETEC infection, n=3,. **B)** HMOSs inhibit CD14 expression, n=3. **C)** cell surface CD14 is reduced by HMOSs, n=3. . *, p≤0.05; **, p≤0.01 by ANOVA.



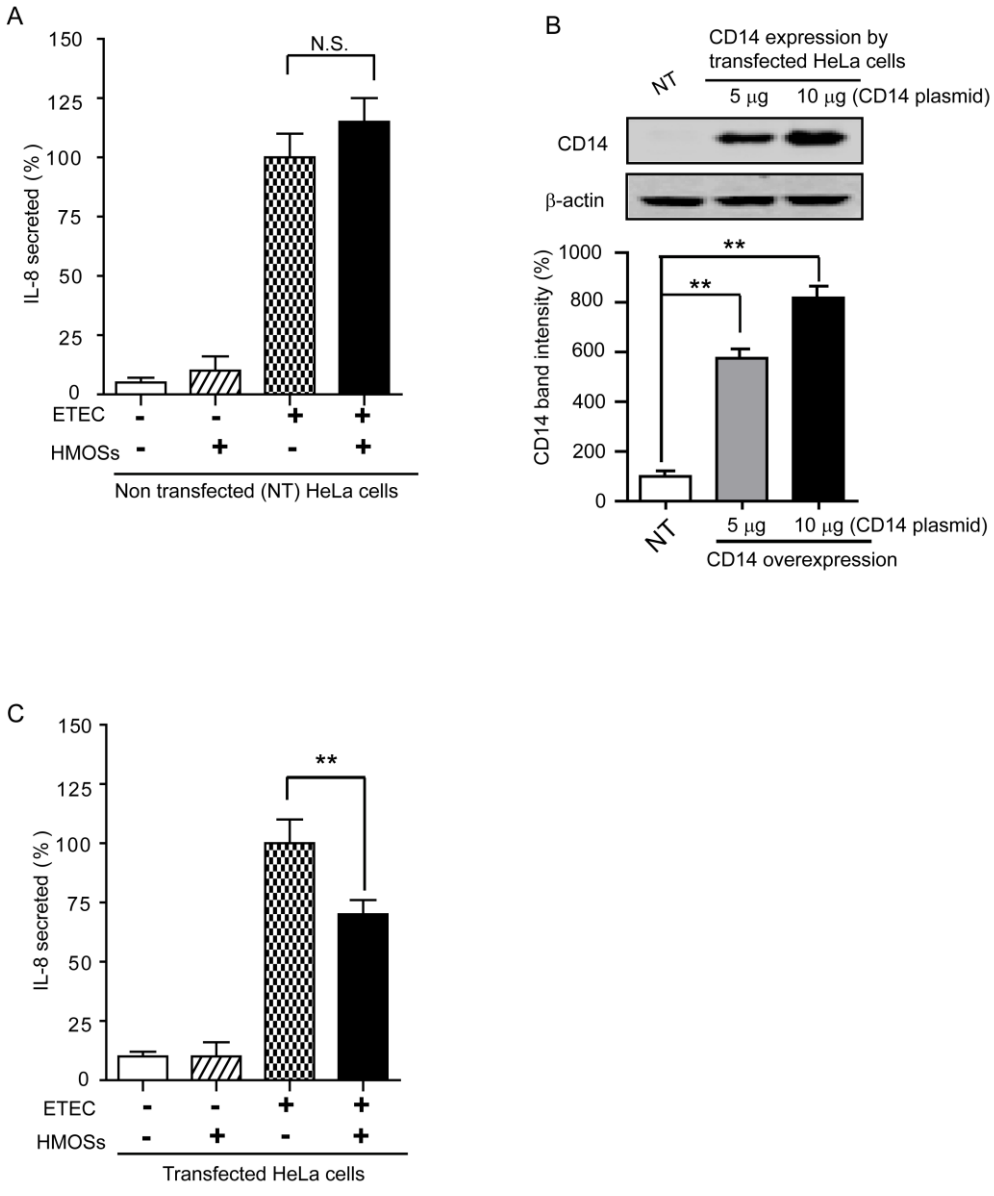

**Supplemental figure 3. T84 expression of CD14 is sensitive to knock-down and HMOSs. T84 and shRNA T84 cells were treated with 5 mg/mL HMOSs for 48 hours before exposure to ETEC (MOI 20). A)** CD14 expression levels among IECs, n=3 **B)** CD14 expression in pRS-shCD14 RNA transfected T84 cells; the pRS vector and scrambled pRS-RNA are negative controls, n=3. **C)** IL-8 secretion induced by ETEC invasion of T84 cells is attenuated by CD14 knockdown, **and** by HMOSs in controls; residual IL-8 in CD14 knocked-down T84 cells is unchanged by HMOS treatment, consistent with hMOS inhibition being specific for CD14 mediated IL-8 induction, n=6. **, p≤0.01; ***, p≤0.001 by ANOVA.

Supplemental figure 3



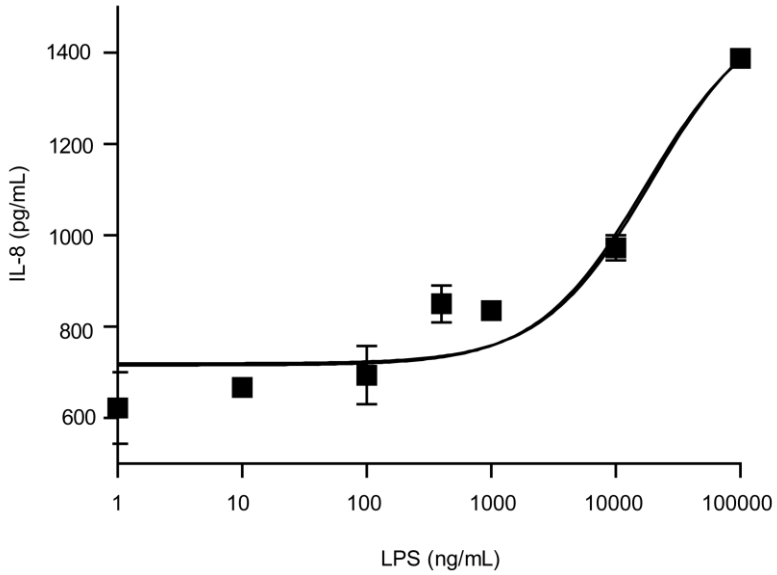
**Supplemental figure 4. Overexpression of CD14 increases the IL-8 response to ETEC infection, which is attenuated by HMOS.** HeLa cells, intrinsically low in CD14, when transfected with the gene for CD14, overexpressed CD14 without inducing toxicity. A) Prior to transfection, HeLa cell IL-8 induction by ETEC infection is not sensitive to HMOS treatment (n=6). **B)** HeLa cells transfected by a CD14 plasmid construct overexpress CD14 in proportion to the plasmid dose (n=3). **C)** IL-8 induction by ETEC infection in CD14 overexpressing HeLa cells is inhibited by HMOS, consistent with HMOS inhibition of IL-8 being mediated through CD14 (n=6). IL-8 data (A&C) normalized to untreated ETEC infection (100%). ** indicates significant difference between mean values, p≤0.01.

Supplemental figure 4



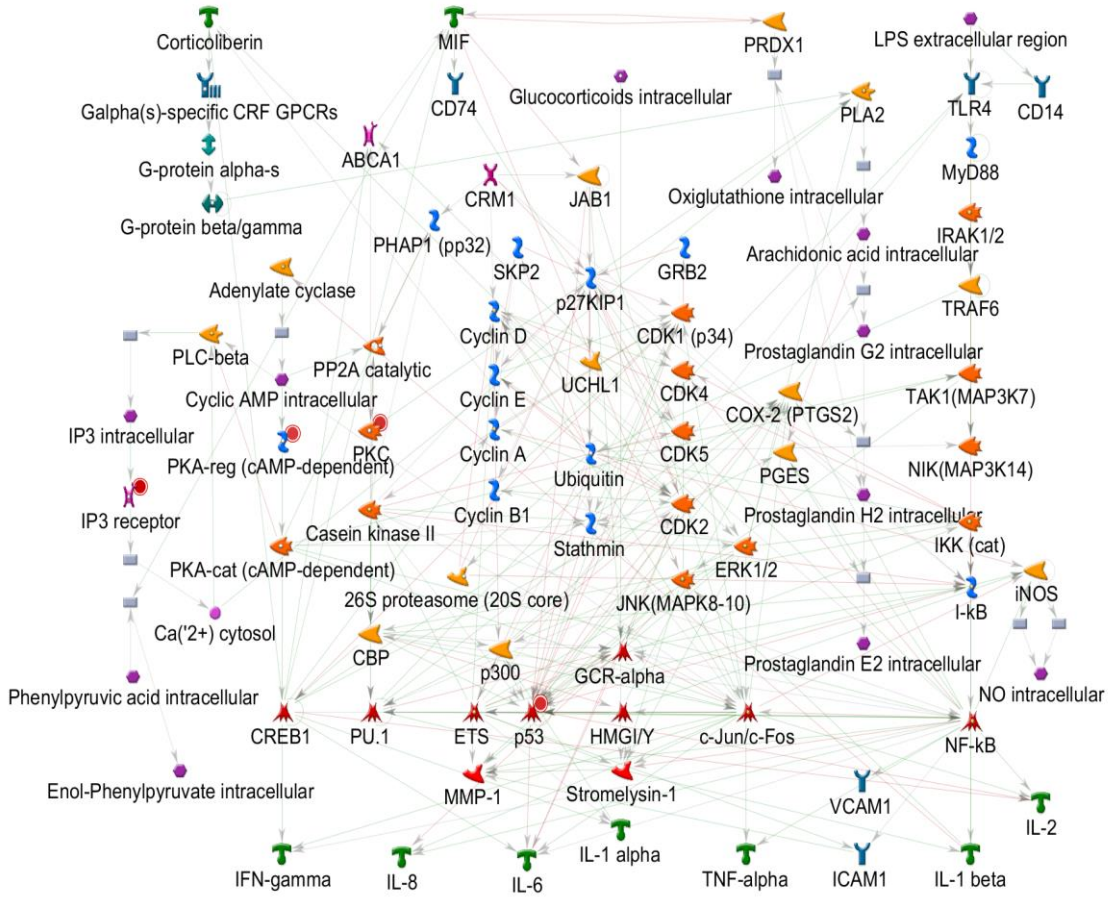
**Supplemental figure 5. IL-8 response to 1-100,000 ng/mL LPS in T84 cells. T84** IECs respond at 10 μg/mL, and a dose of 100 μg/mL (~IC50) was used for T84 cell stimulation in these studies.

Supplemental figure 5




**Supplemental figure 6. Signaling mediators of the MIF pathway whose expression is altered by 2'-FL.** T84 cells were treated with 2'-FL for 48 hours, the proteins extracted, and applied to a signaling molecule antibody array. Meta core analysis identified the MIF network signaling pathways whose intermediates are most effected by 2'-FL. Genes whose expression is significantly induced by 2'-FL treatment are labeled red, and include IP3 receptor, MMP-1 and Stromelysin-1. Genes whose expression is significantly repressed by 2'-FL treatment are labeled green, and include the cytokines IFN-γ, IL-8, IL-6, IL-1α, TNF-α, IL-1β and IL-2.

Supplemental figure 6




**Supplemental figure** 7. Comparison of H&E stained colon sections from AIEC infected mice with or without 2'-FL pretreatment. Images are representative of six mice each of PBS controls, AIEC infected, and 2'-FL pretreated prior to AIEC inoculation. Red arrows indicate inflammatory cell infiltrates and epithelial cell sloughing induced by AIEC infection, which are not seen in colons from 2'-FL pretreated mice or saline controls. Bars= 50 μM.

Supplemental figure 7