

Figure S1. Gastric pathology

**Figure S1. Representative H&E images of stomach from male mice of different groups at 10 WPI and 21WPI.** A: Uninfected male at 10 WPI. B: uninfected male 21 WPI. C: mono-Hp male at 10 WPI. D: mono-Hp at 21 WPI. E: mono-Hh at 10 WPI. F: mono-Hh male at 21 WPI. G: Hh+Hp male at 10 WPI. G: Hh+Hp male at 21WPI. All sections showing normal gastric corpus and adjacent squamous –columnar junction with no discernible inflammation or oxyntic loss. BAR, all images: 150 $\mu$ M. Gastric pathology in females were similar to the corresponding groups of males.

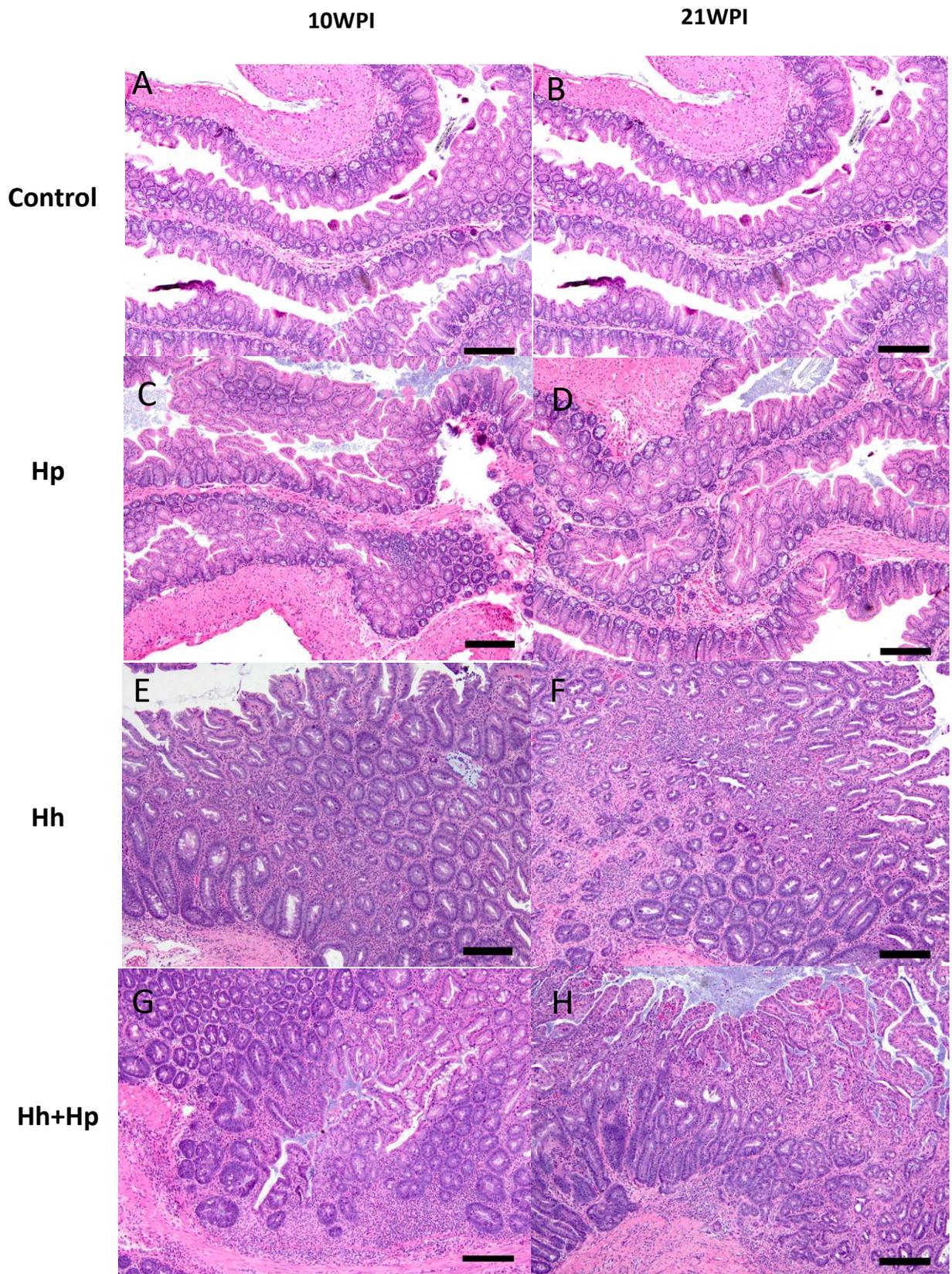


Figure S2. Cecal Histology

**Figure S2: Representative H&E images of cecum from male mice of different groups at 10 and 21WPI.** A: Uninfected at 10 WPI. B: uninfected at 21 WPI. C: mono-Hp at 10 WPI. D: mono-Hp at 21 WPI. E: mono-Hh at 10wpi. F: mono-Hh at 21 WPI. G: Hh+Hp at 10 WPI. H: Hh+Hp at 21 WPI. Panels A-D showing none to patchy lamina proprial inflammatory aggregates comparable across the four groups. Panel E showing severe diffuse mucosal and sub-mucosal inflammation (chiefly neutrophils and macrophages) with mild edema, glandular hyperplasia and mild dysplasia. Panel F showing severe mucosal and sub-mucosal inflammation, fibrosis, papillary hyperplasia and high grade dysplastic glandular proliferation with invasion and partial effacement of the muscularis mucosa. Panel G showing severe mucosa inflammation, mild erosions, crypt loss, prominent epithelial hyperplasia and high grade glandular dysplasia. Panel H showing severe inflammation and villo-papillary adenocarcinomatous epithelial proliferation with early invasion into the submucosa. BAR, all images: 150 $\mu$ M. The pathological features in the ceca of females were similar to the corresponding groups of males.

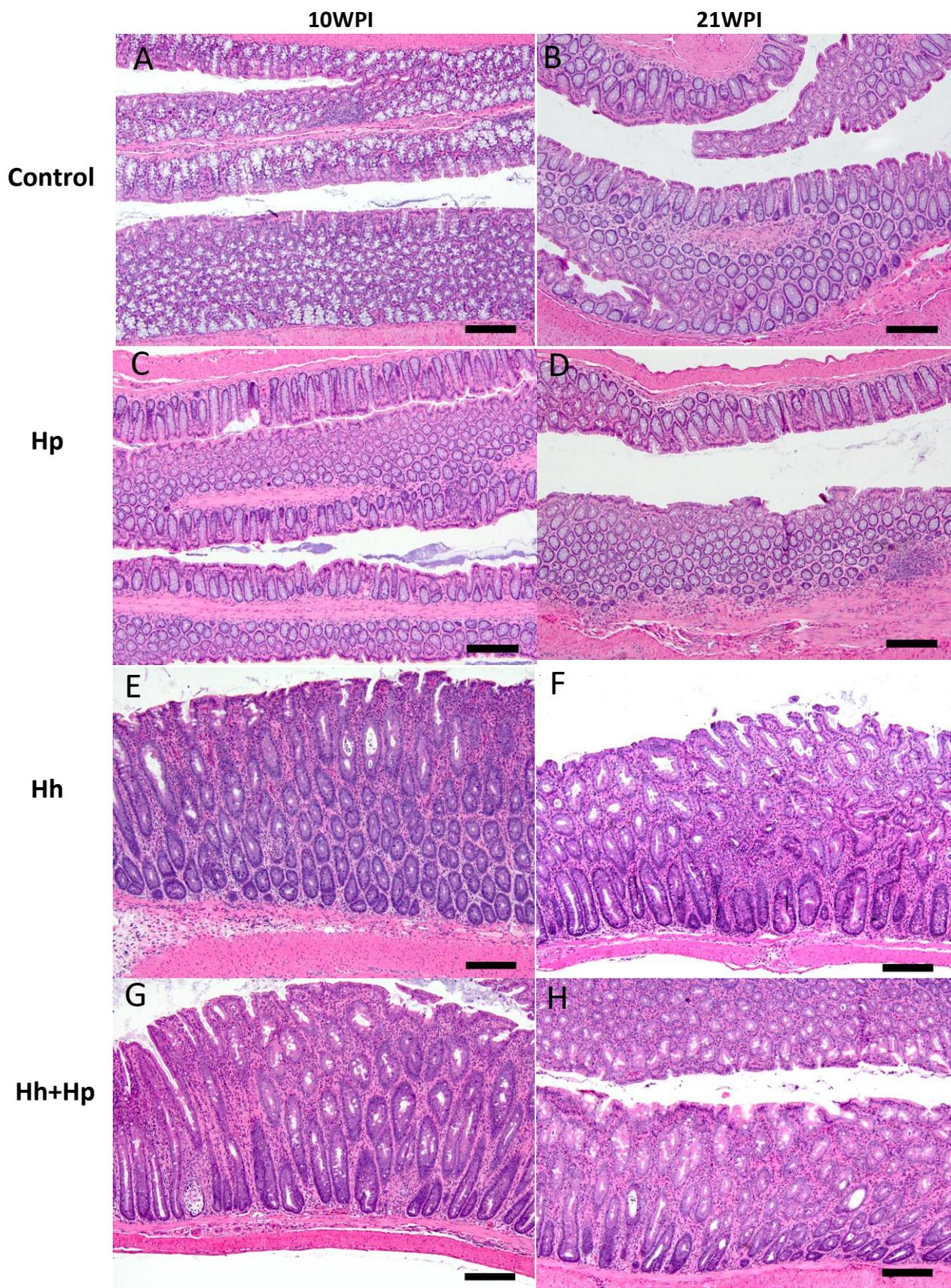


Figure S3. Female colonic Histology

**Figure S3: Representative H&E images of colon from female mice of different groups at 10 WPI and 21 WPI.** A: uninfected female at 10 WPI. B: uninfected female at 21 WPI. C: mono-Hp female at 10 WPI. D: mono-Hp female 21 WPI. E: mono-Hh female at 10 WPI. F: mono-Hh female at 21 WPI. G: Hh+Hp female at 10 WPI. H: Hh+Hp female at 21WPI. Panels A-D depict normal colons with no significant inflammation. Panels E-H that show moderate to severe inflammation, epithelial defects, moderate epithelial hyperplasia and mild to moderate dysplasia in mono-Hh groups at both time points. BAR, all images: 150 $\mu$ M.

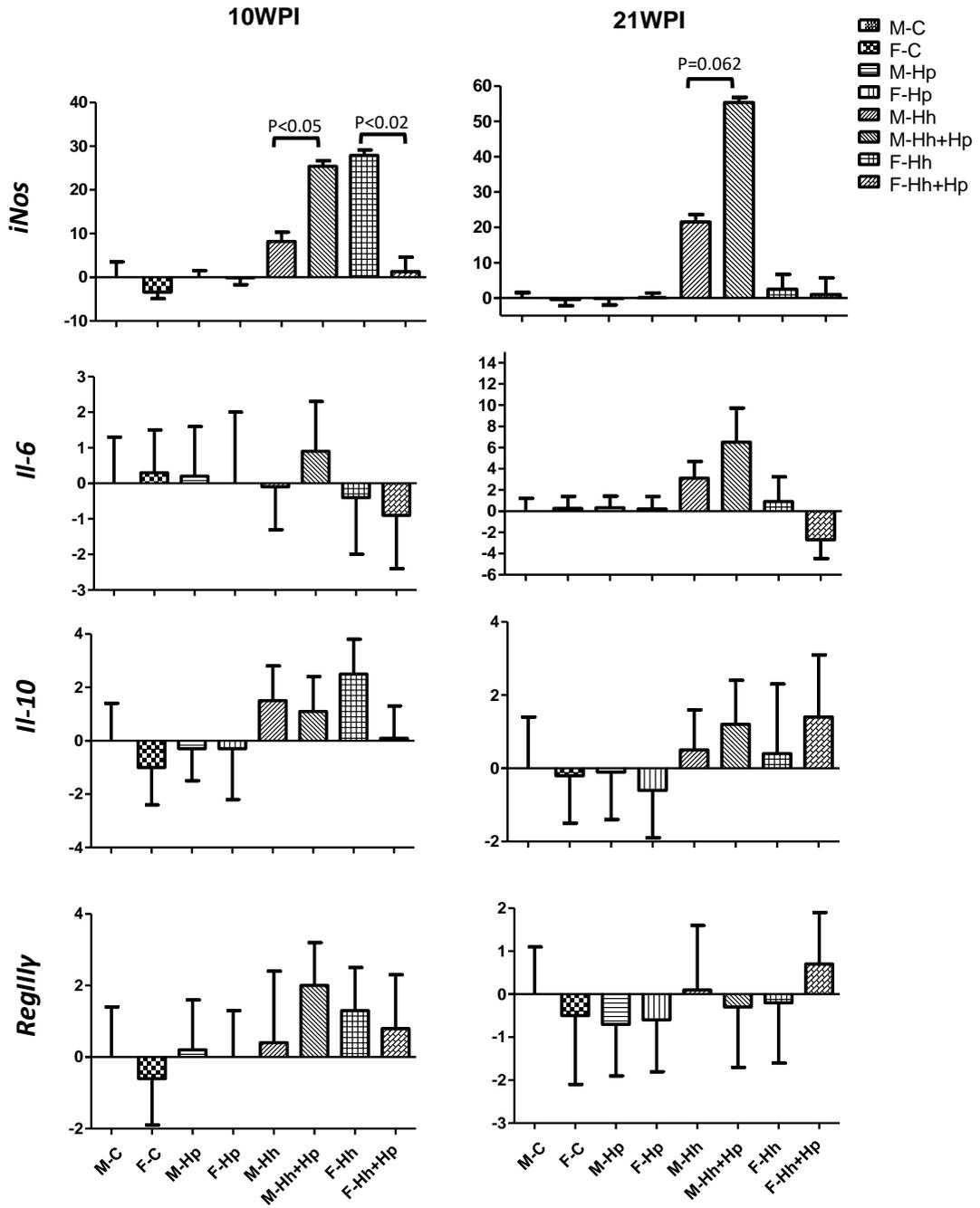
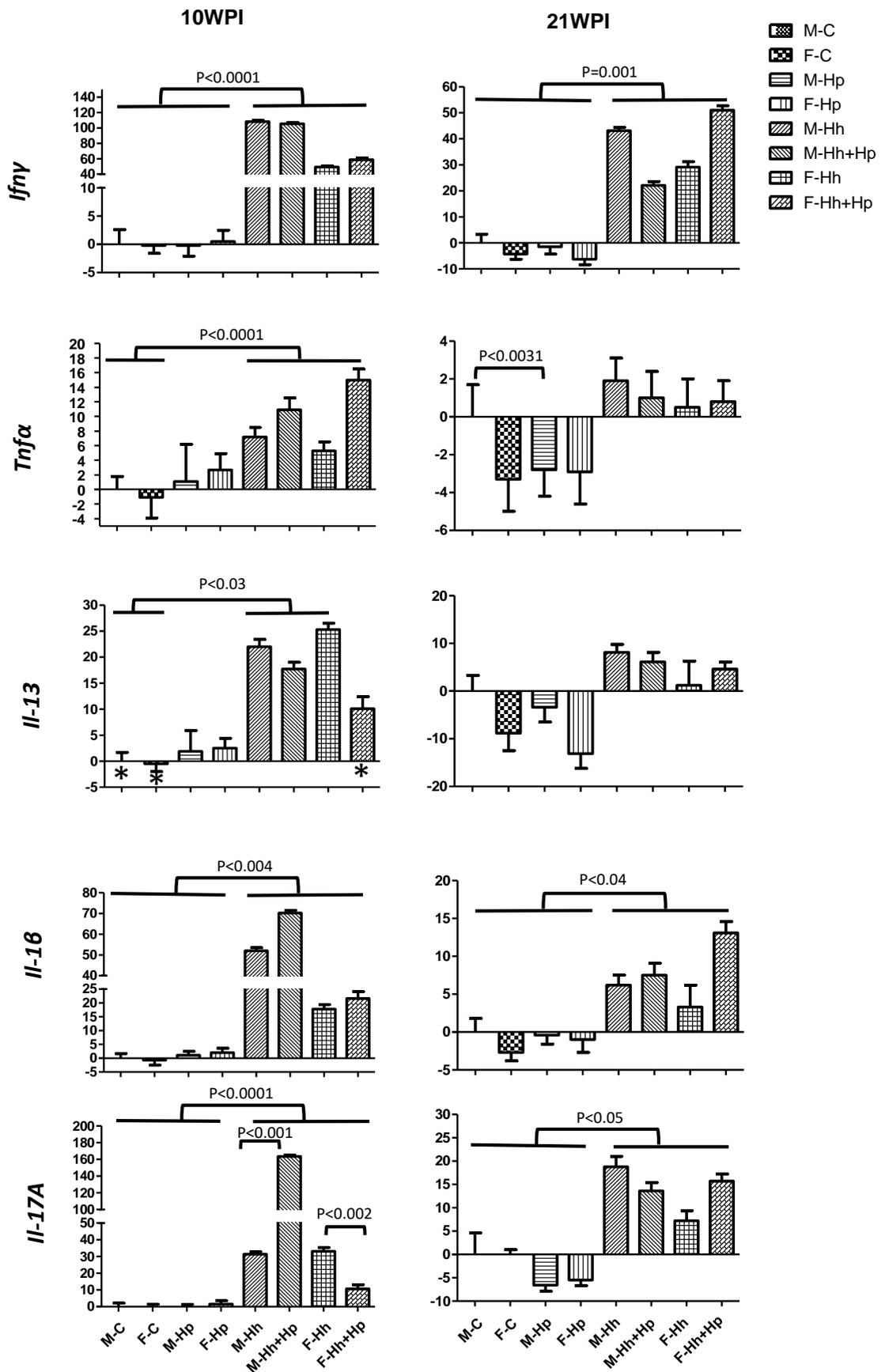


Figure S4

**Figure S4. Co-infection with Hp in males did not increase mRNA levels of genes being implicated in colonic carcinogenesis when compared to mono-Hh males.** Total RNA prepared from colonic tissues of mice infected or sham-dosed were evaluated by qPCR for expression levels of mRNA for select cytokines, which then were normalized to the expression of the house-keeping gene *Gapdh*. The Y axis represents the mean fold change ( $\pm$  standard deviation) of the mRNA levels in reference to uninfected male controls.



**Figure S5. Cecal gene expression  
(continued on the next page)**

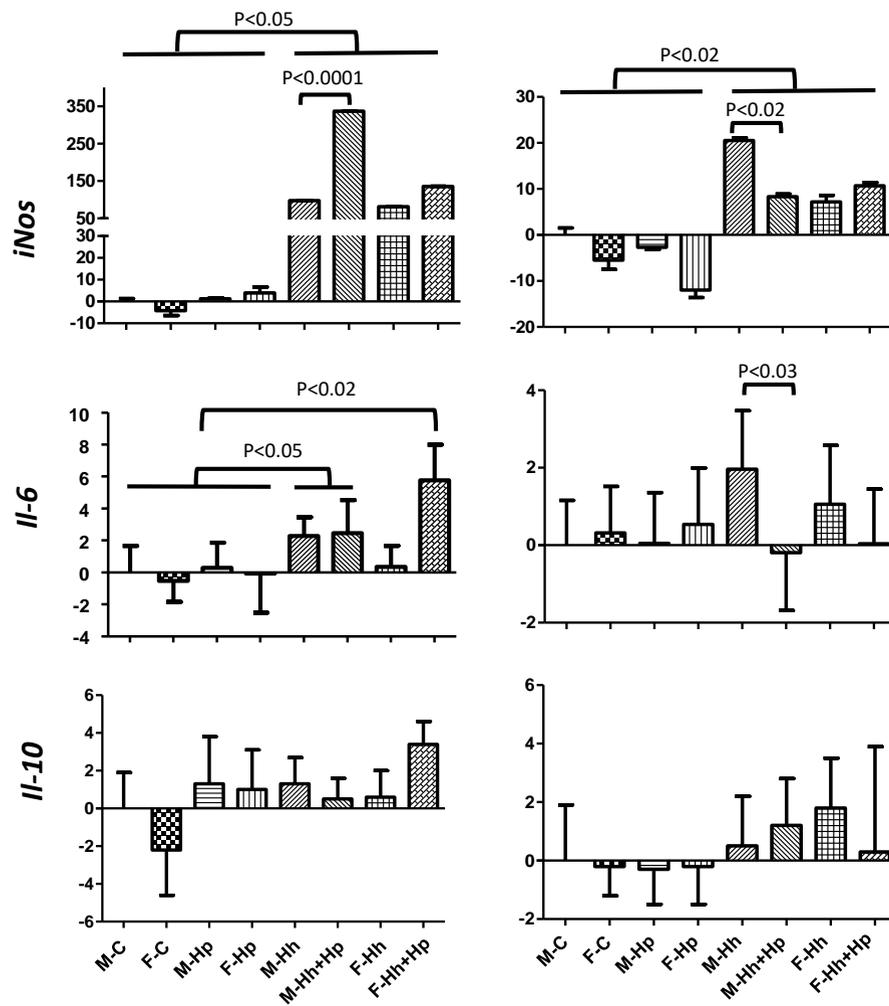


Figure S5. Cecal gene expression

**Figure S5. mRNA levels of cecal ILC1s and ILC3s-associated genes were not enhanced in Hh+Hp males compared to mono-Hh counterparts.** Total RNA prepared from colonic tissues of mice infected or sham-dosed were evaluated by qPCR for expression levels of mRNA for select cytokines, which then were normalized to the expression of the house-keeping gene *Gapdh*. The Y axis represents the mean fold change ( $\pm$  standard deviation) of the mRNA levels in reference to uninfected male controls.