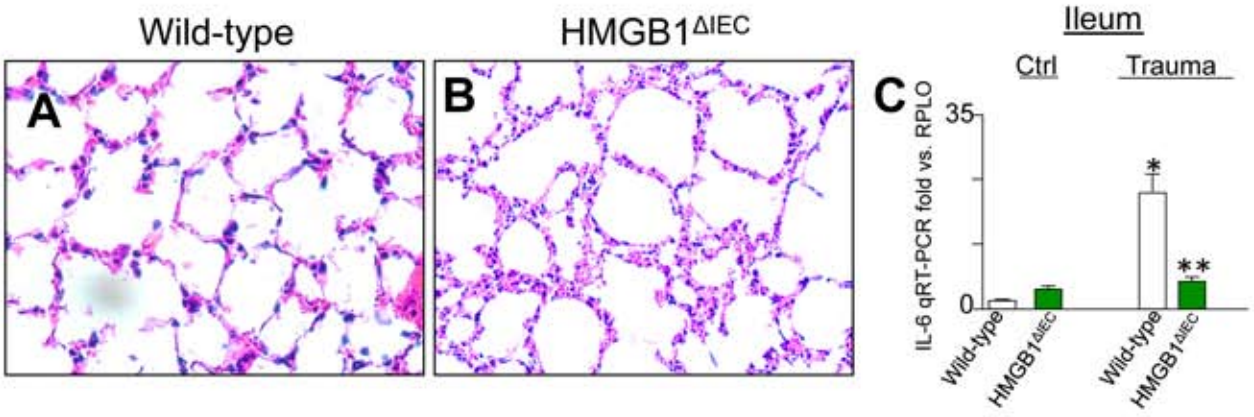
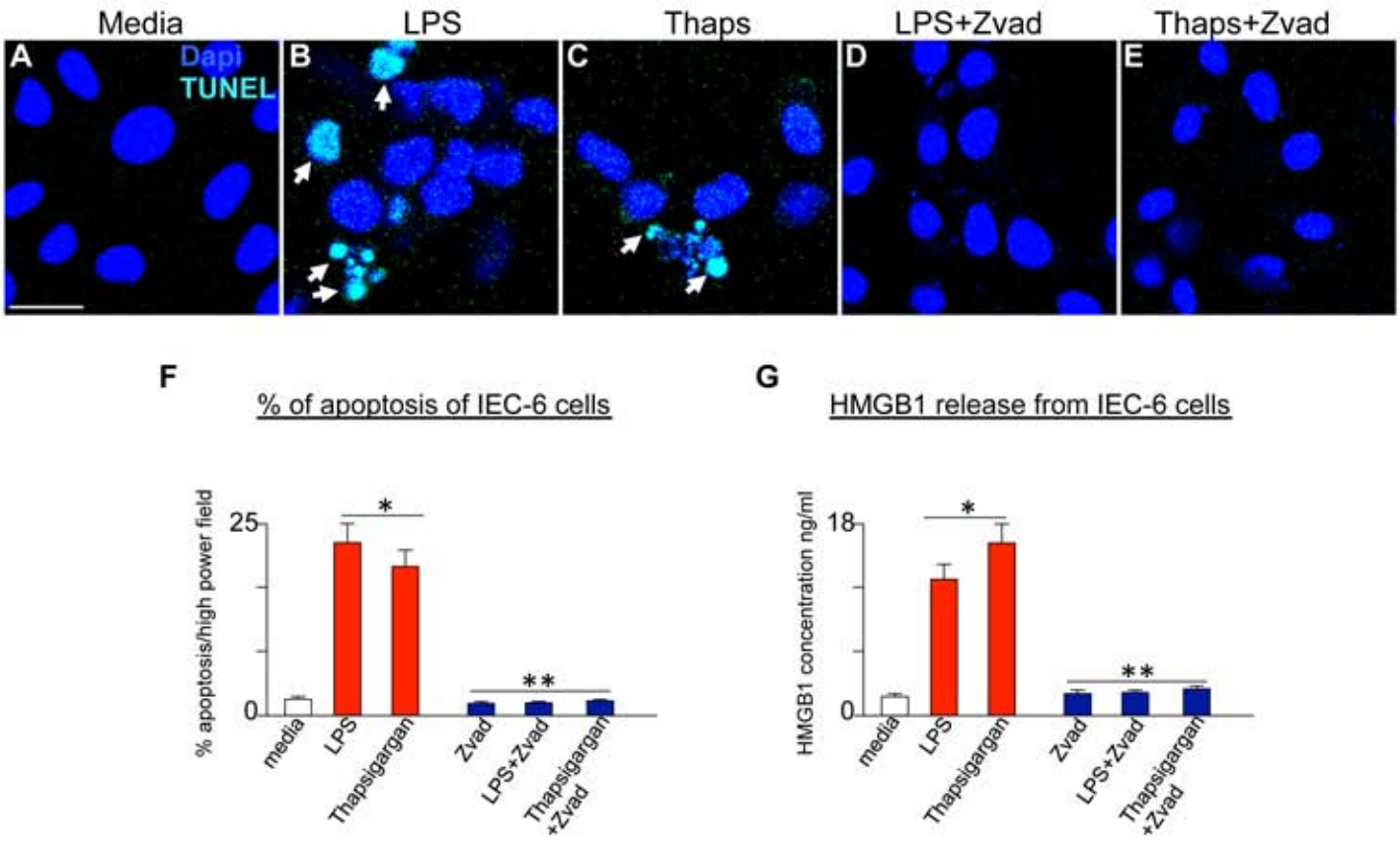
Supplemental Figure 1. Sodhi et al.



Supplemental Figure 1. *Mice lacking HMGB1 in the intestinal epithelium (HMGB1$^{\Delta IEC}$) are protected from trauma induced intestinal injury.* **A-B**: Representative micrographs of lung from wither wild-type (**A**) or HMGB1$^{\Delta IEC}$ (**B**) mice stained with H&E. **C**: qRT-PCR showing the expression of IL-6 in the ileum of wild-type (white bars), HMGB1$^{\Delta IEC}$ mice (green bars) that were either untreated (Ctrl) or exposed to trauma/hemorrhagic shock (Trauma). *p<0.05 control vs. trauma; **p<0.05 wild-type trauma vs. wild-type control or HMGB1$^{\Delta IEC}$ control; **p<0.05 wild-type trauma versus HMGB1$^{\Delta IEC}$ trauma; Representative of 3 separate experiments with over 5 mice per group.

Supplemental Figure 2. Sodhi et al.



Supplemental Figure 2. *The release of HMGB1 from IEC-6 cells is dependent on apoptosis.* **A-E:** Confluent IEC-6 enterocytes that were treated with either media (**A**), LPS (50 mg/ml for 6h, **B**), thapsigargin (Thaps, 0.5 mM, 6h, **C**), LPS (50 mg/ml for 6h) in addition to the pan-caspase apoptosis inhibitor Z-VAD-FMK (10mM, 30 min prior, **D**), or Thapsigargin + Z-Vad (**E**). Slides were stained for TUNEL and DAPI; arrows show apoptotic cells. F-G: Quantification of apoptosis per high power field (**F**) and concentration of HMGB1 in the media as measured by ELISA (**G**) under the indicated conditions; *$p<0.05$ vs. media (i.e. red vs. white bars); **$p<0.05$ vs. LPS or thapsigargin (i.e. blue vs. red bars); representative of 3 separate experiments with over 100 high power fields examined per experimental condition.