SUPPLEMENTARY FIGURES



FIGURE S1. Pattern of Gpnmb expression in mouse kidney, other mouse tissues and macrophages. (*A-D*) Control antibody (*A*) or anti-N terminal Gpnmb detection in d7 post IRI kidney outer medulla (*B-C*) [T-tubule]. Note intratubular phagocytes [arrowhead].

Co-labeling for macrophages [arrowheads] (D) in inner medulla shows positivity in intracellular compartments. Anti-N terminal Gpnmb detection normal lung (E) showing positive staining in alveolar macrophages. (F) Low-power view of spleen showing high expression of Gpnmb in the red pulp [R], and only a few positive cells in white pulp [W], identified as tingible body macrophages (arrows). (G) Gpnmb (red) and CD68 (green) labeling of aortic arch atheromatous plaque from d165 ApoE-/- mice fed on high fat diet. The aortic lumen is denoted [L]. Gpnmb and CD68 co-localize in lesional macrophages. (H) Immunofluorescence for Gpnmb (red) and macrophage marker CD68 (green) in fibrotic scars in mouse livers after 12 weeks of CCl_4 injury. Note large round phagocytes (long arrow) co-express CD68 and Gpnmb whereas smaller CD68 +ve Mos (small arrow) and Kupffer cells (short arrow) lack Gpnmb. (I) Western blot of whole cell lysates from BMMøs detecting Gpnmb using anti-C terminal antibodies (upper) or comparing both anti-N and anti-C termini antibodies (lower). (J) Detection of Gpnmb in unstimulated or 24h stimulated BMMøs whole cell lysates. Note that both IFNy and LPS down-regulate Gpnmb expression. (K) Detection of Gpnmb in blood monocytes compared with BMM ϕ s (bar = 50 μ m).



FIGURE S2. Characterization of autophagosomes in kidney tubule cells *in vivo* and *in vitro*. (*A*) Confocal images of antibody detected endogenous LC3 in normal proximal tubule of rat kidney and 5d post IRI. Note redistribution of LC3 in the injured tubules from a cytosolic and basement membrane distribution to an endosomal distribution. (*B*) RT-PCR for Atg protein transcripts from healthy proximal tubule cells purified from mouse kidney 'C' or injured proximal tubules purified from d5 post IRI kidney'I'. Note that both *Atg5* and *Atg7* are upregulated following injury. (*C*) Fluorescent micrographs of

Gpnmb-RFP+ expressing LLC-PK1 kidney epithelial cells at timepoints following chloroquine treatment. Note rapid accumulation and expansion of Gpnmb-RFP+ vesicles.(D) Immunoblot detecting Gpnmb-RFP in Gpnmb-RFP cells at timepoints after chloroquine treatment.



FIGURE S3. Acidification of apoptotic body containing phagosomes is delayed in *Gpnmb*^{-/-} peritoneal macrophages *in vivo*. Graph showing the % phagocytosis by d4 thioglycollate peritoneal macrophages at 30min and 4h after IP injection of 10million apoptotic thymocytes (solid lines) and also the % of macrophages with lysotracker red +ve acidified phagosomes. Phagosomes in *Gpnmb*^{+/+} macrophages were observed to undergo acidification while those of *Gpnmb*^{-/-} macrophages did not (n=4/group * P < 0.05).



FIGURE S4. Characterization of Gpnmb compartments in macrophages. (A) Confocal images of Gpnmb (green) and lysotracker red in control or chloroquine treated BMMφs.(B) Confocal images for Atg12 and CD68 in control BMMφs