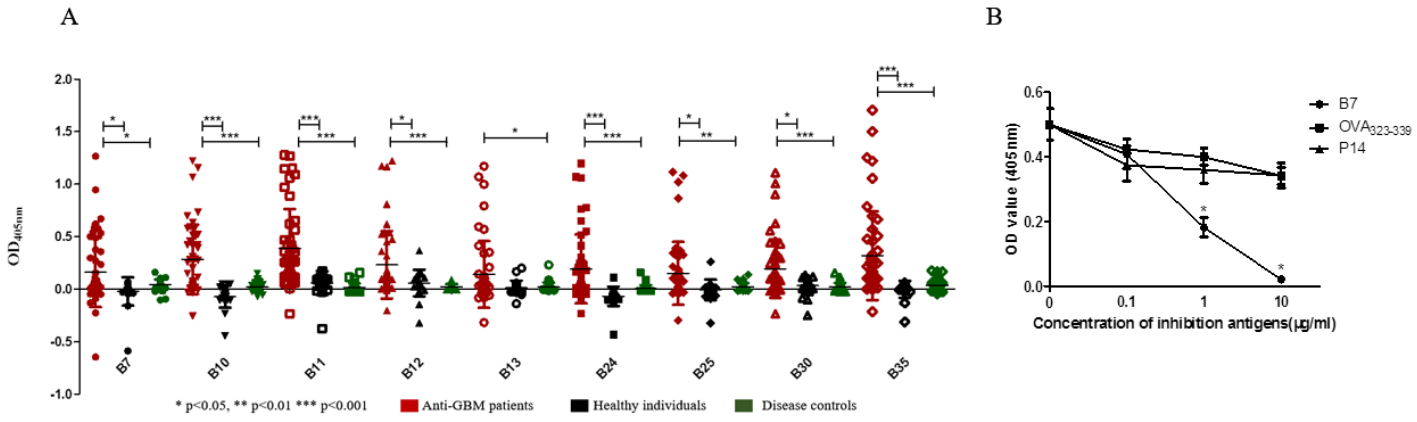
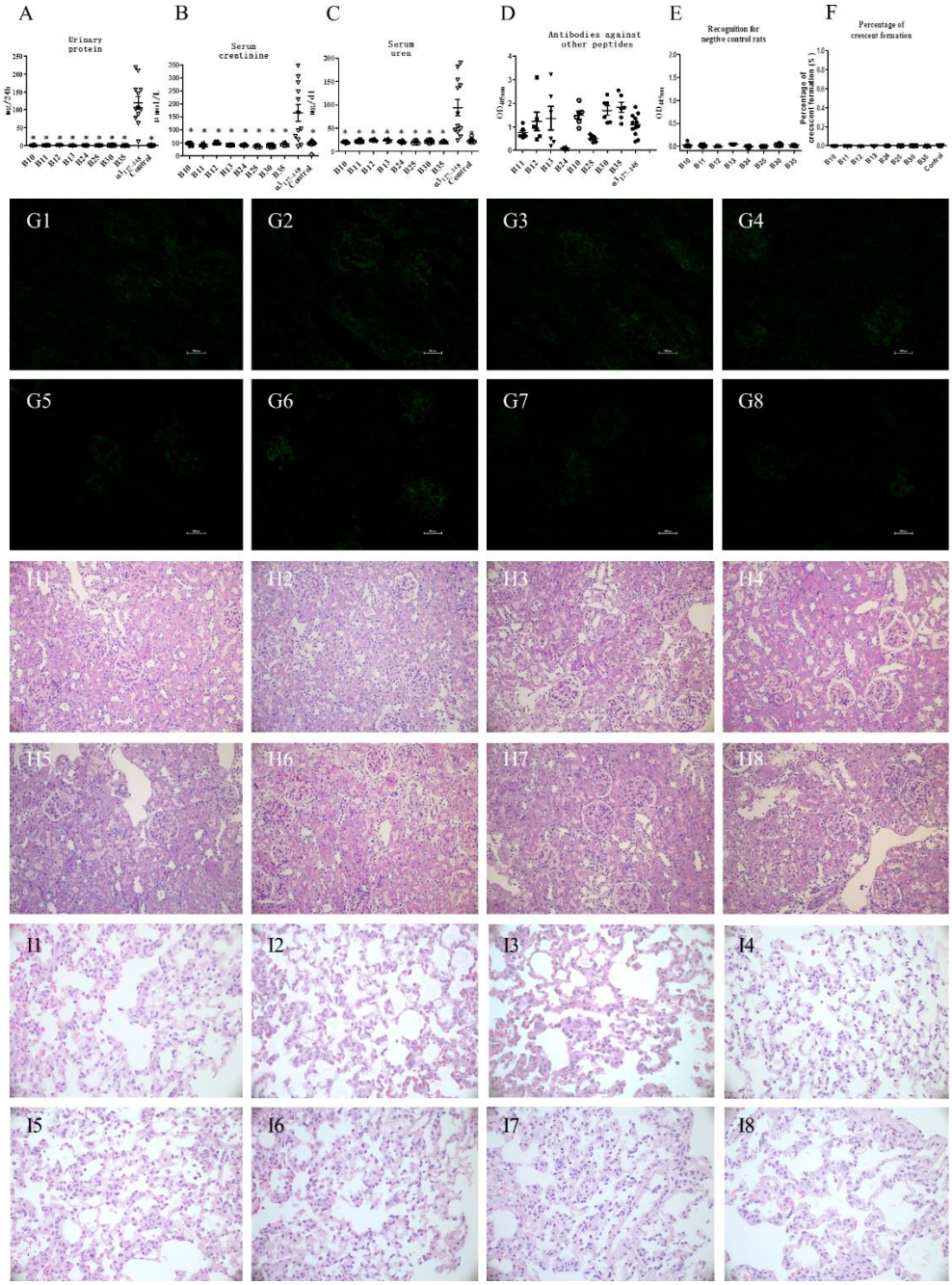
Supplemental Table of Contents

Supplementary Figure 1. The specificity of microbial peptides recognition by anti-GBM patients. The peptides could be recognized by sera from anti-GBM patients, while the recognition in healthy individuals and disease controls was scarcely seen (A). Competitive inhibition assay was performed and the results showed that the binding of anti-B7 antibodies to B7 could be inhibited by B7 but not by P14 (B).



Supplementary Figure 2. Peptides B10, B11, B12, B13, B24, B25, B30 and B35 couldn't induce any kidney or pulmonary lesion in WKY rats. The 8 peptides were injected to WKY rats by the same protocol as B7. Seven of them developed antibodies respectively (D), but the levels of proteinuria (A), serum creatine (B), BUN (C) and percentage of crescents (F) were all comparable to negative controls. The negative control rats didn't develop any antibodies to these peptides (E). No linear deposit of IgG (G), crescentic formation (H), or pulmonary lesion (I) was shown after these peptides' immunization.



Supplementary Figure 3. Actinomyces lysate protein and recombinant UDP-N-acetylenolpyruvoylglucosamine reductase protein couldn't induce anti-GBM disease in WKY rats. Actinomyces lysate protein at 1mg/kg, and recombinant UDP-N-acetylenolpyruvoylglucosamine reductase protein (RP) at 10/5/2/1mg/kg were injected to WKY rats by the same protocol as B7. All rats developed antibodies towards the specific antigen (E). Serum creatinine (B), serum urea (C) and glomerular crescent (D) were all comparable to negative controls. No IgG deposits along GBM (F), crescent formation (G) or pulmonary lesion (H) was observed. 1-6 represented Actinomyces lysate protein, 10mg/kg RP, 5mg/kg RP, 2mg/kg RP, 1mg/kg RP and negative control group respectively.