**Background** Properdin, released predominantly from neutrophils, is the only known positive regulator of complement alternative pathway via stabilizing C3bBb and amplifying activation. This study, aimed to explore the effect and the dynamic change of properdin on renal ischemia-reperfusion injury (IRI) and repair.

**Methods** IR-related injury was established in mouse kidneys *in vivo* subjected to 30-min ischemia followed by 6-72 h, and 1, 2 and 8 w reperfusion, and mouse renal epithelial cells (TCMK-1) *in vitro* via the stimulation of 200 μM H2O2 for 24 h. In addition, the modification of properdin was performed by either intraperitoneally injected CHBP (24 nmol/kg) into mice after reperfusion or pre-treated TCMK-1 cells with siRNA (15 μmol/l). Finally, the changes of properdin mRNA and protein, complement activation, apoptosis and inflammation-associated factors were assessed, and the correlations between properdin and these parameters were further analysed.

**Results** The level of serum creatinine (SCr) and the score of tubulointerstitial damage (TID) were gradually increased by IRI and peaked at 48 h. The expression of properdin, apoptosis and inflammation related factors HMGB 1 and caspase-3 was increased in a time-dependent manner, and peaked from 12 to 72 h. Interestingly, propertin mRNA was raised from 72 h when acutely increased properdin protein started to decline. Properdin protein was positively correlated with HMGB1, caspase-3, renal function and histology. Furthermore, the increased properdin in IR kidneys was reversed by CHBP treatment at 48 h and 2 w. In addition, the same change trend in properdin protein was seen in TCMK-1 cells stimulated by H2O2, whilesiRNA target properdin reduced properdin mRNA and protein, but increased the protein expression of HMGB 1 and caspase-3.

**Conclusions** The acute increase and then decrease of properdin was seen in IRI mouse model and renal epithelial cells. This dynamic change of properdin was associated with renal function and structure, as well as inflammation and apoptosis mediators. The expression of properdin could be modified by CHBP and its siRNA in mouse IRI kidneys and cells, but its underlying significance and mechanisms, especially the involvement of properdin in IRI and recovery, are worthy to be further investigated.