Figure S1

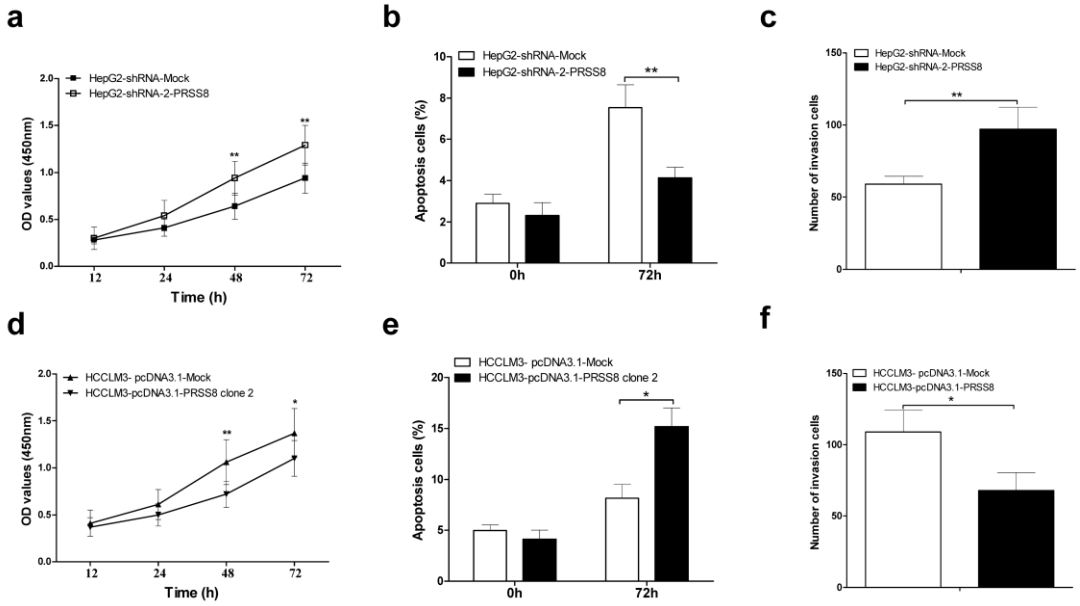

Figure S1. Silencing of PRSS8 promotes, while overexpression of PRSS8 inhibits HCC cell growth and invasion. PRSS8 was silenced in HepG2 cells using lentivirus-delivered shRNA-2. **a.** The cell viability influenced by PRSS8 was assessed by time course CCK-8 assay. **b**. The cell apoptosis influenced by PRSS8 was assessed by Annexin V/PI apoptosis detection assay. **c.** The cell invasion influenced by PRSS8 was assessed by Matrigel precoated Transwell assay. PRSS8 was overexpressed in HCCLM3 cells using the pcDNA3.1 plasmid. The cell viability (**d**), cell apoptosis (**e**) and cell invasion (**f**) were measured. Data are shown as mean ± SD from three independent experiments. $^{*}P<0.05$, $^{**}P<0.01$.

Figure S2

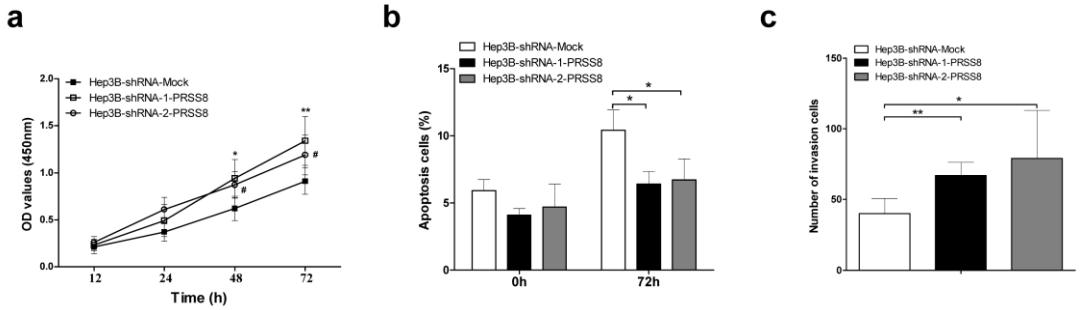

Figure S2. Silencing of PRSS8 promotes HCC cell growth and invasion in Hep3B cells. PRSS8 was silenced in Hep3B cells using two lentivirus-delivered shRNAs. The cell viability (**a**), cell apoptosis (**b**) and cell invasion (**c**) were measured. Data are shown as mean ± SD from three independent experiments. $^{*}P<0.05$, $^{**}P<0.01$.

Figure S3

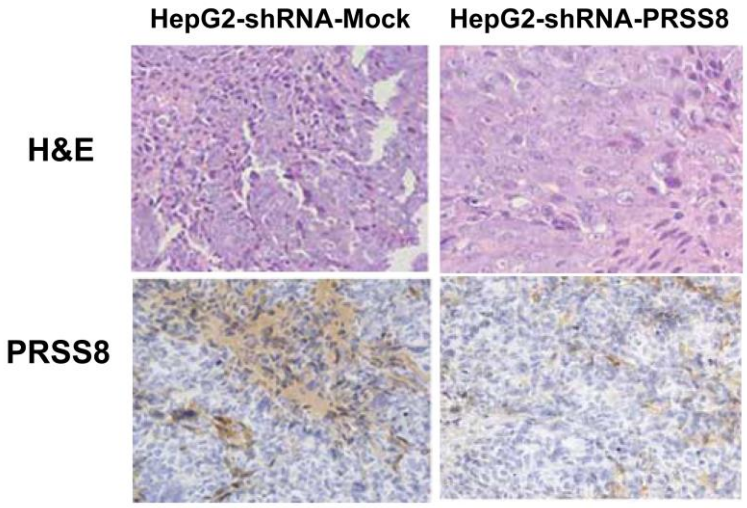

Figure S3. Staining with hematoxylin and eosin (H&E) or immunohistochemical staining for PRSS8 in HepG2-shRNA-Mock and HepG2-shRNA- PRSS8 cell derived xenografted tumors in mice at the experimental end point.