Recent evidence shows that IgA contributes to aggravating inflammation or dismantling antitumor immunity in diseased liver.

In this study, we demonstrate that there is an increase in serum IgA levels in HCC patients, which is associated with intrahepatic infiltration of inflammatory IgA⁺PD-L1^high^ monocytes.

IgA complex binds to monocytes and induces intracellular signaling cascade including YAP-TAZ pathway, resulting in the upregulation of PD-L1 and activation of the cells.

Our findings suggest that targeting IgA signaling in chronically inflamed, fibrotic livers may enhance anti-tumor immune response by suppressing inflammatory IgA⁺PD-L1^high^ monocytes.