

Figure S1 Hepatocytes interacted more strongly with other cell types than with T/NK cells. (A,B) Hepatocytes had stronger and more numerous interactions with other cell types than did T/NK cells. (C) Fibroblasts interacted with T/NK cells and B cells mainly via MIF-CD74 and MIF-CXCR4. (D,E) C4 showed more interaction events (higher interaction count) with other subclusters, but the interaction strength (intensity) was relatively low. (F) C6 interacted via FN1-SDC1 and FN1-SDC4, while C5 interacted with C4 via VTN-(ITGAV + ITGB5) and VTN-(ITGAV + ITGB1). (G) The VTN-mediated pathway was used to visualize the interactions between cell subclusters. NK, natural killer.

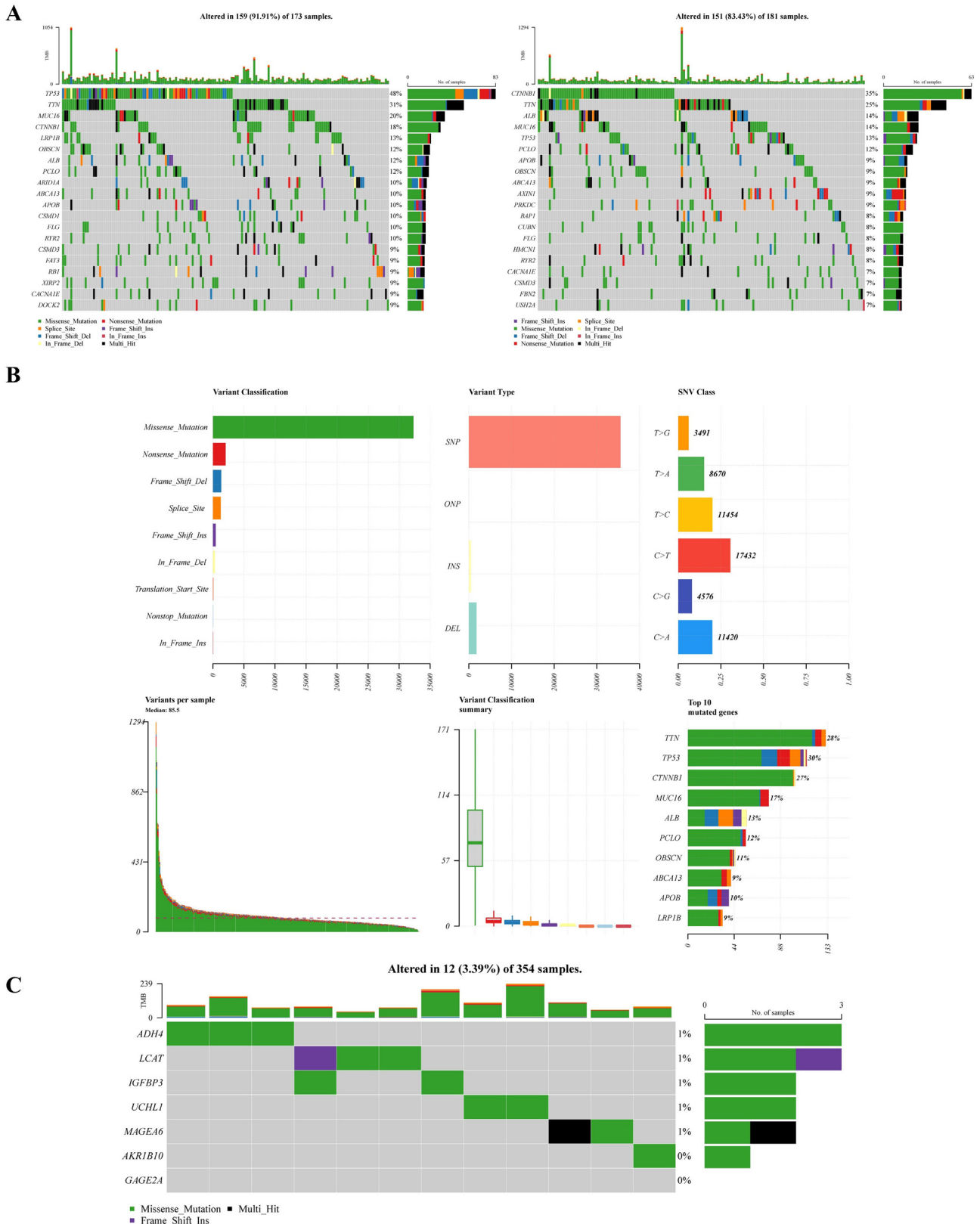


Figure S2 Figure S2 Missense mutations in each of the seven prognostic genes. (A) Mutation analysis in high- and low-risk groups identified different patterns. High-risk group top mutants were *TP53* (48%), *TTN* (31%), *MUC16* (20%), *CTNNB1* (18%), and *LRP1B* (13%), while low-risk group top mutants were *CTNNB1* (35%), *TTN* (25%), *ALB* (14%), *MUC16* (14%), and *TP53* (13%). (B) Common mutation types in HCC were splice site, frame shift ins, in-frame del, missense, and nonsense. (C) All seven prognostic genes showed missense mutations, and *LCAT* also had frame shift ins. HCC, hepatocellular carcinoma.