Supporting Information

Anti-hepatoma activity of the stiff branched β-D-glucan and effects of molecular weight

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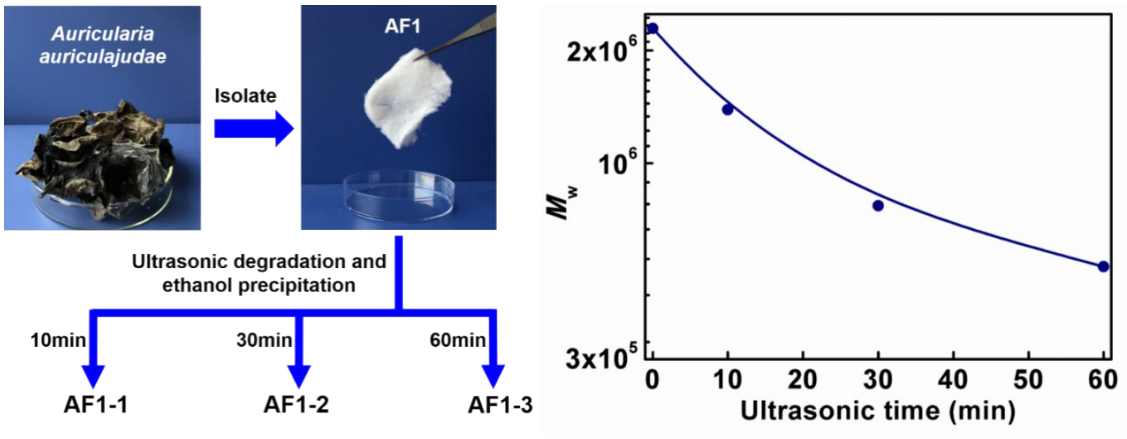


Fig. S1 The fractionation process of polysaccharides AF1 from the *Auricularia auriculajudae* (left) and the ultrasonic time dependence of M_w for the AF1 fractions in water at 25 ◦C (right).

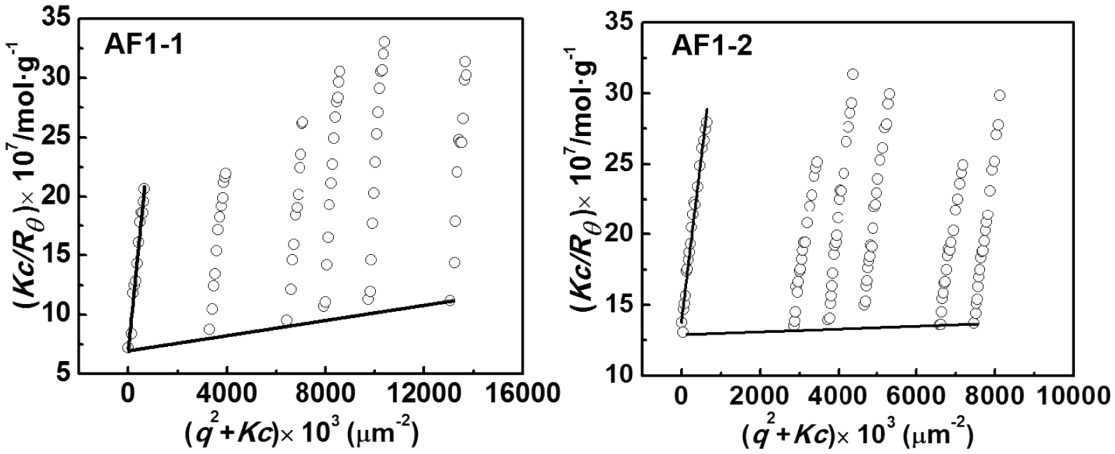


Fig. S2 Zimm plots of AF1-1, AF1-2 solution in water at 25 ◦C.

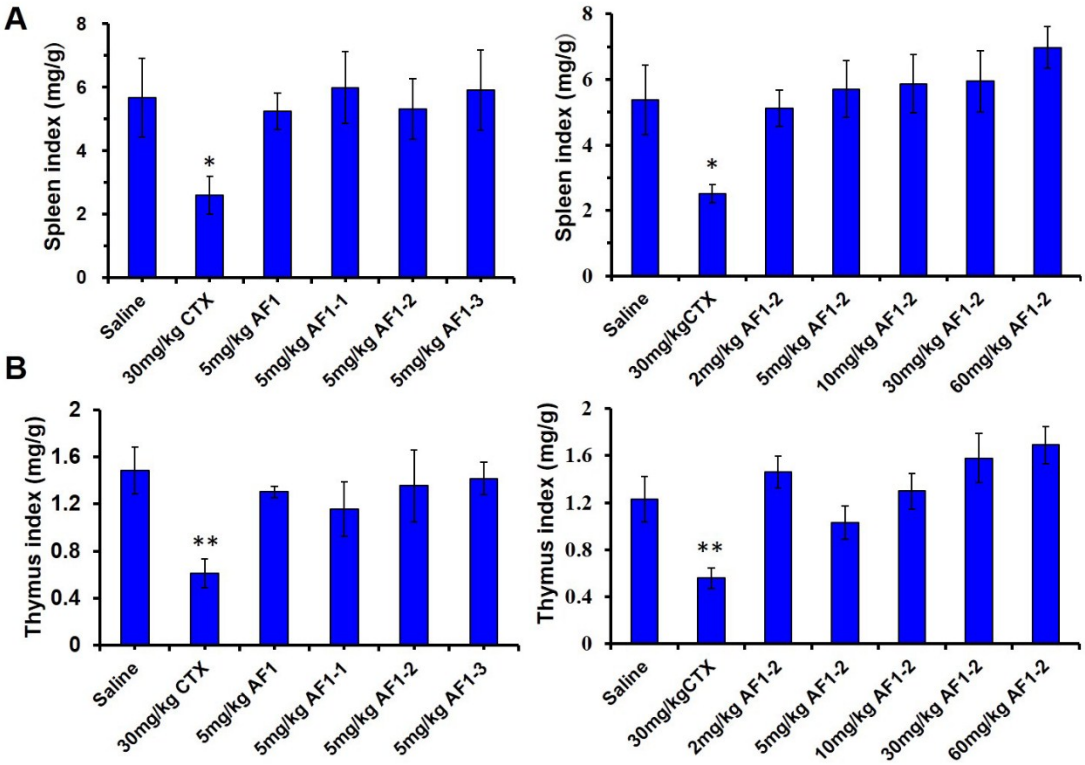
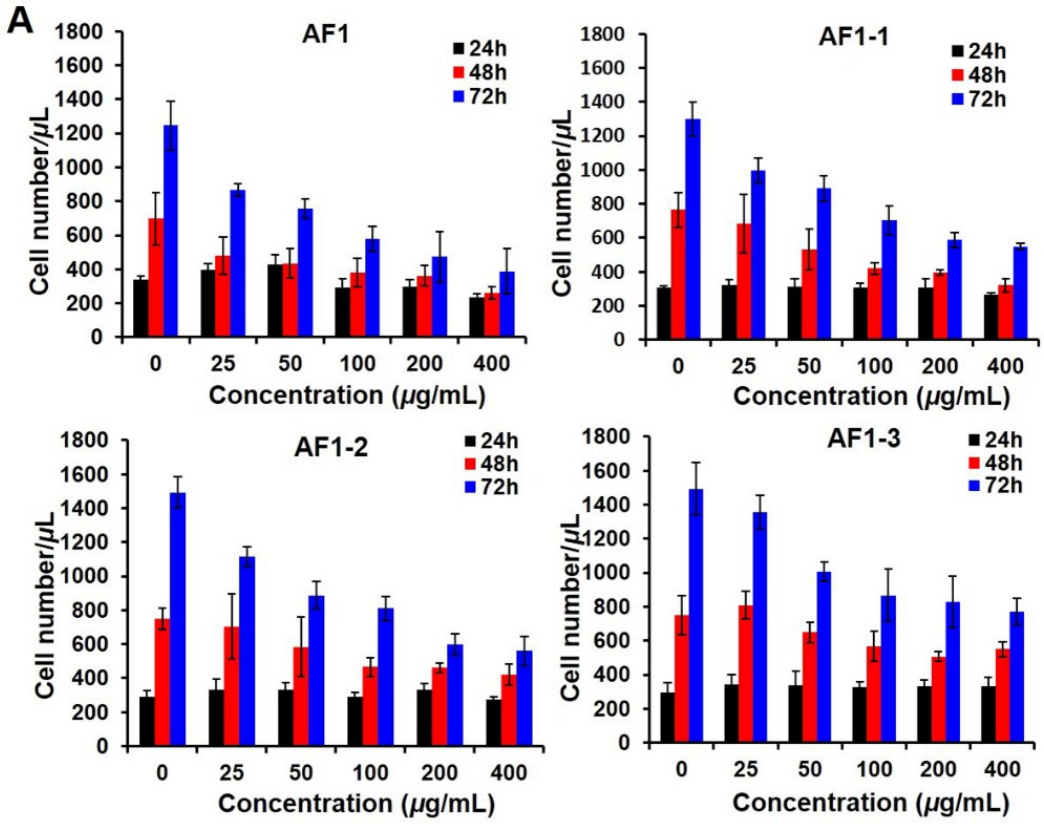


Fig. S3 Anti-hepatoma activity of the AF1, AF1-1, AF1-2 and AF1-3 samples in vivo. (A) Spleen index (mg/g) of different group, **p*<0.05 when compared with control. (B) Thymus index (mg/g) of different group, **p*<0.05 when compared with control. Indicated values were mean ± SD (n = 6).



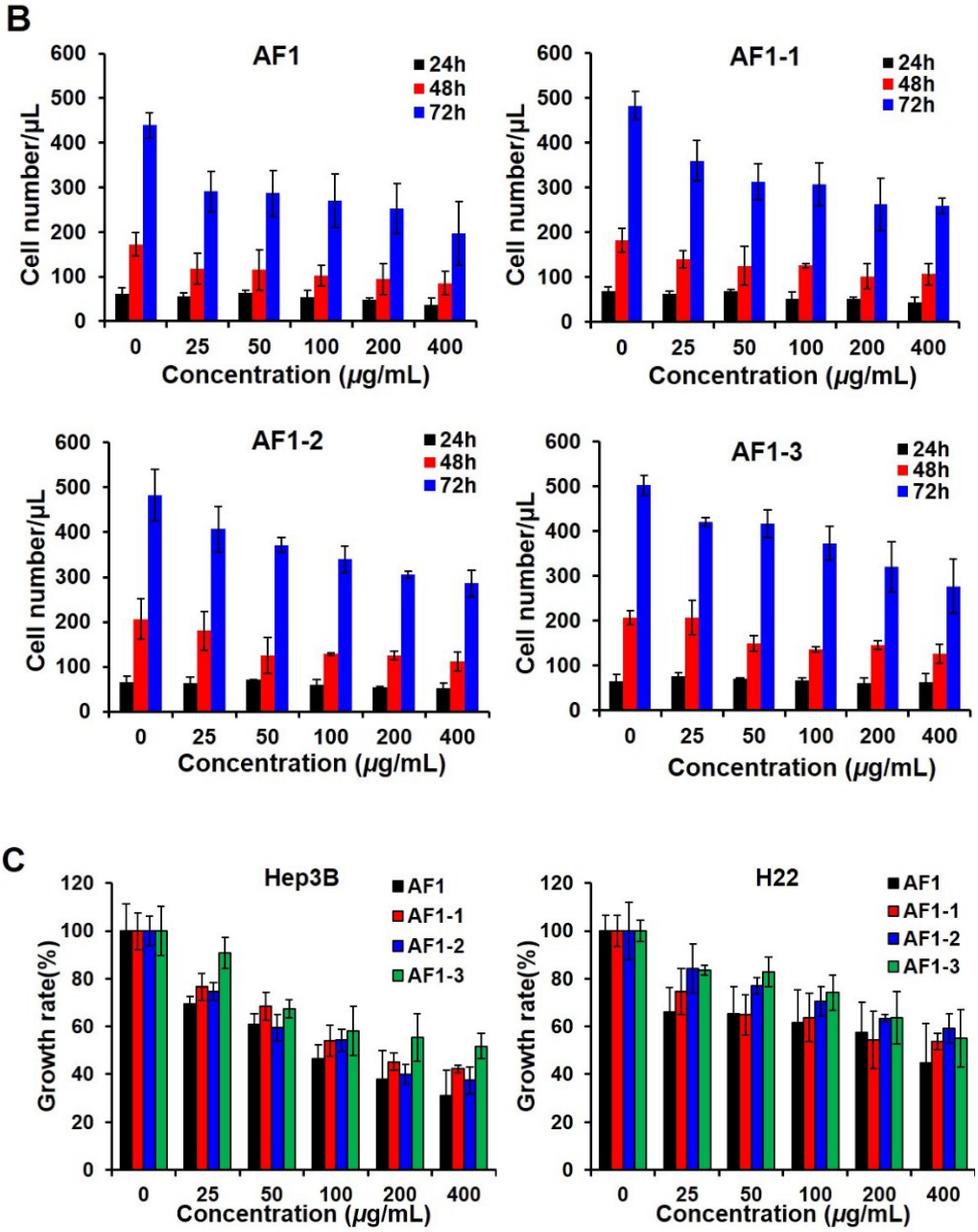
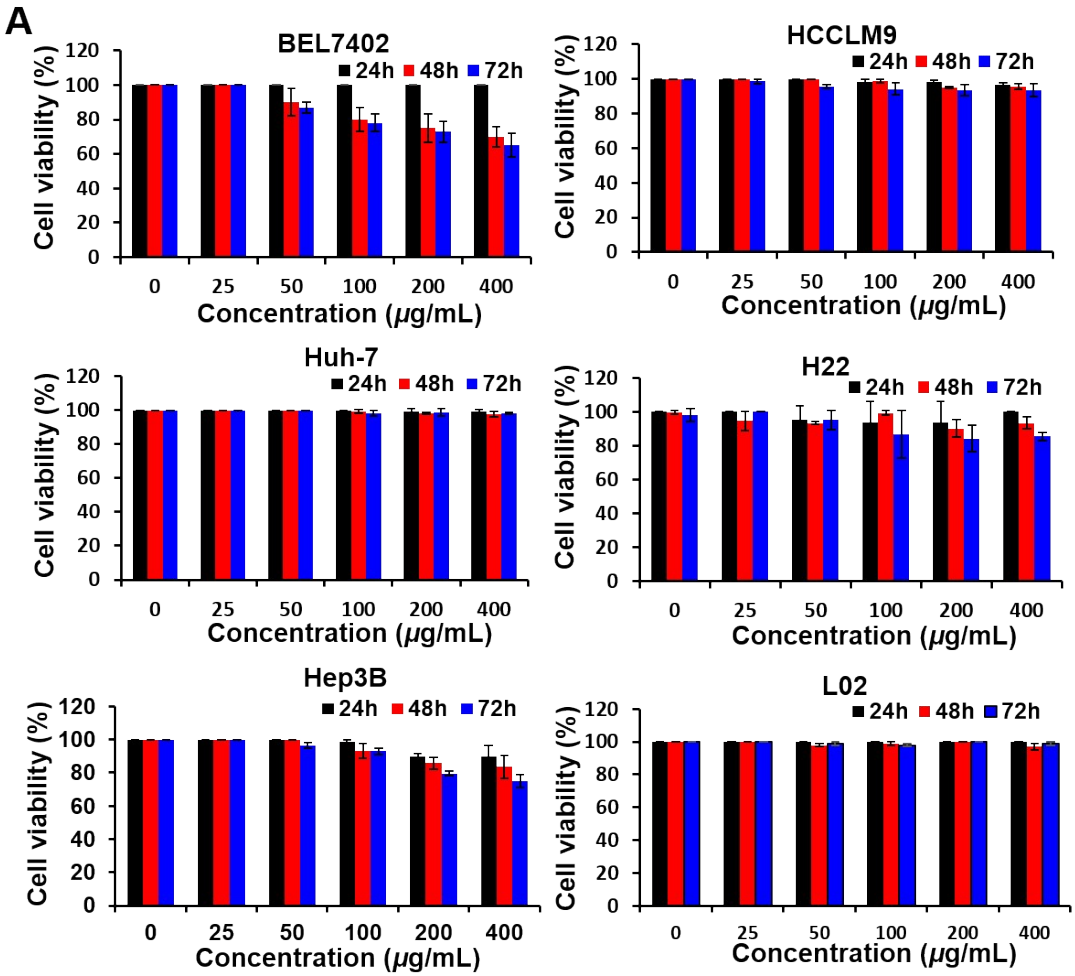


Fig. S4 Cell proliferation inhibition effects of the AF1, AF1-1, AF1-2 and AF1-3 samples on liver cancer cells. Liver cancer cell lines Hep3B (A), H22 (B) were treated with indicated concentrations of the AF1, AF1-1, AF1-2 and AF1-3 samples for 24, 48 and 72 hours. (C) The growth ratio of Hep3B, H22 were treated with indicated concentrations of the AF1, AF1-1, AF1-2 and AF1-3 samples for 72 hours. Cell proliferation was measured by counting the total number of cells. The data represent the average of at least three independent experiments ± SD.


A
BEL7402
HCCLM9
Huh-7
H22
Hep3B
L02
24h
48h
72h
Cell viability (%)
0
20
40
60
80
100
120
0
25
50
100
200
400
Concentration (μg/mL)

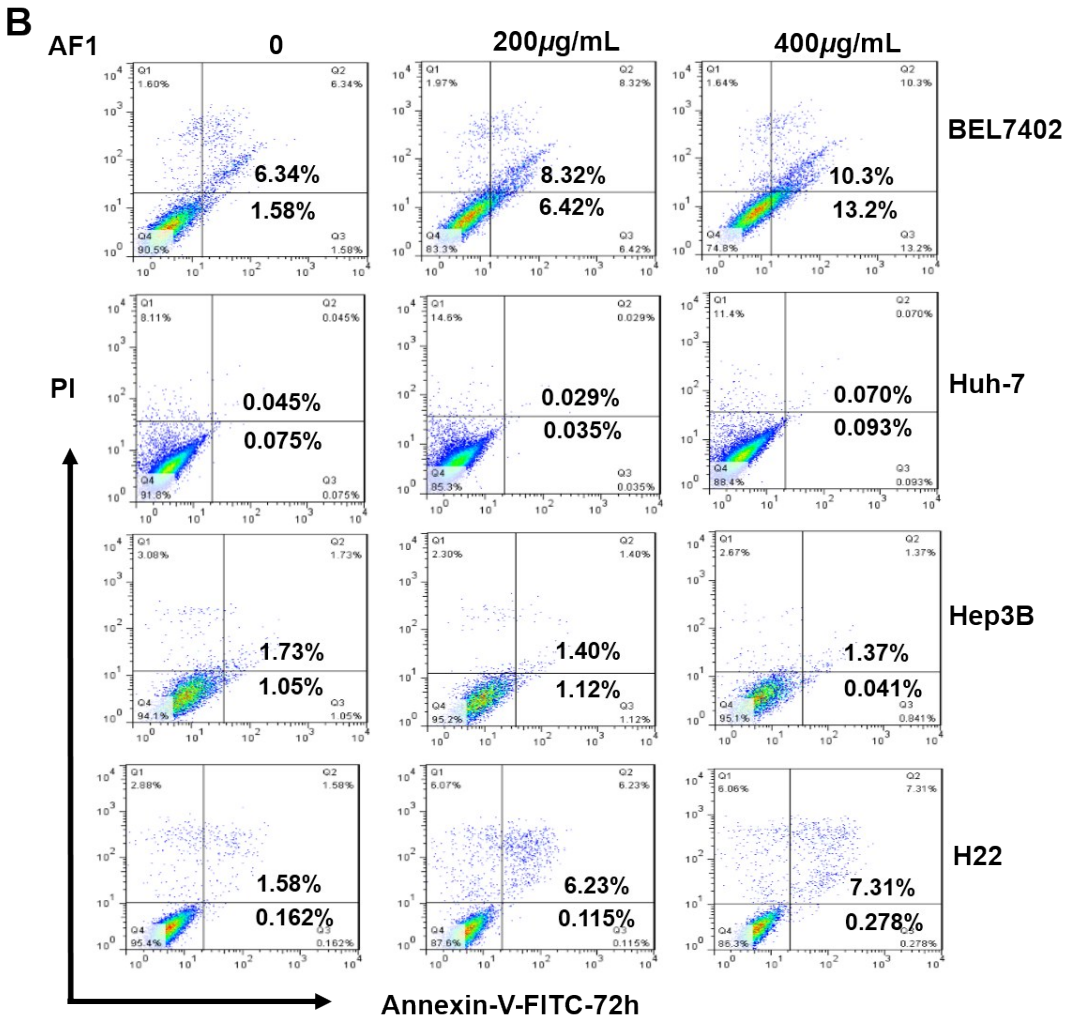


Fig. S5 Cell proliferation and viability effects of AF1 on liver cancer cells. (A) Cell viability was determined by trypan blue exclusion assays. (B) BEL7402, Huh7, Hep3B, H22 were treated with 0, 200μg/mL, 400μg/mL concentrations of AF1 for 72 hours. Apoptosis was determined by annexin V-FITC/PI staining. The data represent the average of at least three independent experiments ± SD.