Bitter melon extract attenuating hepatic steatosis may be mediated by FGF21 and AMPK/Sirt1 signaling in mice

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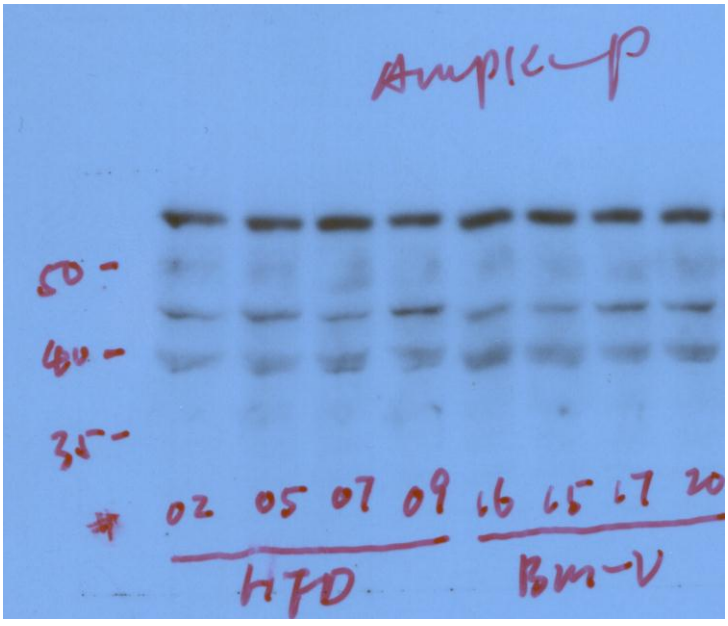
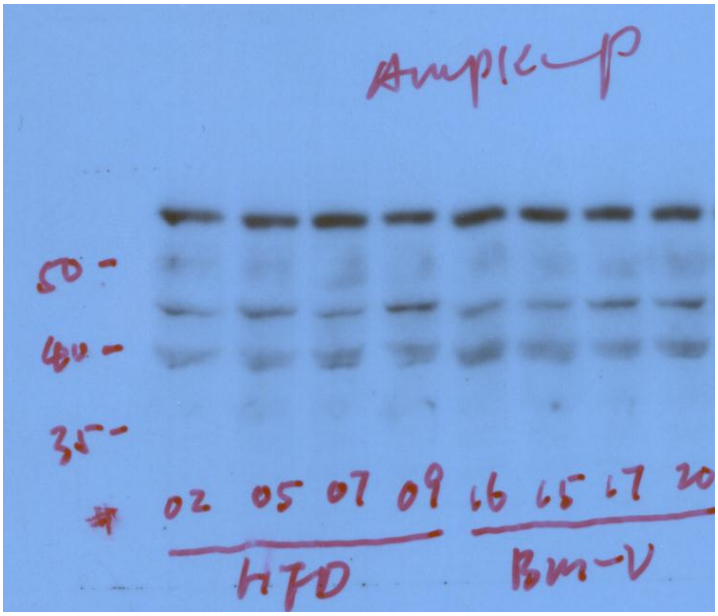
Table 1 The components of BM-V extract analyzed using a HPLC/LC-MS

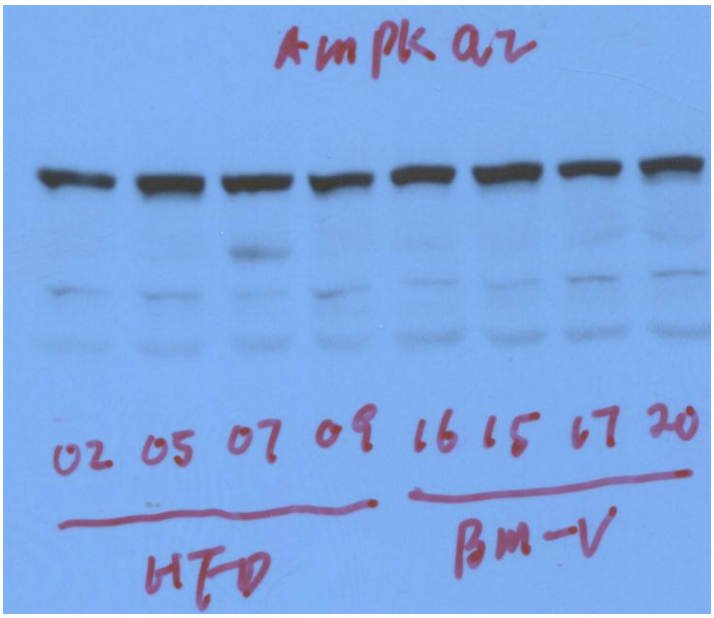
| Momordicosides (Verdure MCA015EPPH) | A | F1 | G | K | L | Total |
|---|---|---|---|---|---|---|
| BM-V | 0.48% | 0.58% | 0.27% | 0.14% | 1.84% | 3.30% |



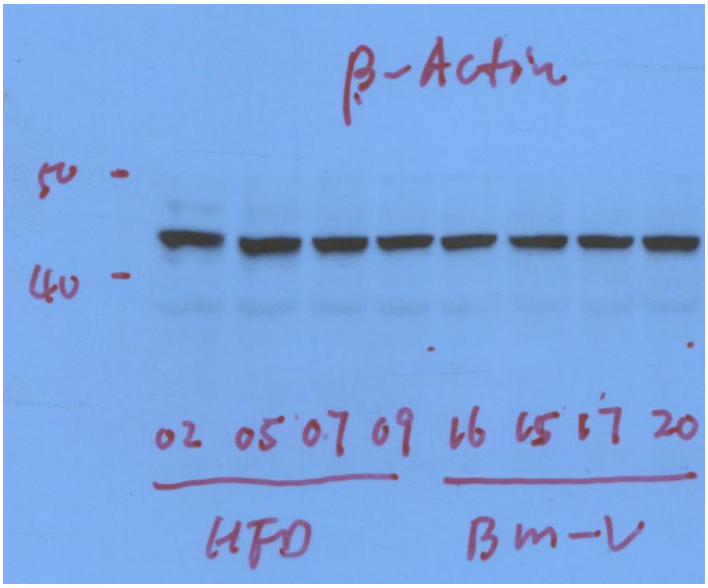

AMPK p




AMPKα1

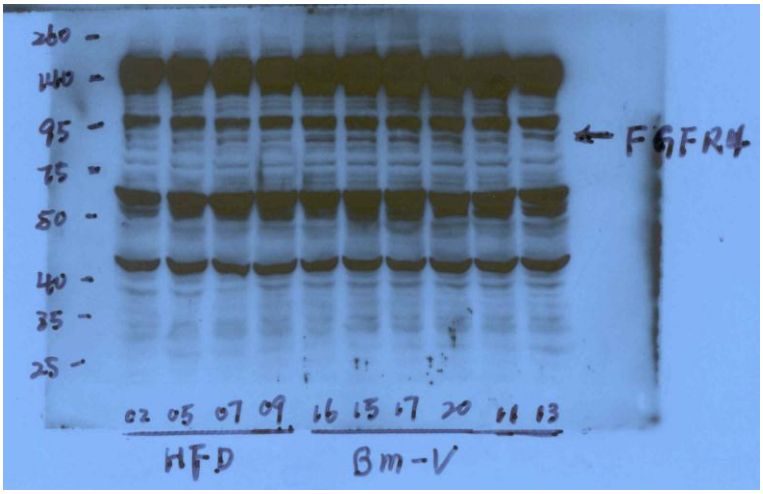



AMPKα2

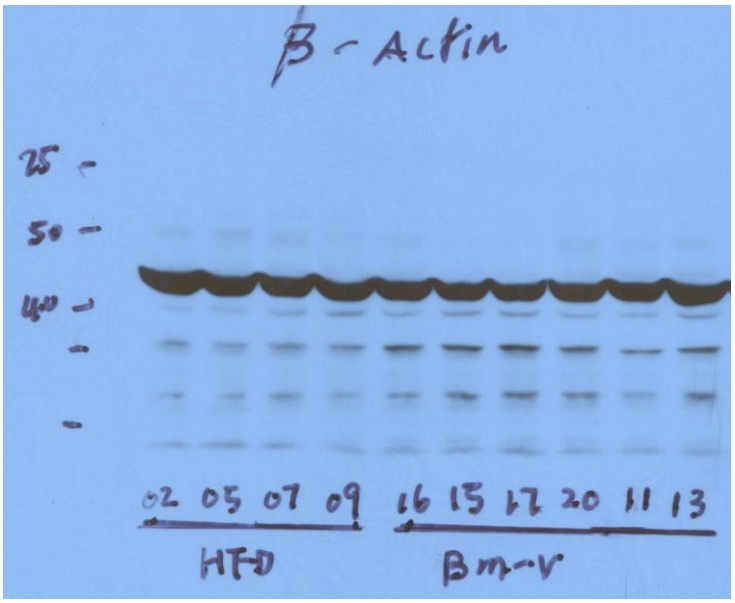



B-Actin1

Figure 5. Effects of BM extracts on AMPK-Sirt1 signaling pathways in mice liver. Fifty μg of liver lysates was subjected to SDS-PAGEs, AMPK p, AMPK α1, AMPKα2 and Sirt1 were detected with corresponding specific antibodies. The results were normalized using β-actin as protein loading control. The data were represented as mean ± SEM (n=10/group), * P< 0.05, BM-V vs. HFD group.




FGFR4




B-Actin2

Figure 3. The effects of BM extracts on fasting plasma and liver FGF21 levels as well as FGF21 signaling in mice. FGF21 was measured using a mouse FGF21 ELISA kit from R & D Systems Inc (Minneapolis, MN). (A) Fasting plasma FGF21 concentrations. (B) Liver FGF21 content. Mean ± SEM (n=10/group). (C) FGF21 signaling proteins were measured by Western blotting assay. Results were normalized by β-actin content. BM-V significantly increased FGFR1, FGFR3, FGFR4 and PGC-1α, slightly reduced PPARα, but did not affect b-Klotho protein abundance in comparison with HFD animals. Mean ± SEM (n=10/group). * P < 0.05, ** P<0.01, and *** P <0.001, BM-V group vs. HFD group. # P<0.05, glucose concentrations at week 6 or week 12 vs. week 0 in HFD animals.