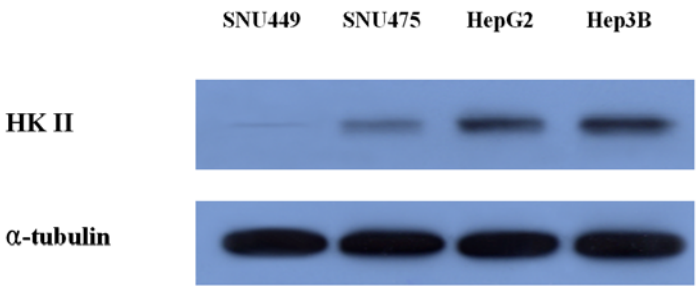
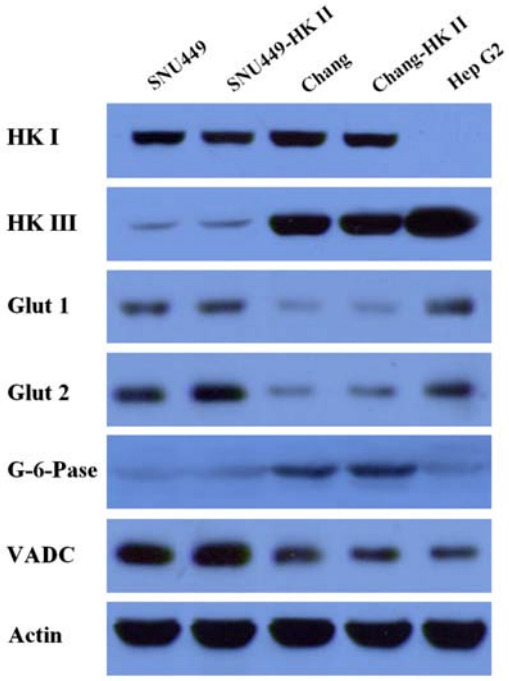
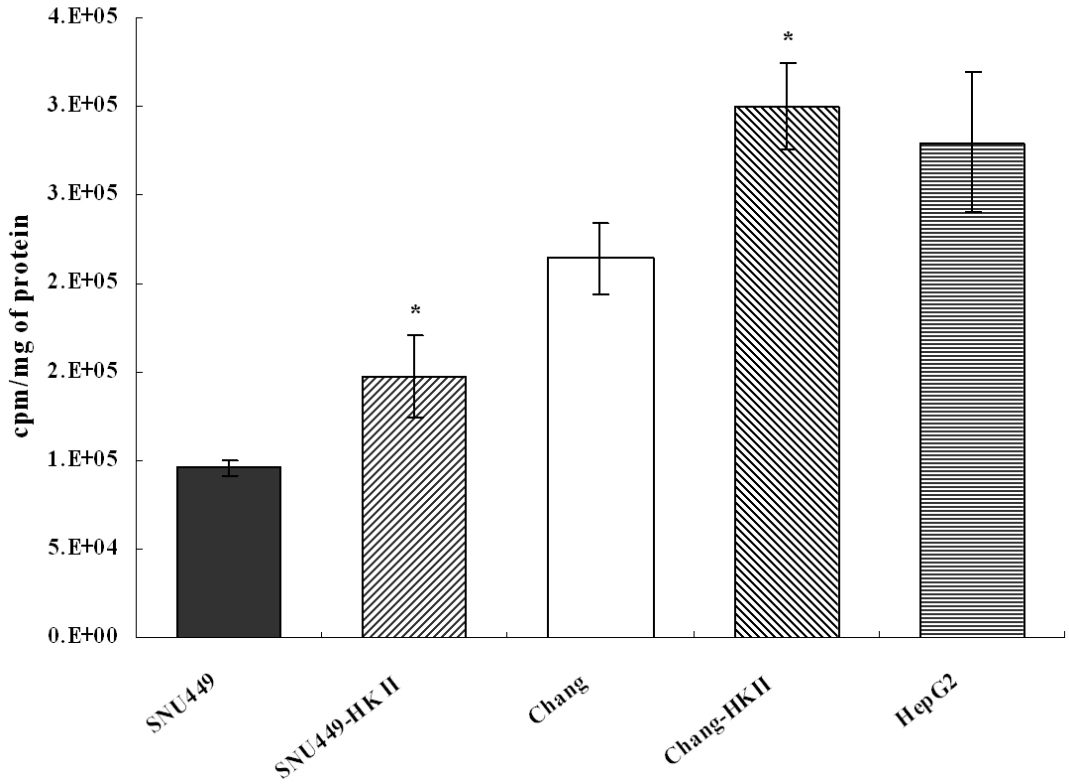


Supplemental Figure 1. Expression profile of HKII in various HCC cell lines. Human HCC cell line, SNU449 shows low expression of HKII on Western blot analysis. α-tubulin was used as loading controls for Western blot analysis.

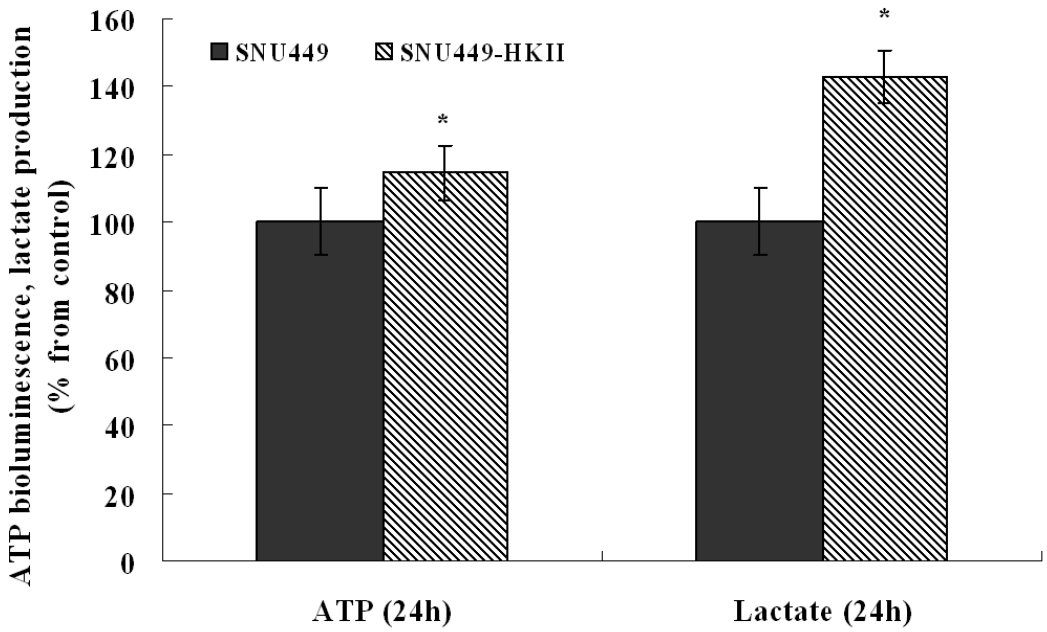


Supplemental Figure 2. Expression profile of isoforms of glycolytic enzymes and glucose transporters in established stable cells. There is no alteration of G6Pase, HKI and HKIII isozymes and glucose transporters in both SNU-449 and control Chang cells before and after HKII transfection. HepG2 was used as a control for each gene.

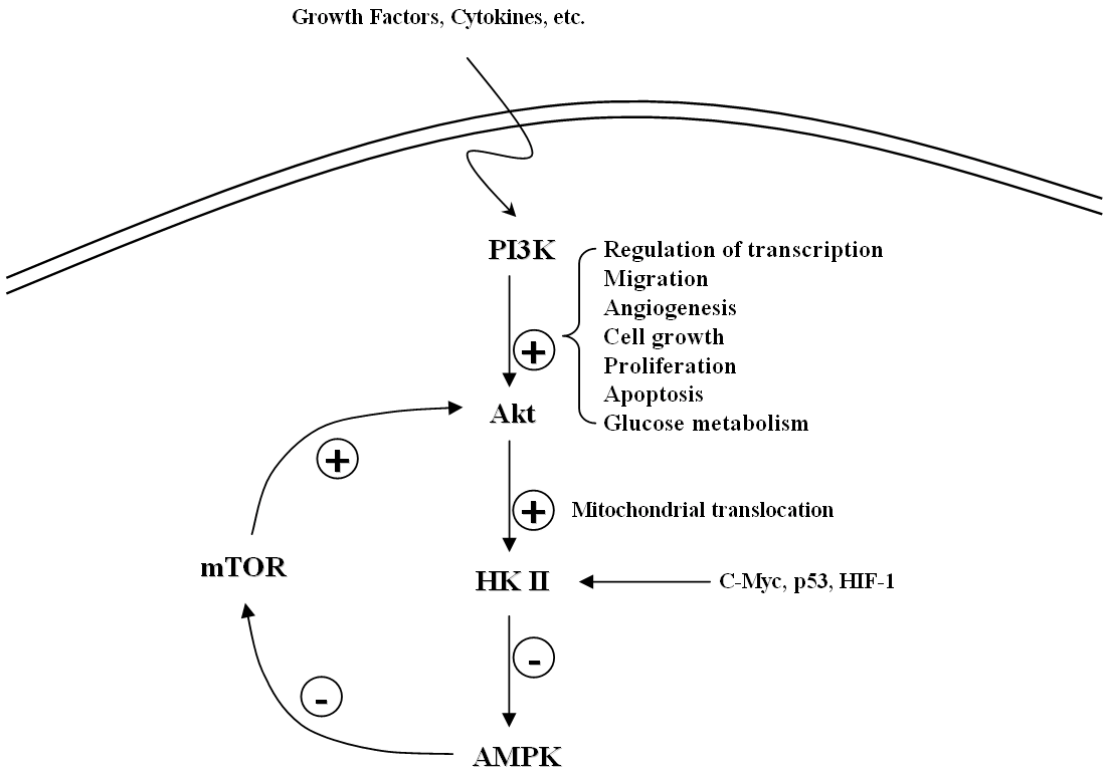


Supplemental Figure 3. Measurement of ^{18}F-FDG uptake levels in established stable cells. Results are expressed as mean±SD of cpm/mg protein of triplicate samples obtained from single experiment representative of 3 separate experiments.

*p< 0.05, **p< 0.01



Supplemental Figure 4. Effects of over-expressed HKII on ATP and lactate production. ATP and lactate production was significantly increased after HKII transfection. The results are presented as the means±SD from 6 independent experiments (*$p<0.01$).



Supplemental Figure 5. Schematic overviews of over-expressed HKII on PI3K/Akt and AMPK pathway. Activation of Akt by growth factors and PI3K leads to translocation of HKIIs to the mitochondrial outer membrane. Mitochondrial-bound HK II is crucial for energy supply as a form of ATP. Excessive ATP production inactivates AMPK but Akt is activated through mTOR signaling. Akt and its downstream signaling pathways play important roles in tumor proliferation and progression.

+ ; Activation

- ; Inactivation