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A New HCC Model Induced by Co-overexpression FGF19 and Met in Mice

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Hepatocellular carcinoma (HCC) is one of the leading causes of cancer-related deaths worldwide. Sorafenib is the only targeted therapy approved for HCC. Thus, developing useful animal models may shed light on the biology of HCC tumor and provide a platform to evaluate the potential therapeutic agents.

In the current study, we found FGF19 and cMet work synergistically to induce tumor after 13 weeks post co-delivery FGF19 and cMet to the mice hepatocytes using the sleeping beauty transposon/transposase system via hydrodynamic tail vein injection. H&E staining showed the tumor is HCC, meanwhile, the immunochemistry staining results showed that Glycogen synthase (GS) genitive and cell cycle D1(CCND1) was upregulated in the tumor area, ki67 also increased in the tumor nodule. pERK and pmTOR was also activated in the tumor areas by IHC staining.

Such data showed mTOR and ERK pathway might be required for HCC induced by FGF19/Met. The molecular mechanisms and target therapy could be carried based on this model in the near future, including single gene target and combination target therapy.

Keywords: HCC Model; FGF19, Met, mTOR, ERK.