

CCCACTT-3'; *WNT2*: 5'-AGAGTGCCAACACCAGTTCC-3' and 5'-TACAGGAGCCACTCACACCA-3'; *WNT3A*: 5'-GCGGGCATCCAG-GAGTGCAG-3' and 5'-CTCTGCACAGGAGCGTGTAC-3'; *WNT4*: 5'-GAGAAGTGTGGCTGTGACCGG-3' and 5'-ATGTTGTCCGAG-CATCCTGACC-3'; *WNT5A*: 5'-TGACAACTGGCAGAAAAACAA-3' and 5'-TCCCTGAATGGAACAAACAA-3'; *WNT5B*: 5'-GCAGTT-GACCTGACCTGCTA-3' and 5'-TTCTGTCACCTGCTACAGCC-3'; *WNT7A*: 5'-CCAAATGGGCCCTGGACGAGTG-3' and 5'-CCGGTGGTAC-TGGCCTTGCT-3'; *WNT7B*: 5'-TCTCTGCTTGCGTCTCTAC-3' and 5'-GCCAGGCCAGGAATCTTGTG-3'; *WNT9B*: 5'-GGGTGTGTGTTG-GTACAATC-3' and 5'-TCCAACAGGTACGAACAGCA-3'; *WNT10A*: 5'-GGCGCTCCTGTTCTCTACTGCT-3' and 5'-GATAGCAGAGGC-GGCCACGTCAAGG-3'; *WNT11*: 5'-CTGAATCAGACGCAACACTG-TAAC-3' and 5'-CTCTCTCCAGGTCAAGCAGGTAG-3'.

Assessment of glucagon-mediated Wnt gene expression was performed in primary hepatocytes that were serum-starved overnight. Cells were stimulated with glucagon (100 nM) for 2 hours before RNA isolation and subsequent Wnt isoform analysis.

For the analysis of both G6Pase and PEPCK, the following primers were used: *G6PC*: 5'-ATGAACATTCTCCATGACTTTGGG-3' and 5'-GACAGGGAACTGCTTATTATAGG-3'; *PCK1*: 5'-CATAACGGTCTG-GACTTCTCTGC-3' and 5'-GAATGGGATGACATACATGGTGCG-3'.

For Wnt target gene analysis, the following primers were used: *AXIN2*: 5'-AACCTATGCCGTTTCTCT-3' and 5'-CTGGTCACCCAACAAGGAGT-3'; *CD1*: 5'-TCTCCTGCTACCGCACAAC-3' and 5'-TTCCTC-CACTTCCCCCTC-3'; *MYC*: 5'-CTAGTCCGACCAGCGTCAC-3' and 5'-GTACCCAATCCTGAACCAC-3'; *DKK1*: 5'-GAGGGGAAATT-GAGGAAAGC-3' and 5'-GCAGGTGTGGAGCCTAGAAG-3'; *FRZB*: 5'-TCCAACAAGTGTACCGAGCG-3' and 5'-CTCCATACATATTG-TAAGCG-3'; *WISP2*: 5'-ATACAGGTGCCAGGAAGGTG-3' and 5'-CAAGGGCAGAAAGTTGGTGT-3'.

Analysis of gene expression in lipid metabolism was achieved with the following primers: *CPT1A*: 5'-TGCACTACGGAGTCCTGCAA-3' and 5'-GGACAAACCTCCATGGCTCAG-3'; *ACOX1*: 5'-GCCTGCTGT-GTGGGTATGTCATT-3' and 5'-GTCATGGCCGGTGCAT-3'; *ACCA1*: 5'-ATGCTTCCATGCTGAGATTGT-3' and 5'-TCCATCCTGAAGG-CAGGCTT-3'.

All gene expression results were normalized to an internal control with the following primer set: *ACTA1*: 5'-TGGCATTGTTACCAACTGGGACG-3' and 5'-GCTTCTCTTGATGTCACGCACG-3'.

Statistical analysis

For experiments with multiple comparisons, data were first analyzed by analysis of variance (ANOVA) with Bonferroni correction. For single comparisons, a Student's *t* test was performed on data before normalization.

SUPPLEMENTARY MATERIALS

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- Fig. S1. Deletion of hepatic β -catenin does not alter serum insulin concentration.
- Fig. S2. Quantification of FoxO1 subcellular localization.
- Fig. S3. Insulin tolerance tests (ITTs) in flox/flox β -catenin mice after injection with adenoviruses encoding GFP or Cre recombinase.
- Fig. S4. Subcellular localization of FoxO1 in primary hepatocytes after insulin stimulation.
- Fig. S5. Deletion of β -catenin decreases the mRNA abundance of FoxO1 transcriptional targets.
- Fig. S6. Knockdown of FoxO1 decreases the mRNA abundance of β -catenin transcriptional targets.
- Fig. S7. Wnt stimulation increases binding of β -catenin to the *G6PC* promoter.
- Fig. S8. Wnt3a stimulates hepatocyte oxygen consumption.
- Fig. S9. Effects of glucagon stimulation on the abundance of mRNAs encoding Wnt ligands.
- Fig. S10. The effect of β -catenin on the abundance of mRNAs encoding lipid enzymes and on triglyceride concentrations.

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