CORRECTION OPEN

Check for updates

Correction: C-terminal truncated HBx initiates hepatocarcinogenesis by downregulating TXNIP and reprogramming glucose metabolism

Yu Zhang, Qian Yan, Lanqi Gong, Hang Xu, Beilei Liu, Xiaona Fang, Dandan Yu, Lei Li, Ting Wei, Ying Wang, Ching Ngar Wong, Zhaojie Lyu, Ying Tang, Pak Chung Sham and Xin-Yuan Guan

Oncogene (2021) 40:5451–5453; https://doi.org/10.1038/s41388-021-01942-y

Correction to: *Oncogene* https://doi.org/10.1038/s41388-020-01593-5, published online 15 December 2020

Unfortunately, an error occurred in Fig. 5 and in legend to Fig. 5. The corrected Fig. 5 with the corrected legend is given below. The original article has been corrected.

Published online: 26 July 2021

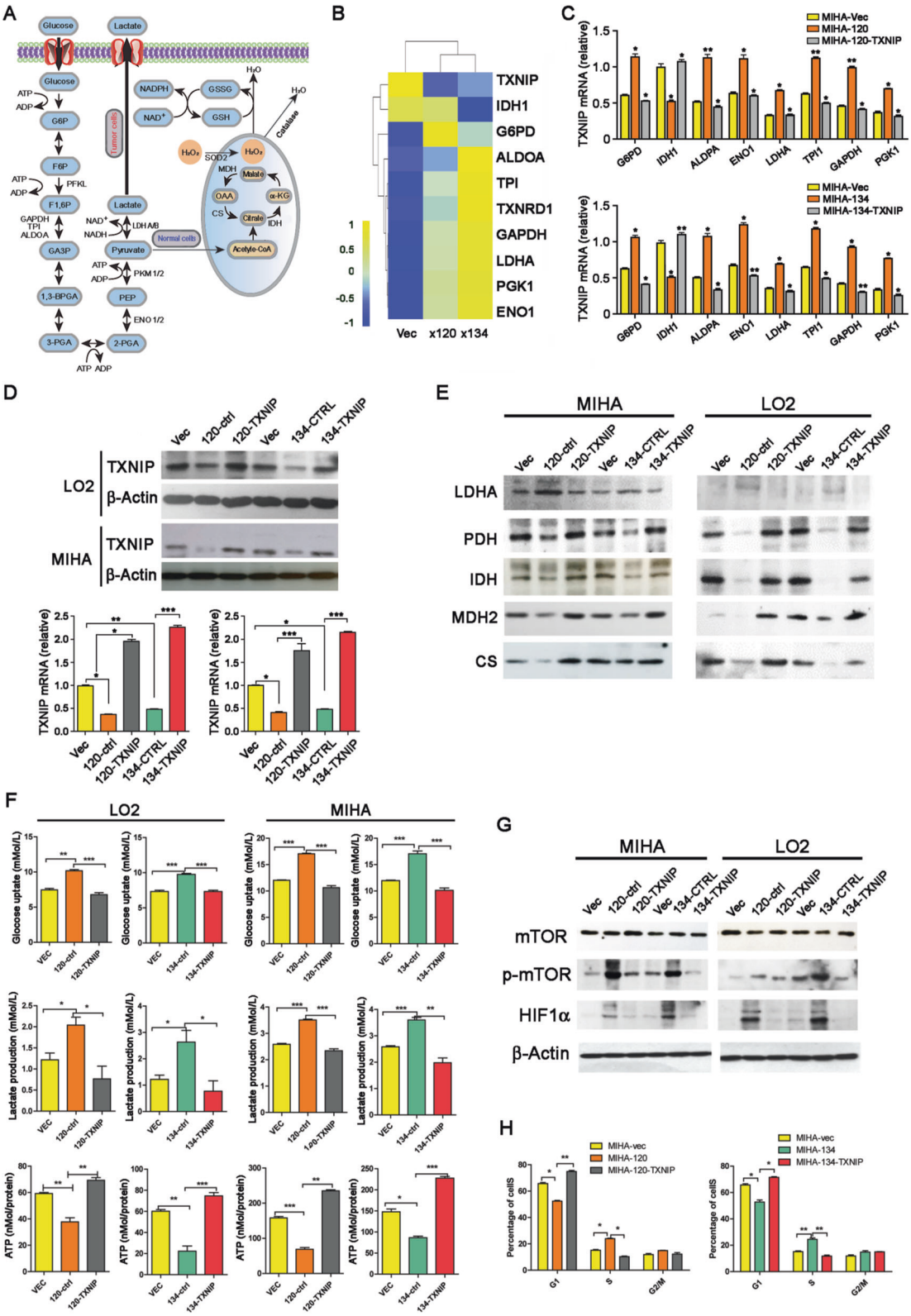
A
Glucose
Lactate
Glucose
ATP
ADP
G6P
F6P
ATP
ADP
PFKL
F1,6P
GAPDH
TPI
ALDOA
GA3P
1,3-BPGA
PEP
ENO 1/2
3-PGA
2-PGA
ATP ADP
Tumor cells
Lactate
NAD+
NADH
LDHA/B
Normal cells
Pyruvate
ATP
ADP
PKM 1/2
NADPH
GSSG
NAD+
GSH
H2O
Catalase
H2O2
SOD2
H2O2
MDH
Malate
OAA
α-KG
CS
Citrate
IDH
Acetyle-CoA
B
TXNIP
IDH1
G6PD
ALDOA
TPI
TXNRD1
GAPDH
LDHA
PGK1
ENO1
1
0.5
0
-0.5
-1
Vec
x120
x134
C
MIHA-Vec
MIHA-120
MIHA-120-TXNIP
TXNIP mRNA (relative)
G6PD
IDH1
ALDPA
ENO1
LDHA
TPI1
GAPDH
PGK1
MIHA-Vec
MIHA-134
MIHA-134-TXNIP
TXNIP mRNA (relative)
G6PD
IDH1
ALDPA
ENO1
LDHA
TPI1
GAPDH
PGK1
D
Vec
120-ctrl
120-TXNIP
Vec
134-CTRL
134-TXNIP
LO2
TXNIP
β-Actin
MIHA
TXNIP
β-Actin
TXNIP mRNA (relative)
Vec
120-ctrl
120-TXNIP
134-CTRL
134-TXNIP
TXNIP mRNA (relative)
Vec
120-ctrl
120-TXNIP
134-CTRL
134-TXNIP
E
MIHA
LO2
Vec
120-ctrl
120-TXNIP
Vec
134-CTRL
134-TXNIP
LDHA
PDH
IDH
MDH2
CS
F
LO2
MIHA
Glocose uptake (mMol/L)
Lactate production (mMol/L)
ATP (nMol/protein)
VEC
120-ctrl
120-TXNIP
134-ctrl
134-TXNIP
G
MIHA
LO2
Vec
120-ctrl
120-TXNIP
Vec
134-CTRL
134-TXNIP
mTOR
p-mTOR
HIF1α
β-Actin
H
MIHA-vec
MIHA-120
MIHA-120-TXNIP
Percentage of cellS
G1
S
G2/M
MIHA-vec
MIHA-134
MIHA-134-TXNIP
Percentage of cellS
G1
S
G2/M

Fig. 5 TXNIP induced glucose metabolism reprogramming from glycolysis to mitochondrial respiration. A Schematic representation of the biological process of glucose metabolism in normal cells and cancer cells. **B** Heatmap showing the relative expression level of several genes involved in glucose metabolism in Ct-HBx and vectors containing samples as indicated by RNA sequencing, each matrix representing the relative expression level of an individual gene; high and low expressions are indicated by yellow and blue color. **C** The expression level of the gene panel indicated above was validated by qRT-PCR in MIHA cells transduced with truncated HBx mutants compared with the vector group; also, the expression was further compared after re-introduction of TXNIP into Ct-HBx-expressing cells. **D** Re-introduction of TXNIP into Ct-HBx-(HBx-120, HBx-134) expressing cells was confirmed at the protein and genomic level by western blotting and qRT-PCR. **E** The expression level of several key enzymes and molecules that participated in glycolysis and Krebs cycle are determined by western blotting. The expression of internal reference β-actin can be referred to in (**D**). **F** Level of glucose uptake, lactate secretion, and relative ATP production activity was compared among vector, Ct-HBx as well as TXNIP overexpression samples. **G** The activation of the mTOR-HIF1α axis was detected by western blotting; β-actin was used as an internal reference. **H** Analysis of cell distribution in each stage of the cell cycle in each transfected MIHA cell.