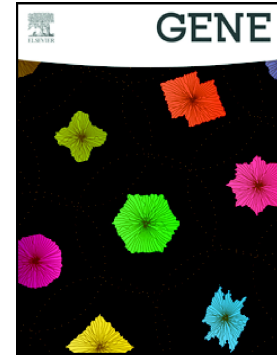


## Accepted Manuscript

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PII: S0378-1119(18)31037-0  
DOI: doi:[10.1016/j.gene.2018.10.012](https://doi.org/10.1016/j.gene.2018.10.012)  
Reference: GENE 43268  
To appear in: *Gene*  
Received date: 23 August 2018  
Revised date: 25 September 2018  
Accepted date: 4 October 2018

Please cite this article as: Pengjiao Xi, Jie Xue, Zhaoxia Wu, Haomin Wang, Jie Han, Huimin Liang, Derun Tian , Liver kinase B1 induces browning phenotype in 3T3-L1 adipocytes. *Gene* (2018), doi:[10.1016/j.gene.2018.10.012](https://doi.org/10.1016/j.gene.2018.10.012)

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**Liver kinase B1 induces browning phenotype in 3T3-L1 adipocytes**

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**ABSTRACT**

Induction of brown adipocytes in white adipose tissue is a promising therapy for combating human obesity and its associated disorders. Liver kinase B1 (LKB1) is a tumor inhibitor and metabolic modulator. Recent data suggest that LKB1 is necessary for adipogenesis, but its role in the browning of white adipocytes remains unknown. The objective of this study was to reveal the effect of LKB1 on browning. In our study, we showed that overexpression of LKB1 in 3T3-L1 adipocytes up-regulated the expression of brown adipocyte markers, including UCP1, PGC-1 $\alpha$ , Cidea, and PRDM16, and beige-cell-specific genes, such as CD137 and Tmem26. It was possible that the expression of UCP1 and other beige markers was increased by activation of PPAR $\gamma$ , resulting in the browning of 3T3-L1 preadipocytes. The browning effect was abolished by a PPAR $\gamma$  inhibitor (GW9662). Moreover, these effects were dramatically abrogated by silencing of LKB1. Additionally, LKB1 decreased the expression levels of adipogenesis proteins (C/EBP $\alpha$  and SREBP1) and up-regulated lipid catabolism protein, perilipin (PLIN). In summary, the study suggested that LKB1 induces the browning of white adipocytes, in addition to promoting lipid metabolism. Therefore, LKB1 may be a helpful therapeutic candidate for treating obesity.

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