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Control of hepatic gluconeogenesis by the promyelocytic leukemia zinc finger protein

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T he promyelocytic leukemia zinc finger (PLZF) protein is involved in major biological processes by exerting its transcriptional regulative function. Recently, PLZF has been implicated in the regulation of energy metabolism, although its precise role and the detailed mechanism remain unknown. Here, we show the hepatic expression of PLZF was induced in fasted or diabetic mice. Gain- and loss-of-function studies indicated that PLZF promoted the expression of gluconeogenic genes and hepatic glucose output, which led to hyperglycemia. PLZF also regulates the activity of Akt signaling pathway. Mechanistically, PLZF transcription was activated by the synergistic interaction of PGC-1a and the glucocorticoid receptor. In addition, PGC-1a was recruited to the GR binding site on the proximal PLZF promoter and turned the chromatin structure into an active form. Finally, PLZF is required for PGC-1a-induced gluconeogenesis. Taken together, our results suggest that PLZF is a critical transcriptional factor in the regulation of hepatic gluconeogenesis.

Biography

Chang Liu obtained his PhD degree from Nanjing University in 2005. From 2006 to 2008, he accepted Postdoc training in the University of Michigan. He then went back to China and became a full Professor in the Department of Cellular Biology, Nanjing Normal University. His group focuses on the integration of circadian clock and energy metabolism and the metabolic roles of transcriptional factors/cofactors. Serving as the corresponding author, he has published 24 scientific papers in reputed journals, such as *Hepatology, Diabetes*, and *Journal of Pathology*. He also serves in several editorial boards and study sections.

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