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Data Article

Data on the decreased expression of FOXO1 by miR-1271 in HepG2 hepatocytes

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ABSTRACT

Obesity and metabolic diseases are closely associated with insulin resistance. Obesity-induced miRNAs are also considered to be potential contributors to the development of insulin resistance and type 2 diabetes. Previously, the expression of miR-1271 was reported to be upregulated in the liver of diet-induced obese mice (Yang et al., 2016) [1]. In this data article, multiple *in silico* analysis predicted FOXO1 gene to be a direct target of miR-1271. Dual luciferase reporter gene analysis showed that miR-1271 suppressed FOXO1 expression by direct binding to 3′UTR. The overexpression of miR-1271 reduced the protein expression of FOXO1, thereby reducing the transcription of PEPCK, a downstream target of FOXO1. The data is related to a research article entitled "MiR-1271 upregulated by saturated fatty acid palmitate provokes impaired insulin signaling by repressing INSR and IRS-1 expression in HepG2 cells" (Yang et al., 2016) [1].

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Specifications Table

Subject area	*Cell Biology*
More specific subject area	*Obesity, Metabolism, MicroRNA*
Type of data	*Figure and text*
How data was acquired	*Analysis of Dual luciferase reporter gene assay and immunoblotting*
Data format	*Analyzed*
Experimental factors	*Transfection of miR-1271, Analysis of FOXO1 and PEPCK expression*
Experimental features	*HepG2 hepatocytes were transfected with scRNA or miR-1271 mimic.* Expression of *FOXO1 was analyzed with Dual-luciferase reporter gene assay and immunoblotting. mRNA of PEPCK was determined by qRT-PCR.*
Data source location	*Dongguk University School of Medicine, Gyeongju-si, Gyeongsangbuk-do 38067, Korea*
Data accessibility	*The data are supplied with this article*

Value of the data

- The data are useful in understanding the regulatory relationship between miR-1271 and FOXO1 expression.
- The data can be compared with other obesity-related miRNAs involved in the pathogenesis of metabolic diseases.
- The modulation of miR-1271 expression can be applied further in functional studies of the cellular and systemic responses related with SFA-induced insulin resistance.

1. Data

The expression of certain miRNAs targeting the molecules transducing insulin signaling is regulated aberrantly in saturated fatty acids (SFA)-induced obesity, and linked intimately to the pathogenesis of insulin resistance [2,3]. Previously, a dysregulation of miR-1271 expression was reported to be linked causally to the development of hepatic insulin resistance [1]. This data article assessed the targets of miR-1271 on the insulin signaling pathway using *in silico* analysis, such as TargetScan, Pictar, and miRWalk. FOXO1 was found to be one such predicted target of miR-1271 that belongs to this pathway (Fig. 1A). This data article also presents accompanying data collected from a dual-luciferase reporter gene assay and immunoblotting to determine if FOXO1 would be a validated target of miR-1271 in hepatocytes. First, the direct binding of miR-1271 to *FOXO1* 3′UTR was determined using a Dual luciferase-based reporter assay. Luciferase reporter constructs containing either a tentative miR-1271 target sequence in *FOXO1* 3′UTR (wild-type; FOXO1 3U*wt*), or three nucleotides mutant of the tentative target sequence (FOXO1 3U*mut*) were generated in the pmirGLO vector (Fig. 1B), as described in the Section 2. These reporter constructs, which included a Firefly luciferase cassette to allow normalization of the internal Renilla luciferase activity, were transfected transiently together with a scRNA control or miR-1271 mimic. As shown in Fig. 1C, co-transfection with the miR-1271 mimic and reporter construct (FOXO1 3U*wt*) inhibited the luciferase activity compared to the scRNA control. Mutations in the tentative miR-1271 binding site in the *FOXO1* 3′UTR (FOXO1 3U*mut*) abrogated the repressive effect of miR-1271 (Fig. 1C). This suggests that miR-1271 targets *FOXO1* 3′ UTR directly via its binding site. Moreover, the transfection of miR-1271 mimics decreased significantly the protein expression of FOXO1 in HepG2 cells, whereas the expression of the β-actin control was unaffected (Fig. 2A). Interestingly, the transfection of miR-1271 decreased significantly the mRNA level of PEPCK, a downstream target gene of FOXO1 (Fig. 2B). Further analysis of the data