Correction

Correction for: Endothelin-1-mediated miR-let-7g-5p triggers interlukin-6 and TNF-α to cause myopathy and chronic adipose inflammation in elderly patients with diabetes mellitus

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This article has been corrected: The authors found two mistakes in the legend to **Figure 8**:

in the title, “ET-1 suppresses production of NFkB, TNF-α and IL-6 by increasing miR-let-7g-5p expression,” the authors accidentally transposed the words "suppresses" and "increasing." The correct text should be “ET-1 **increases** production of NFkB, TNF-α and IL-6 by **suppressing** miR-let-7g-5p expression”;

in the text of the legend for panel **8B**, the word “melatonin” should be replaced with “ET-1."

The corrected legend to **Figure 8** is presented below.

Figure 8. ET-1 increases production of NFkB, TNF-α and IL-6 by suppressing miR-let-7g-5p expression. (**A**) Open-source software (TargetScan, miRDB, and miRWalk) sought to identify miRNAs that could possibly interfere with NFkB, IL-6 and TNF- α transcription. (**B**) Cells were incubated with ET-1 (0-50 nM) for 24 h and miR-let-7g-5p expression was examined by qPCR. (**C**) Cells were pretreated with BQ123+BQ788, Ly294002, Akt inhibitor for 30 min, then stimulated with ET-1 for 24 h. miR-let-7g-5p expression was examined by qPCR. (**D**, **E**) Cells were transfected with the miR-let-7g-5p mimic and then treated with ET-1 (50 nM). TNF-α and IL-6 expression was evaluated by qPCR. The wild-type and mutant Ikbkb 3′-UTRs contained the miR-let-7g-5p binding site. (**F**) Cells were transfected with the miR-let-7g-5p mimic and then treated with ET-1 (50 nM). (**G**) Cells were transfected with 3′-UTR plasmids as indicated then stimulated with ET-1 dose concentration. Then, cells were transfected with indicated luciferase plasmids for 24 h then stimulated with ET-1 for 24 h. Relative luciferase activity was measured. Results are expressed as the mean ± SEM. $^{*}P < 0.05$ compared with controls; $^{\#}P < 0.05$ compared with the melatonin-treated group.