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Establishment of skeletal muscle-specific PGC-1 α overexpression model via in vivo local transfection

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 $\label{eq:objective} \begin{array}{c} \textbf{Objective} \ \text{To establish a skeletal muscle-specific PGC-1} \alpha \ \text{overexpression mouse model via in vivo local transfection.} \end{array}$

Methods For the PGC-1 α in vivo transfection study, the Male FVB/N mice were randomly divided into 2 groups: subject to green fluorescent protein (GFP) transfection (Con-GFP), and subject to PGC-1 α in vivo transfection (Con-PGC-1 α). Plasmid DNA solution (2.5 mg/ml GFP or 2.7 mg/ml Flag-PGC-1 α) were injected into the proximal (6 ml) and distal (6 ml) ends of the muscle belly. Following the injections, electric pulses were applied through 2 stainless steel pin electrodes laid on top of the proximal and distal myotendinous junctions. Then skeletal muscle and myocardium were isolated, and PGC-1 α , mtTFA, NF- κ B, MnSOD, FNDC5 protein expression were measured with Western blot.

Results n skeletal muscle, compared with con-GFP group, the expression of PGC-1 α (+125%, *p*<0.01), mtTFA (+210%, *p*<0.01), and FNDC5 (+47%, *p*<0.05) were significantly increased in con-PGC-1 α group. However, NF- κ B and MnSOD protein level had no change in con-PGC-1 α group. In myocardium, compared with con-GFP group, the expression of mtTFA (+130%, *p*<0.01) and FNDC5 (+55%, *p*<0.05) were significantly increased in con-PGC-1 α group.

Conclusions Skeletal muscle-specific PGC-1 α overexpression model via in vivo local transfection was established, which was supported by elevated expression of PGC-1 α and its downstream FNDC5 and mtTFA. Furthermore, skeletal muscle-specific PGC-1 α overexpression induced increase in myocardial mitochondrial biogenesis, while relative mechanism remains to be determined.