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Data Article

Lean and Obese Zucker Rat Extensor Digitorum Longus Muscle high-frequency electrical stimulation (HFES) Data: Regulation of MAPKs Associated Proteins

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ABSTRACT

Anaerobic exercise has been advocated as a prescribed treatment for the management of diabetes: however, alterations in exercise-induced signaling remain largely unexplored in the diabetic muscle. Here, we compare the basal and the in situ contraction-induced phosphorylation of the mitogen-activated protein kinases (MAPKs) ERK 1/2, p38, and JNK in the lean and obese (fa/fa) Zucker rat extensor digitorum longus (EDL) muscle following a single bout of contractile stimuli. This article represents data associated with prior publications from our (Katta et al., 2009a, 2009b, 2008) [1–3]

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High-frequency electrical stimulation (HFES)
Zucker rat
Extensor Digitorum Longus

and concurrent Data in Brief articles (Ginjupalli et al., 2017a, 2017b; Rice et al., 2017a, 2017b) [4–7].

Specifications Table

Subject area	*Biology*
More specific subject area	*Diabetic skeletal muscle response to exercise*
Type of data	*Graph, figure*
How data was acquired	*Immunoblotting*
Data format	*Analyzed*
Experimental factors	*A high-frequency electrical stimulation (HFES) was used to produce 10 sets of 6 contractions over a 22-min period. Tissues were collected and protein was then isolated from tissue for western blot analysis.*
Experimental features	*EDL obtained from Lean and Obese male Zucker rats were used in this experiment*
Data source location	*Huntington, WV USA*
Data accessibility	*Data is with this article and is related to articles published and in review* [1–7].

Value of the data

- The data presented in this Brief is vital to understanding the effect of diabetes on skeletal muscle mechanotransduction.
- This data gives insight into the how diabetes alters tissue response to stimuli.
- This data provides a more thorough understanding of the MAPKs involvement in exercise mediated signaling in both diabetic and non-diabetic muscle tissue.

1. Data

1.1. ERK 1/2

To determine the effect of HFES on EDL from OSXZ and LNZ animals we evaluated the phosphorylation of ERK 1/2 at threonine 202 and tyrosine 204 (p44/p42 thr 202/tyr 204). EDL basal phosphorylation of p44 thr 202/tyr 204 demonstrated no significant difference in the OSXZ when compared to LNZ (Fig. 1A). HFES resulted in an increase in phosphorylation of p44 thr 202/tyr 204 in the LNZ EDL (126.9 $\pm$ 3.8%, 126.3 $\pm$ 4.1%, and 407.2 $\pm$ 31.6%, at 0, 1,and 3 h, $p < 0.05$) when compared to LNZ contralateral control (Fig. 1A). HFES resulted in an increase in phosphorylation of p44 thr 202/tyr 204 in the OSXZ EDL (242.7 $\pm$ 49.5% at 0 h, $p < 0.05$) when compared to OSXZ contralateral control (Fig. 1A). EDL basal phosphorylation of p42 thr 202/tyr 204 was higher (88.8 $\pm$ 2.7%, $p < 0.05$) in the OSXZ when compared to LNZ (Fig. 1B). HFES resulted in an increase in phosphorylation of p42 thr 202/tyr 204 in the LNZ EDL (290.6 $\pm$ 20.5%, 271.0 $\pm$ 4.1%, and 460.3 $\pm$ 16.7%, at 0, 1,and 3 h, $p < 0.05$) when compared to LNZ contralateral control (Fig. 1B). HFES resulted in an increase in phosphorylation of p42 thr 202/tyr 204 in the OSXZ EDL (371.9 $\pm$ 17.8% and 100.0 $\pm$ 28.4%, at 0 and 3 h, $p < 0.05$) when compared to OSXZ contralateral control (Fig. 1B).