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Data Article

Lean and Obese Zucker Rat Extensor Digitorum Longus Muscle high-frequency electrical stimulation (HFES) Data: Regulation of p70S6kinase 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 AKT, GSK3beta, mTor, p70s6K, Pten, and Shp2 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 lab (Katta et al., 2009a, 2009b; Tullgren

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High-frequency electrical stimulation (HFES)
Zucker rat
Extensor Digitorum Longus
p70s6k

et al., 1991) [1–3] 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 mTor pathway involvement in exercise mediated signaling in both diabetic and non-diabetic muscle tissue.

1. Data

1.1. AKT

To determine the effect of HFES on EDL from diabetic male obese syndrome-X Zucker (OSXZ) diabetic and nondiabetic male normal lean Zucker (LNZ) animals we evaluated the phosphorylation of AKT at Threonine 308 and Serine 473. EDL basal phosphorylation of AKT Thr 308 was lower (15.0 ± 5.9%, $p < 0.05$) in the OSXZ when compared to LNZ (Fig. 1A). HFES resulted in a decrease in phosphorylation of AKT Thr 308 in the LNZ EDL (10.9 ± 1.6% at 3 h, $p < 0.05$) when compared to LNZ contralateral control (Fig. 1A). HFES resulted in an increase in phosphorylation of AKT Thr 308 in the OSXZ EDL (11.9 ± 1.5%, at 3 h, $p < 0.05$) when compared to OSXZ contralateral control (Fig. 1A). EDL basal phosphorylation of AKT Ser 473 was lower (64.6 ± 2.6%, $p < 0.05$) in the OSXZ when compared to LNZ (Fig. 1B). HFES resulted in an increase in phosphorylation of AKT Ser 473 in the LNZ EDL (17.8 ± 1.6%, and 51.5 ± 4.9%, at 0 and 3 h, $p < 0.05$) and decrease (23.2 ± 1.0%, 1 h, $p < 0.05$) when compared to LNZ contralateral control (Fig. 1B). HFES resulted in an increase in phosphorylation of AKT Ser 473 in the OSXZ EDL (63.0 ± 0.3%, 33.7 ± 1.6%, and 47.9 ± 8.7%, at 0, 1 and 3 h, $p < 0.05$) when compared to OSXZ contralateral control (Fig. 1B).

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