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Data Article

High-frequency electrical stimulation (HFES) Data Lean and Obese Zucker Rat Soleus Muscle: Regulation of p70S6kinase Associated Proteins

Kevin M. Rice [a,b,c,d,*], Anjaiah Katta [a], Nandini D.P.K. Manne [e], Ravikumar Arvapalli [a], Gautam K. Ginjupalli [a], Miaozong Wu [f], Shinichi Asano [g], Eric R. Blough [a,c,h,i]

[a] Center for Diagnostic Nanosystems, Marshall University, Huntington, WV, USA
[b] Department of Internal Medicine, Joan C. Edwards School of Medicine, Marshall University, Huntington, WV, USA
[c] Biotechnology Graduate Program West Virginia State University, Institute, WV, USA
[d] Department of Health and Human Service, School of Kinesiology, Marshall University, Huntington, WV, USA
[e] Department of Public Heath, Marshall University, Huntington, WV, USA
[f] College of Health, Science, and Technology, University of Central Missouri, Warrensburg, MO, USA
[g] School of Education, Health, and Human Performance, Fairmont State University, Fairmont, WV, USA
[h] Department of Pharmaceutical Sciences and Research, School of Pharmacy, Marshall University, Huntington, WV, USA
[i] Department of Pharmacology, Physiology and Toxicology, Joan C. Edwards School of Medicine, Marshall University, Huntington, WV, USA

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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 proteins in the lean and obese (fa/fa) Zucker rat soleus 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 et al., 1991) [1–3] and concurrent Data in Brief articles (Ginjupalli et al., 2017a, 2017b; Rice et al., 2017a, 2017b) [4–7].

* Correspondence to: Center for Diagnostic Nanosystems, Room 241D Robert C. Byrd Biotechnology Science Center, 1700 3rd Ave. Marshall University, Huntington, WV 25755-1090, USA. Fax: +304 696 3766.
E-mail address: rice9@marshall.edu (K.M. Rice).

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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-minute period. Tissues were collected and protein was then isolated from tissue for western blot analysis.*
Experimental features	*Soleus 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 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 high-frequency electrical stimulation (HFES) on soleus in diabetic male obese syndrome-X Zucker (OSXZ) diabetic and nondiabetic male normal lean Zucker (LNZ) animals we evaluated the expression of AKT. Soleus basal AKT content was lower (9.8 $\pm$ 1.9%, $p < 0.05$) in the OSXZ when compared to LNZ. HFES resulted in a decrease in AKT in the LNZ soleus (22.7 $\pm$ 1.9%, 9.8 $\pm$ 3.2%, at 0 and 3 hours, $p < 0.05$) when compared to LNZ contralateral control. However, HFES elicited no change in the OSZX soleus when compared to contralateral OXSZ control (Fig. 1).

To determine the effect of HFES on soleus in OSXZ and LNZ animals we evaluated the phosphorylation of AKT at serine 473. Soleus basal phosphorylation of AKT ser 473 demonstrated no difference in the OSXZ when compared to LNZ. HFES elicited not change in phosphorylation of AKT ser 473 in the LNZ soleus when compared to LNZ contralateral control. HFES resulted in an increase in phosphorylation of AKT ser 473 in the OSXZ soleus (58.0 $\pm$ 16.7%, at 0 h, $p < 0.05$) when compared to OSXZ contralateral control (Fig. 1).

To determine the effect of HFES on soleus in OSXZ and LNZ animals we evaluated the ratio of phosphorylation of AKT ser 473 to total AKT. Soleus basal phosphorylation of AKT ser 473 to total AKT demonstrated no difference in the OSXZ when compared to LNZ. HFES resulted in an increase in phosphorylation of AKT ser 473 to total AKT in the LNZ soleus (70.1 $\pm$ 29.8%, and 64.3 $\pm$ 19.1%, at 0 and 3 h, $p < 0.05$) when compared to LNZ contralateral control. HFES resulted in an increase in phosphorylation of AKT ser 473 to total AKT in the OSXZ soleus (63.17% at 0 h, $p < 0.05$) when compared to OSXZ contralateral control (Fig. 1).