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Data Article

Data on skeletal muscle apoptosis, autophagy, and morphology in mice treated with doxorubicin

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ABSTRACT

Skeletal muscle apoptosis and autophagy are catabolic processes that contribute to muscle atrophy during aging, disease, and following muscle injury. In this article, we present data on skeletal muscle apoptosis, autophagy, and morphology in C57BL/6 mice following doxorubicin administration. More specifically, time-course data on caspase-3, caspase-8, caspase-9, calpain, and cathepsin activity are presented, along with data on ATG7, p62, LC3-I, and LC3-II protein expression. Data on skeletal muscle reactive oxygen species (ROS) production, muscle morphology, as well as body and muscle weights are also presented.

Specification Table

Subject area	*Biology*
More specific subject area	*Skeletal muscle, apoptosis, autophagy*
Type of data	*Images, graphs, figures*
How data was acquired	*Microscopy, spectrofluorometry, immunoblotting*
Data format	*Analyzed*

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Experimental factors	*C57BL/6 mice received a single intraperitoneal injection of doxorubicin and were sacrificed at given time points.*
Experimental features	*C57BL/6 mice were sacrificed prior to (i.e. day 0) and 1, 3, 5, 7, and 9 days after a single intraperitoneal injection of doxorubicin. Different skeletal muscles were isolated and weighed, and samples prepared for microscopy, spectrofluorometric assays, and immunoblotting.*
Data source location	*University of Waterloo, Waterloo, Ontario, Canada*
Data accessibility	*All data are provided with this article*

Value of the data

- Provides a simultaneous assessment of skeletal muscle apoptosis, autophagy, and morphology in response to systemic doxorubicin administration.
- Provides time-course data on skeletal muscle morphology and degradative processes following doxorubicin administration.
- Valuable for researchers interested in the relationship between apoptosis and autophagy in skeletal muscle.
- Help other researchers determine the utility of doxorubicin as a myotoxic agent.

1. Data

Here we present data regarding the ability of systemic doxorubicin administration to induce skeletal muscle atrophy and morphological changes. Given that apoptosis and autophagy can impact muscle morphology and wasting [1,2] we also provide data on apoptotic and autophagic signaling in several muscles following doxorubicin administration.

1.1. Body and muscle weights following doxorubicin administration

There was a trend ($p=0.07$) towards a significant decrease in body weight after doxorubicin administration (Fig. 1A). There were no statistically significant differences in soleus, plantaris, or gastrocnemius weights with doxorubicin. Further, there were no significant differences in muscle weights when normalized to body weight for both the soleus (Fig. 1B) or plantaris (Fig. 1C) following doxorubicin administration. However, post hoc analysis revealed a significant ($p<0.05$) difference in gastrocnemius weight when normalized to body weight at day 1 compared to day 9 (Fig. 1D).

1.2. Apoptotic enzyme activity and reactive oxygen species (ROS) generation following doxorubicin treatment

There were no statistically significant differences in caspase-3 (Fig. 2A), caspase-8 (Fig. 2B), caspase-9 (Fig. 2C), or calpain (Fig. 2D) activity, nor ROS production (Fig. 2E) in mixed gastrocnemius between any of the measured time points (Day 0, 1, 3, 5, 7, and 9) following doxorubicin administration.

1.3. Autophagic protein expression and cathepsin activity after doxorubicin administration

There were no differences in ATG7 (Fig. 3A and C) or p62 (Fig. 3A and D) protein between any of the measured time points in the soleus following doxorubicin administration. Post hoc analysis revealed a significant ($p<0.05$) increase in LC3-I protein in soleus on day 3 post-doxorubicin injection compared to day 1 (Fig. 3A and E). There was no statistically significant difference in LC3-II protein (Fig. 3A and F), nor the LC3-II/LC3-I protein ratio (Fig. 3A and G) in the soleus at any time point following doxorubicin administration. There were no significant differences in ATG7 protein (Fig. 3B