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Data in Brief

Genome-wide expression analysis comparing hypertrophic changes in normal and dysferlinopathy mice

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ABSTRACT

Because myostatin normally limits skeletal muscle growth, there are extensive efforts to develop myostatin inhibitors for clinical use. One potential concern is that in muscle degenerative diseases, inducing hypertrophy may increase stress on dystrophic fibers. Our study shows that blocking this pathway in *dysferlin* deficient mice results in early improvement in histopathology but ultimately accelerates muscle degeneration. Hence, benefits of this approach should be weighed against these potential detrimental effects. Here, we present detailed experimental methods and analysis for the gene expression profiling described in our recently published study in Human Molecular Genetics (Lee et al., 2015). Our data sets have been deposited in the Gene Expression Omnibus (GEO) database (GSE62945) and are available at http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE62945. Our data provide a resource for exploring molecular mechanisms that are related to hypertrophy-induced, accelerated muscular degeneration in dysferlinopathy.

Specifications	
Organism/cell line/tissue	*Mus musculus*/quadriceps muscle
Sex	Male
Sequencer or array type	Affymetrix Mouse Exon 1.0 ST arrays
Data format	Raw data: CEL. Processed data: SOFT, MINiML, TXT.
Experimental factors	Genetically and pharmacologically induced muscular hypertrophy in normal vs. dysferlinopathy
Experimental features	Genome-wide expression analysis comparing hypertrophic changes in normal (*wild-type*, *wt*) and dystrophic (*dysferlin*-deficient, $Dysf^{-/-}$) mouse muscles induced by genetic (follistatin overexpression, *F66*) and pharmacological (administration of activin type II soluble receptor, ACVR2B/Fc) approaches
Consent	N/A
Sample source location	N/A

1. Direct link to deposited data

Raw and processed microarray data is available in GEO under accession GSE62945 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE62945.

Genotype	ACVR2B/Fc treatment	Replicate	GEO accession URL
wt	No	3	http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536838 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536839 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536840
wt	Yes	3	http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536841 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536842 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536843
$Dysf^{-/-}$	No	3	http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536844 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536845 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536846
$Dysf^{-/-}$	Yes	3	http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536847 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536848 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536849
F66	No	3	http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536850 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536851 http://www.ncbi.nlm.nih.gov/geo/query/

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(*continued*)

Genotype	ACVR2B/Fc treatment	Replicate	GEO accession URL
F66;Dysf$^{-/-}$	No	3	acc.cgi?acc=GSM1536852 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536853 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536854 http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSM1536855

2. Experimental design, materials and methods

2.1. Study design

The identification of myostatin as a negative regulator of skeletal muscle mass raised the possibility that blocking the myostatin signaling could have important applications for treating patients with muscle degenerative diseases [1]. However, there is one theoretical concern that inducing muscle hypertrophy may cause additional membrane stress,

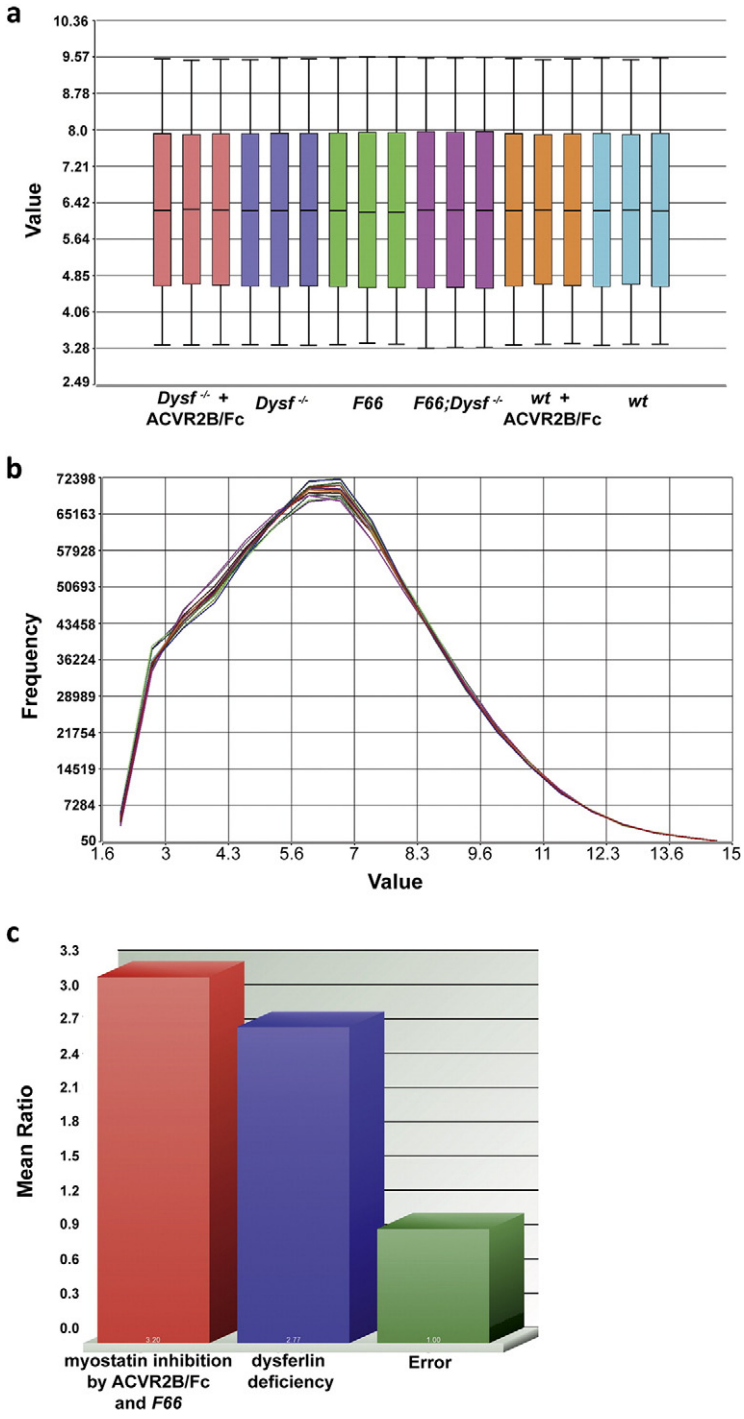

Fig. 1. Microarray quality assessment of 18 chips for three biological replicates in 6 different groups; *wt*, *Dysf*$^{-/-}$, *F66*, *F66;Dysf*$^{-/-}$, and ACVR2B/Fc-injected *wt* and *Dysf*$^{-/-}$ mice. (a) Box plots of 18 chips show median-centered raw data distributions. (b) Line graphs of 18 chips also support that all the chips' probes have signals of similar distribution and median value. (c) The magnitude of between class variation (myostatin inhibition by ACVR2B/Fc and *F66*, and dysferlin deficiency) is compared to that of within-class variation (Error), demonstrating biological variation to be far greater than experimental noise.