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Data Article

Data on protein abundance alteration induced by chronic exercise in mdx mice model of Duchenne muscular dystrophy and potential modulation by apocynin and taurine



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ABSTRACT

Here we present original data related to the research paper entitled “Proteome analysis in dystrophic mdx mouse muscle reveals a drastic alteration of Key Metabolic and Contractile Proteins after chronic exercise and the potential modulation by anti-oxidant compounds” (Gamberi et al., 2018) [1]. The dystrophin-deficient mdx mouse is the most common animal model for Duchenne muscular dystrophy. The mdx mice phenotype of the disorder is milder than in human sufferers and it can be worsened by chronic treadmill exercise. Apocynin and taurine are two antioxidant compounds proved to be beneficial on some pathology related parameters (Schröder and Schoser, 2009) [2]. This article reports the detailed proteomic data on protein abundance alterations, in tibialis anterior muscle of mdx mice, induced by chronic exercise protocol. A selected group of mdx mice was also treated with apocynin and taurine during this protocol. Detailed MS data, comparison between mdx vs wild type, exercised mdx vs wild

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type, and complete analysis of spot variation are provided. Furthermore, in wild type mice subjected to the same exercise protocol, the abundance of key proteins, resulted modified in exercised mdx, were analyzed by western blot.

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Specifications Table

Subject area	Biology
More specific subject area	<i>Mdx mice model for Duchenne muscular dystrophy.</i>
Type of data	<i>Table, text file, graph</i>
How data was acquired	<i>2DE gels were analyzed with Progenesis SameSpots software v4.0 (Nonlinear Dynamics, UK). MS and MSMS data were obtained with Ultraflex III MALDI- TOF/TOF mass spectrometer (Bruker Daltonics)</i>
Data format	<i>Analyzed</i>
Experimental factors	<i>Effect of chronic exercise on muscle protein abundance in mdx mice model for Duchene muscular dystrophy. Modulation by two natural compounds apocynin and taurine</i>
Experimental features	<i>Animal model. Male mdx mice divided in:</i> <i>-sedentary mdx (mdx) mice</i> <i>-exercised mdx (mdx exe) mice</i> <i>- mdx exercised mice treated with taurine (mdx exe tau)</i> <i>-mdx exercised mice treated with apocynin (mdx exe apo)</i> <i>-C57/BL wild-type mice exercised (wt exe) and control (wt).</i> <i>Age-matched male wild-type mice (C57BL/10) has been used as referring phenotype. The training protocol consisted of a 30 min running on a horizontal treadmill (Columbus Instruments, USA) at 12 m/min, twice a week for at least 4 weeks. The doses of taurine and apocynin were 1 g/kg (orally) and 38 mg/kg (1.5 mmol/l in drinking water) respectively.</i> <i>Proteomics: 2DE and MS were used in order to identify differences in protein abundance between groups.</i>
Data source location	<i>Male mdx mice (C57BL/10ScSn-Dmdmdx/J from Jackson Laboratories) and C57/BL wild-type (wt) mice (from Jackson Laboratories)</i>
Data accessibility	<i>Data is provided by this article</i>

Value of the data

- These data report for the first time the effect of chronic exercise protocol on protein abundance in mdx mice.
- These data can provide information about muscle damage induced by an inappropriate exercise in dystrophic patients.
- These data show the ability of taurine and apocynin to counteract some of exercise-induced protein alterations.

Table 1

Differentially abundant protein spots that significantly differed between groups, identified by MALDI-TOF/TOF mass spectrometry analysis. The complete list of the proteins, identified by MALDI-TOF is reported in [1].

Spot No ^a	Protein name	AC ^b	Gene Name	Cellular component Go term	Theoretical	Observed	Mascot search results			Peptide Sequence ^g
					Mr (kDa)/ pI	Mr (kDa)/ pI ^c	Score ^d	Matched Pept. ^e	Seq. cov- erage (%) ^f	
Sarcomere organization and muscle contraction										
1	LIM domain-binding protein 3	Q9JKS4	Ldb3	Z-disc	77.6/7.9	30.1/9.7	86	9/45	17%	[21-32] K.DFNMPLTISR.I [37-69] K.AAQSQLSQGDLVVAIDGVNTDTMTHLEAQNK.I [70-83] K.SASYNLSLTLQK.S
3	LIM domain-binding protein 3	Q9JKS4	Ldb3	Z-disc	77.6/7.9	30.2/9.3	76	8/34	16%	[21-32] K.DFNMPLTISR.I [273-294] R.ILAQMTGTGFMDPPDEEALR.R
6	Myozenin-1	Q9JK37	Myoz1	Cytoskeleton	31.4/8.6	31.7/7.9	121	15/77	67%	[42-57] R.DVMLEELSLLTNR.G [69-90] K.FIYNHPDVFSDSSMDHFQK.F
11	Troponin T, fast skeletal muscle	Q9QZ47	Tnnt3	Troponin complex	32.2/5.3	31.5/7.8	82	10/43	33%	[61-76] K.IPEGEKVFDDIQK.K [159-175] K.ALSSMGANYSSYLAK.A
12	Troponin T, fast skeletal muscle	Q9QZ47	Tnnt3	Troponin complex	32.2/5.3	31.9/9.2	74	8/27	26%	[61-76] K.IPEGEKVFDDIQK.K [159-175] K.ALSSMGANYSSYLAK.A
13	Myosin regulatory light chain 2, skeletal muscle isoform	P97457	MyIpf	Myosin complex	19/4.8	16.1/4.8	88	10/42	63%	[31-42] K.EAFTVIDQNR.D [41-52] R.DGIIDKEDLR.D [63-73] K.NEELDAMMK.E [92-106] K.GADPEDVITGAFK.V
16	Myosin regulatory light chain 2, skeletal muscle isoform	P97457	MyIpf	Myosin complex	19/4.8	17.1/4.9	72	6/36	37%	[31-42] K.EAFTVIDQNR.D [41-52] R.DGIIDKEDLR.D [92-106] K.GADPEDVITGAFK.V
23	Actin, alpha skeletal muscle and Actin, alpha cardiac muscle1	P68134 and P68033	Acta1 and Actc1	Cytoskeleton	42.3/5.2	42.4/5.2	72	14/32	44%	[97-116] R.VAPEEHPHTLLEAPLNPK.A [240-257] K.SYELPDGQVITIGNER.F
	Transitional endoplasmic reticulum ATPase (mix) ^g	Q01853	Vcp	Proteasome complex	89.9/5.1	42.4/5.2	73	8/30	27%	[25-46] R.LIVDEAINEDNSVLSQPK.M [295-313] K.NAPAIIFIDELDAIAPK.R
Metabolism										
<i>(Glucose metabolism)</i>										
30	Fructose-bisphosphate aldolase A	P05064	Aldoa	cytoplasm	39.7/8.3	30.4/7.1	60	6/25	16%	[28-43] K.GILAADESTGSIAR.R [111-135] K.GVVPLAGTNGETTQGLDGLSER.C [173-201] R.YASICQQNGVIVPEILPDGDHDLK.R
34	Triosephosphate isomerase	P17751	Tpi1	cytoplasm	32.7/5.5	25/6.7	91	8/26	34%	[56-65] K.FFVGGNWK.M

Table 1 (continued)

Spot No ^a	Protein name	AC ^b	Gene Name	Cellular component Go term	Theoretical	Observed	Mascot search results			Peptide Sequence ^d
					Mr (kDa)/ pI	Mr (kDa)/ pI ^c	Score ^d	Matched Pept. ^e	Seq. cov- erage (%) ^f	
37	Beta-enolase	P21550	Eno3	cytoplasm	47.3/6.7	46.6/6.3	95	8/22	23%	[150–163] R.HVFGESDELIGQK.V [256–270] R.IYGGSVTGATCK.E [15–29] R.GNPTVEVDLHTAK.G [239–254] K.VVIGMDVAASEFYR.N
48	(Respiratory chain complex) NADH dehydrogenase [ubi- quinone] flavoprotein 2, mitochondrial	Q9D6J6	Ndufv2	mitochondrion	27.6/7	23.9/5.4	71	8/32	38%	[238–247] K.GPGFGVQAGL [110–124] R.VYEVATFYTMYNR.K [41–61] R.DTPENNPDPFDFTPENYK.R [334–348] R.EAYPGDVFLHLSR.L
51	ATP synthase subunit alpha, mitochondrial	Q03265	Atp5a1	mitochondrion	59.8/9.22	22.5/6.6	72	7/22	17%	
55	Energy transfert Creatine kinase M-type	P07310	Ckm	cytoplasm	43.2/6.6	24.3/6.3	61	7/20	21%	[116–131] K.GGDDLDPNYVLSR.V [156–171] K.LSVEALNSLTGEFK.G [116–131] K.GGDDLDPNYVLSR.V [156–171] K.LSVEALNSLTGEFK.G [156–171] K.LSVEALNSLTGEFK.G
57	Creatine kinase M-type	P07310	Ckm	Cytoplasm	43.2/6.6	29/6.6	66	10/39	27%	[210–215] R.DWPDAR.G [223–237] K.SFLVWVNEEDHLR.V [259–267] K.IEEIFKK.A [269–381] K.GQSIDDMIPAQK. [341–359] R.LGSSEVEQVLVVDGVK.L
58	Creatine kinase M-type	P07310	Ckm	Cytoplasm	43.2/6.6	29.7/6.6	61	7/35	21%	[9–22] K.IIFVVGPGSGK.G [31–45] K.YGYTHLSTGDLLR.A [9–22] K.IIFVVGPGSGK.G [131–139] K.RGETSGR.V [139–148] R.VDDNEETIKK.R
60	Creatine kinase M-type	P07310	Ckm	Cytoplasm	43.2/6.6	17.4/7.9	68	9/34	29%	
70	Adenylate kinase iso- enzyme 1	Q9R0Y5	Ak1	Cytoplasm	21.6/5.7	21.5/5.3	58	5/20	36%	
71	Adenylate kinase iso- enzyme 1	Q9R0Y5	Ak1	Cytoplasm	21.6/5.7	22/5.5	104	11/40	55%	
87	Transport Voltage-dependent anion- selective channel protein 1	Q60932	Vdac1	Mitochondrion	32.5/8.5	29.8/8.6	74	38%	6/21	[109–123] K.LTFDSSFSPNTGK.K [87–107]K.WNTDNTLGTTEIVDQLAR. G [250–270] K.VNNSLIGLGYTQTLKPGIK.L

^a Spot numbers match those reported in the representative 2DE images shown in Fig. 1 and Table 1 in ref. [1]

^b Accession number in Swiss-Prot/UniprotKB.

^c Based on the calculation using Progenesis SameSpots 4.0 software

^d MASCOT MS score (Matrix Science, London, UK; <http://www.matrixscience.com>). MS matching score greater than 56 was required for a significant MS hit (p -value < 0.05).

^e Number of matched peptides correspond to peptide masses matching the top hit from Ms-Fit PMF, searched peptide are also reported.

^f Sequence coverage = (number of the identified residues/total number of amino acid residues in the protein sequence) x100%.

^g Peptide sequence obtained by Maldi TOF/TOF analysis using an Ultraflex III MALDI- TOF/TOF mass spectrometer (Bruker Daltonics).

Table 2

Sequence coverage (in bold) of identified proteins that show an experimental Mr different from expected.

Spot No ^a	AC ^b	Gene Name	^c Sequence coverage	^d Theoretical Mr (kDa)/ pI	^e Observed Mr (kDa)/ pI ^e
1	Q9JKS4	Ldb3	1 MSYSVTLTGP GPWGFRLQGG KDFNMPLTIS RITPGSKAAQ SQLSQGDLVV 51 AIDGVTDTM THLEAQNKIK SASYNLSLTL QKSKRPIPISTTAPPIQSPL 101 PVIPHQKDKPA LDTNGSLATP SPSPEARASP GALEFGDTFS SSFSQTSVCS 151 PLMEASGPVL PLGSPVAKAS SEGAQGSVSP KVLPGPSQPR QYNNPIGLYS 201 AETLREMAQM YQMSLRGKAS GAGLLGSLP VKDLAVDSAS PVYQAVIKTQ 251 SKPEDEADEW ARRSSNLQSR SFRILAQMTG TEYMQDPDEE ALRRSSTPIE 301 HAPVCTSQAT SPLLPASAQS PAAASPIAAS PTLATAAATH AAAASAAGPA 351 ASPVENPRPQ ASAYSPAAAA SPAPSAHTSY SEGPAAPAPK PRVVTASIR 401 PSVYQVPAS SYSPSPGANY SPTPYTPSPA PAYTPSPAPT YTPSPAPTYS 451 PSPAPAYTPS PAPNYTPPS AAYSGGPSES ASRPPVVTDD SFSQKFAPGK 501 STTVSKQTL PRGAPAYNPT GPQVTPLARG TFQRAERFPA SSRTPLCGHC 551 NNVIRGPFLV AMGRSWHPPEE FNCAYCKTSL ADVCFVEEQN NVCERCYEQ 601 FFAPICAKCN TKIMGEVMHA LRQTWHHTCF VCAACKKPFV NSLFHMEDGE 651 PYCEKDYINL FSTKCHGCDP PVEAGDKFIE ALGHTWHHTC FICAVCHVNL 701 EQQPFYSKDD KPLCKKHAHA INV	77.6/.79	30.1/9.7
2	Q9JKS4	Ldb3	1 MSYSVTLTGP GPWGFRLQGG KDFNMPLTIS RITPGSKAAQ SQLSQGDLVV 51 AIDGVTDTM THLEAQNKIK SASYNLSLTL QKSKRPIPISTTAPPIQSPL 101 PVIPHQKDKPA LDTNGSLATP SPSPEARASP GALEFGDTFS SSFSQTSVCS 151 PLMEASGPVL PLGSPVAKAS SEGAQGSVSP KVLPGPSQPR QYNNPIGLYS 201 AETLREMAQM YQMSLRGKAS GAGLLGSLP VKDLAVDSAS PVYQAVIKTQ 251 SKPEDEADEW ARRSSNLQSR SFRILAQMTG TEYMQDPDEE ALRRSSTPIE 301 HAPVCTSQAT SPLLPASAQS PAAASPIAAS PTLATAAATH AAAASAAGPA 351 ASPVENPRPQ ASAYSPAAAA SPAPSAHTSY SEGPAAPAPK PRVVTASIR 401 PSVYQVPAS SYSPSPGANY SPTPYTPSPA PAYTPSPAPT YTPSPAPTYS 451 PSPAPAYTPS PAPNYTPPS AAYSGGPSES ASRPPVVTDD SFSQKFAPGK 501 STTVSKQTL PRGAPAYNPT GPQVTPLARG TFQRAERFPA SSRTPLCGHC 551 NNVIRGPFLV AMGRSWHPPEE FNCAYCKTSL ADVCFVEEQN NVCERCYEQ 601 FFAPICAKCN TKIMGEVMHA LRQTWHHTCF VCAACKKPFV NSLFHMEDGE 651 PYCEKDYINL FSTKCHGCDP PVEAGDKFIE ALGHTWHHTC FICAVCHVNL 701 EQQPFYSKDD KPLCKKHAHA INV	77.6/.79	29.6/9.7

Table 2 (continued)

Spot No ^a	AC ^b	Gene Name	^c Sequence coverage	^d Theoretical Mr (kDa)/ pI	^e Observed Mr (kDa)/ pI ^e
3	Q9JKS4	Ldb3	<p>1 MSYSVTLTGP GPWGFRLQGG KDFNMPLTIS RITPGSKAAQ SQLSQGDIVV 51 AIDGVNTDTM THLEAQNKIK SASYNLSLTL QKSKRPIPI S TTAPPIQSPL 101 PVIPHQKDPALDTNGSLATP SPSPEARASP GALEFGDTFS SFSQTSVCS 151 PLMEASGPVL PLGSPVAKAS SEGAQGSVSP KVLPGPSQPR QYNNPIGLYS 201 AETLREMAQM YQMSLRGKAS GAGLLGGSPL VKDLAVDSAS PVYQAVIKTQ 251 SKPEDEADEW ARRSNLQSR SFRILAQMTG TEYMQDPDEE ALRRSSTPIE 301 HAPVCTSQAT SPLLPASAQS PAAASPIAAS PTLATAAATH AAAASAAGPA 351 ASPVENPRPQ ASAYSPAAAA SPAPSAHTSY SEGPAAPAPK PRVVTASIR 401 PSVYQVPAS SYSPSGANY SPTPYTPSPA PAYTPSPAPT YTPSPAPTYS 451 PSPAPAYTPS PAPNYTTPS AAYSGGPSES ASRPPVWTD SFSQKFAPGK 501 STTVSKQTL PRGAPAYNPT GPQVTLARG TFQRAERFPA SSRTPLCGHC 551 NNVIRGPFLV AMGRSWHPEE FNCAYCKTSL ADVCFVEEQN NVYCERCYEQ 601 FFAPICAKCN TKIMGEVMHA LRQTWHHTCF VCAACKKPFQ NSLFHMDGGE 651 PYCEKDYINL FSTKCHGCFD PVEAGDKFIE ALGHTWHDT C FICAVCHVNL 701 EGQPFYSKKD KPLCKKHAHA INV</p>	77.6/7.9	30.2/9.3
51	Q03265	Atp5a1	<p>1 MLSVRVAAAV ARALPRRAGL VSKNALGSSF VGARNLHASN TRLQKTGTAE 51 MSSILEERIL GADTSVDLEE TGRVLSIGDG IARVHGLRNV QAEEMVEFSS 101 GLKGMSLNLE PDNVGVVVFV NDKLIKEGDV VKRTGAIVDV PVGELLGRV 151 VDALGNAIDG KGPIGSKTRR RVGLKAPGII PRISVREPMQ TGIAKAVDSL 201 PIGRGQRELI IGDRQTGKTS IAIDTIINQK RFNDGTDEKK KLYCIYVAIG 251 QKRSTVAQLV KRLTDADAMK YTIIVSATAS DAAPLQYLAP YSGCSMGEYF 301 RDNGKHAI I YDDLKQAVA YRQMSLLRR PPGREAYPGD VFYLHSLRLE 351 RAAKMNSDFG GGSALTALPVI ETQAGDVSA Y IPTNVISITD GQIFLETFL 401 YKGIRPAINV GLSVSRVGSA AQTRAMKQVA GTMKLELAQY REVAFAAQFQ 451 SDLDAATQQL LSRGVRLTEL LKQGOYSPMA IEEQVAVIYA GVRGYLDKLE 501 PSKITKFENA FLSHVISQHQ SLGNIRSDG KISEQSDAKL KEIVTNFLAG 551 FEP</p>	59.8/9.22	22.5/6.6
55	P07310	Ckm	<p>1 MPFGNTHNKF KLNYPQEEY PDLKSHNNHM AKVLTDPDLYN KLRDKETPSG 51 FTLDDVIQTG VDNPGHPFIM TVGCVAGDEE SYTVFKDLFD PIIQDRHGGY 101 KPTDKHKTDL NHENLKGDD LDPNYVLSR VRTGRSIKGY TLPPHCSRGE 151 RRAVEKLSVE ALNSLTGEFK GKYYPLKSMT EQEQQLIDD HFLFDKPVSP 201 LLLASGMARD WPDARGIWHN DNKSFLVWVN EEDHLRVISM EKGGMKEVF 251 RRFVGLQKI EEIFKAGHP FMWNEHLGYV LTCPSNLGT LRGVHVKLA 301 NLSKHPKFEI ILTRLRQKR GTGGVDTAAV GAVFDISNAD RLGSSVEVQV 351 QLVVDGVKLM VEMEKKLEKG QSIDDMIPAQ K</p>	43.2/6.6	24.3/6.3

56	P07310	Ckm	<p>1 MPFGNTHNKF KLNYPQEEY PDLSKHNNHM AKVLTDPDLYN KLRDKETPSG 51 FTLDDVIQTG VDNPGHPFIM TVGCVAGDEE SYTVFKDLFD PIIQDRHGGY 101 KPTDKHKTDL NHENLKGDD LDPNYLSSR VRTGRSIKGY TLPPHCSRGE 151 RRAVEKLSVE ALNSLTGEFK GKYYPLKSMT EQEQQLIDD HFLFDKPVSP 201 LLLASGMARD WPDARGIWHN DNKSFLVWVN EEDHLRVISM EKGGNMKEVF 251 RRFCVGLQKI EEIFKKAGHP FMWNEHLGYV LTCPSNLGTG LRGVHVHVKLA 301 NLSKHPKFEE ILTRLRQKR GTGGVDAAV GAVFDISNAD RLGSSVEQV 351 QLVVDGVKLM VEMEKKLEKG QSIDDMIPAQ K</p>	43.2/6.6	28.8/6.6
57	P07310	Ckm	<p>1 MPFGNTHNKF KLNYPQEEY PDLSKHNNHM AKVLTDPDLYN KLRDKETPSG 51 FTLDDVIQTG VDNPGHPFIM TVGCVAGDEE SYTVFKDLFD PIIQDRHGGY 101 KPTDKHKTDL NHENLKGDD LDPNYLSSR VRTGRSIKGY TLPPHCSRGE 151 RRAVEKLSVE ALNSLTGEFK GKYYPLKSMT EQEQQLIDD HFLFDKPVSP 201 LLLASGMARD WPDARGIWHN DNKSFLVWVN EEDHLRVISM EKGGNMKEVF 251 RRFCVGLQKI EEIFKKAGHP FMWNEHLGYV LTCPSNLGTG LRGVHVHVKLA 301 NLSKHPKFEE ILTRLRQKR GTGGVDAAV GAVFDISNAD RLGSSVEQV 351 QLVVDGVKLM VEMEKKLEKG QSIDDMIPAQ K</p>	43.2/6.6	29/6.6
58	P07310	Ckm	<p>1 MPFGNTHNKF KLNYPQEEY PDLSKHNNHM AKVLTDPDLYN KLRDKETPSG 51 FTLDDVIQTG VDNPGHPFIM TVGCVAGDEE SYTVFKDLFD PIIQDRHGGY 101 KPTDKHKTDL NHENLKGDD LDPNYLSSR VRTGRSIKGY TLPPHCSRGE 151 RRAVEKLSVE ALNSLTGEFK GKYYPLKSMT EQEQQLIDD HFLFDKPVSP 201 LLLASGMARD WPDARGIWHN DNKSFLVWVN EEDHLRVISM EKGGNMKEVF 251 RRFCVGLQKI EEIFKKAGHP FMWNEHLGYV LTCPSNLGTG LRGVHVHVKLA 301 NLSKHPKFEE ILTRLRQKR GTGGVDAAV GAVFDISNAD RLGSSVEQV 351 QLVVDGVKLM VEMEKKLEKG QSIDDMIPAQ K</p>	43.2/6.6	29.7/6.6
59	P07310	Ckm	<p>1 MPFGNTHNKF KLNYPQEEY PDLSKHNNHM AKVLTDPDLYN KLRDKETPSG 51 FTLDDVIQTG VDNPGHPFIM TVGCVAGDEE SYTVFKDLFD PIIQDRHGGY 101 KPTDKHKTDL NHENLKGDD LDPNYLSSR VRTGRSIKGY TLPPHCSRGE 151 RRAVEKLSVE ALNSLTGEFK GKYYPLKSMT EQEQQLIDD HFLFDKPVSP 201 LLLASGMARD WPDARGIWHN DNKSFLVWVN EEDHLRVISM EKGGNMKEVF 251 RRFCVGLQKI EEIFKKAGHP FMWNEHLGYV LTCPSNLGTG LRGVHVHVKLA 301 NLSKHPKFEE ILTRLRQKR GTGGVDAAV GAVFDISNAD RLGSSVEQV 351 QLVVDGVKLM VEMEKKLEKG QSIDDMIPAQ K</p>	43.2/6.6	24.4/6.5

Table 2 (continued)

Spot No ^a	AC ^b	Gene Name	^c Sequence coverage	^d Theoretical Mr (kDa)/ pI	^e Observed Mr (kDa)/ pI ^e
60	P07310	Ckm	1 MPFGNTHNKF KLNYPQEEY PDLSKHNNHM AKVLTPLDLYN KLRDKETPSG 51 FTLDDVIQGT VDNPGHPFIM TVGCVAGDEE SYTVFKDLFD PIQDRHGGY 101 KPTDKHKTDL NHENLKGDD LDPNYVLSR VRTGRSICY TLPPhCSRGE 151 RRAVEKLSVE ALNSLTGEFK GKYYPLKSMT EQEQQLIDD HFLFDKVPSP 201 LLLASGMARD WPDARGIWHN DNKSFLVWVN EEDHLRVISM EKGGNMKEVF 251 RRFCVGLQKI EEIFKKAGHP FMWNEHLGYV LTCPSNLGTG LRGGVHVKLA 301 NLSKHPKFEE ILTRLRQKR GTGGVDTAAV GAVFDISNAD RLGSEVEQV 351 QLVVDGVKLM VEMEKKLEKG QSIDDMIPAQ K	43.2/6.6	17.4/7.9
90	Q9R1S8	Capn7	1 MDASALERDA VQFARLAVQR DHEGRYSEAV FYYKEAAQAL IYAEMAGSSL 51 ERIQEKINEY LERVQALHSA VQSKSTDPLK SKHQDLERA HFLVTQAFDE 101 DEKGNVEDAI ELYTEAVELC LKTSSETADK TLQNKLKQLA RQALDRAEAL 151 SEPLTKPFCK LKSANMKTCT PPVRTHFPLG PNPFVEKPQA FISPQSCDAQ 201 GQKYTAEIE VLRTTSKING VEYVPFMSVD LRERFAYPMP FCDRLGKPL 251 SPKQKTTFSK WVRPEDLTNN PTMIYTVSSF SIKQTVSDC SFVASLAISA 301 AYERRFNKKL ITSIIYPQNK DGEPEYNPCG KYMVKLHLNG VPRKVIIDDQ 351 LPVDHKGELL CSYSNNKSEL WVSLIEKAYM KVMGGYDFPG SNSNIDLHAL 401 TGWIPERIAM HSDSQTFSKD NSFRMLYQRF HKGDVLITAS TGMTEAEGE 451 KWGLVPTHAY AVLDIREFKG LRFIQLKNPW SHLRWKGRYS ENDVKNWTP 501 LQKYLNFDP 551 STWDAKQGPV KDAYSLANNP QYKLEVQCPQ GGAAVVVLLS RHITDKDDFA 601 NNREFITMVV YKTDGKKVYV PADPPPYIDG IRINSPHYLT KIKLTTPGTH 651 TFLVVSQYE KQNTIHYTVR VYSACSTFS KIPSPYTLK RINGKWSGQS 701 AGCGNFQET HKNNPIYQFH IDKTGPLLIE LRGPRQYSVG FEVVAVSIMG 751 DPGPHGFQRK SSGDYRCGFC YLELENIPAG IFNIIPSTFL PKQEGPFLLD 801 FNSTVPIKTT QLQ	93.3/8.1	17.6/10.3

1 MYKSVSETRH PLQSEEQEVG IDPLFSYSNK TRGDLSQNGR GSNSTLDTEG
51 TFNSYMKWEWE ELFVNNNNYLA TVRQKGINGQ LRSSRFRSIC WKLFCLVLPQ
101 DKSQWISKIK ELRAWYSSIK EIHITNPRKA AGQODLMINN PLSQDEGSLW
151 **NKFFQDKELR SMIEQDVKRT** FPQMFFQQE NVRKILTDVL FCYARENEQL
201 LYKQGMHELL APIIFTLHCD HQAFLHASES AQPSEEMKTL LNPEYLEHDA
251 YAMFSQLMET AEPWFSTFEH DGQKGKETLM APIPFARPQD LGPTVAIVTK
301 VNQIQDHLK KHDIELYMLH NRLEIAPQIY GLRWVRLFG REFPLQDLLV
351 VWDALFADSL NLSLVDYVFT AMLLYIRDAL ISSNYQTCLG LLMHYPIIGD
401 IHSLILKALF LRDPKRNPRP ATYQFHPNLD YYKARGADLM NKSRTNARGA
451 PLNIHKVSNS LINFGRKLIS **PASAPGSMGG PVPGNSSSS FSAIIPTRTS**
501 TEAPRHLLQ QQQQQHQQQ QQQQPQQQQ QHQQQQQQR LMKSESMPVQ
551 LNKGQSSKI SSSPSIESLP GGREFTGSPS PSATKKDSFF SNIARSRSHS
601 KTMGRKESEE ELEAQISFLQ GQLNDLDAMC **KYCAKVMMDMH LVNIQDVLQ**
651 **ENLEKEDQIL** VSLAGLKQJK DILKGLRFN QSQLEAGENE QITIADDDHYC
701 SSGDQGSQV PRAAKQASSE MPGCTGGTTP DDFILVSKED EGHRRARGAFS
751 GQAQPLTLR STSGKSRAPA **CSPLLFSDPL MGPASASASS SNPSSSPDD**
801 SSKESGFTIV SPLDI

^a Spot numbers match those reported in the representative 2DE images shown in Fig. 1 and Table 1 in ref. [1]

^b Accession number in Swiss-Prot/UniprotKB.

^c Sequence coverage refers to the identified peptides of the protein sequence (bold letters).

^d Theoretical molecular mass (Mr) and isoelectric point (pI) according to protein sequence.

^e Molecular mass (Mr) and isoelectric point (pI) based on the calculation using software Progenesis SameSpots

Sarcomere organization and muscle contraction

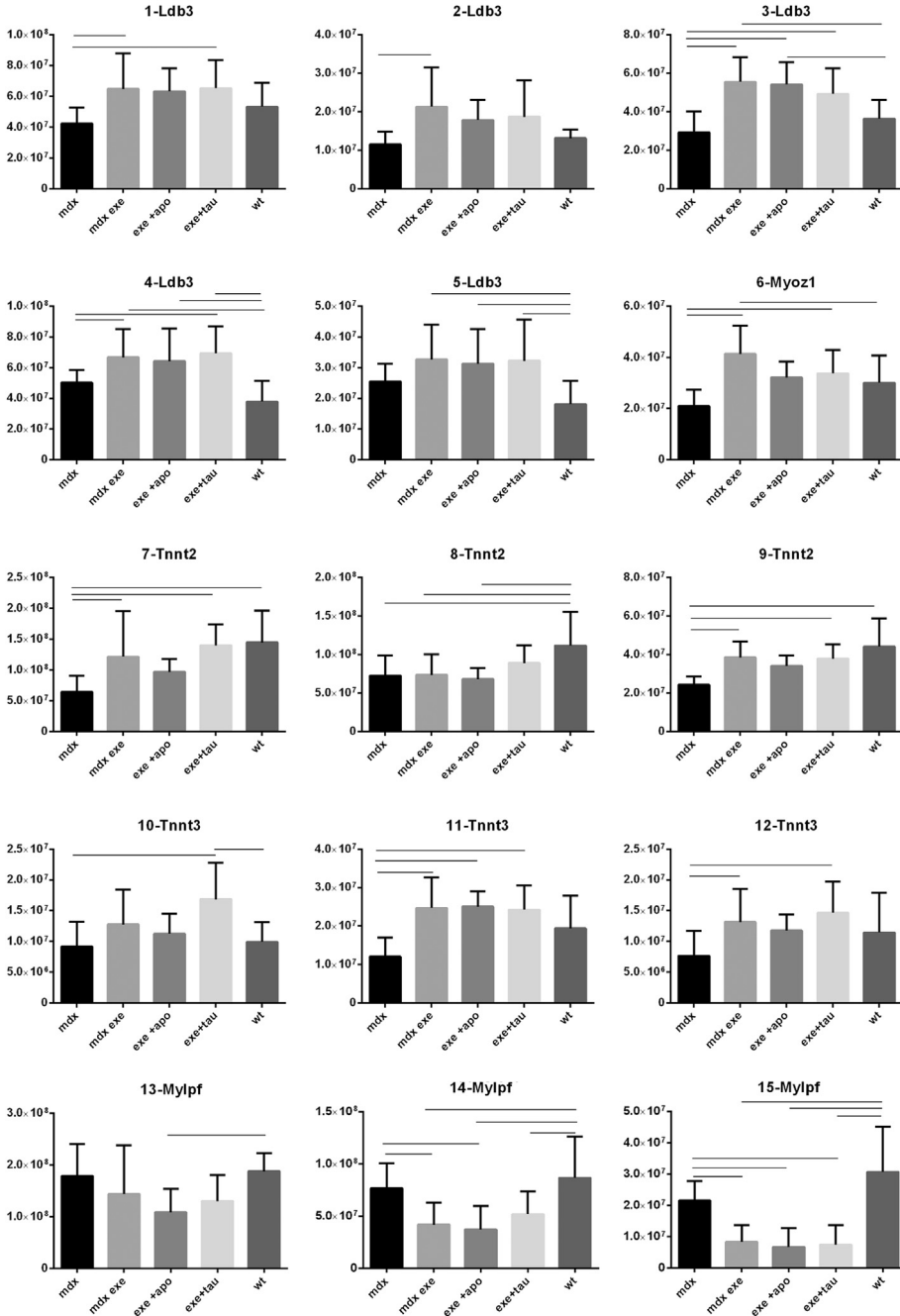


Fig. 1. Histograms represent the abundance of each spot (normalized volume, arbitrary units) in all groups studied, namely mdx, mdx exe, mdx exe apo, mdx exe tau (indicated as mdx+apo and mdx+tau respectively) and wt, evaluated with Progenesis SameSpot software. All spots show a False Discovery Rate (FDR) ≤ 0.05 . The significant differences between groups were calculated with GraphPad Prism v6.0 software, using Tukey correction for multiple comparison. Significant differences between groups are indicated by a line.

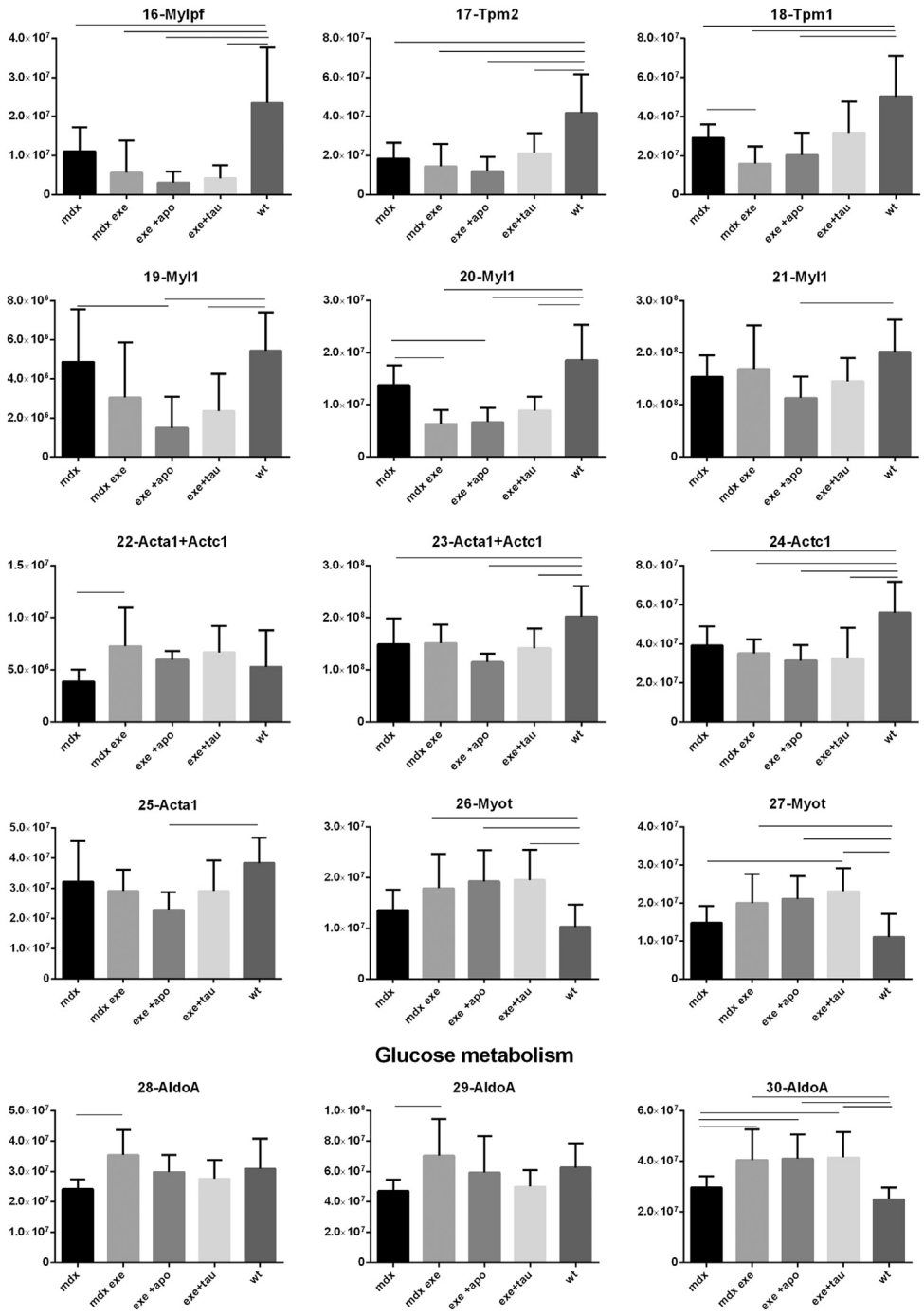


Fig. 1. (continued)

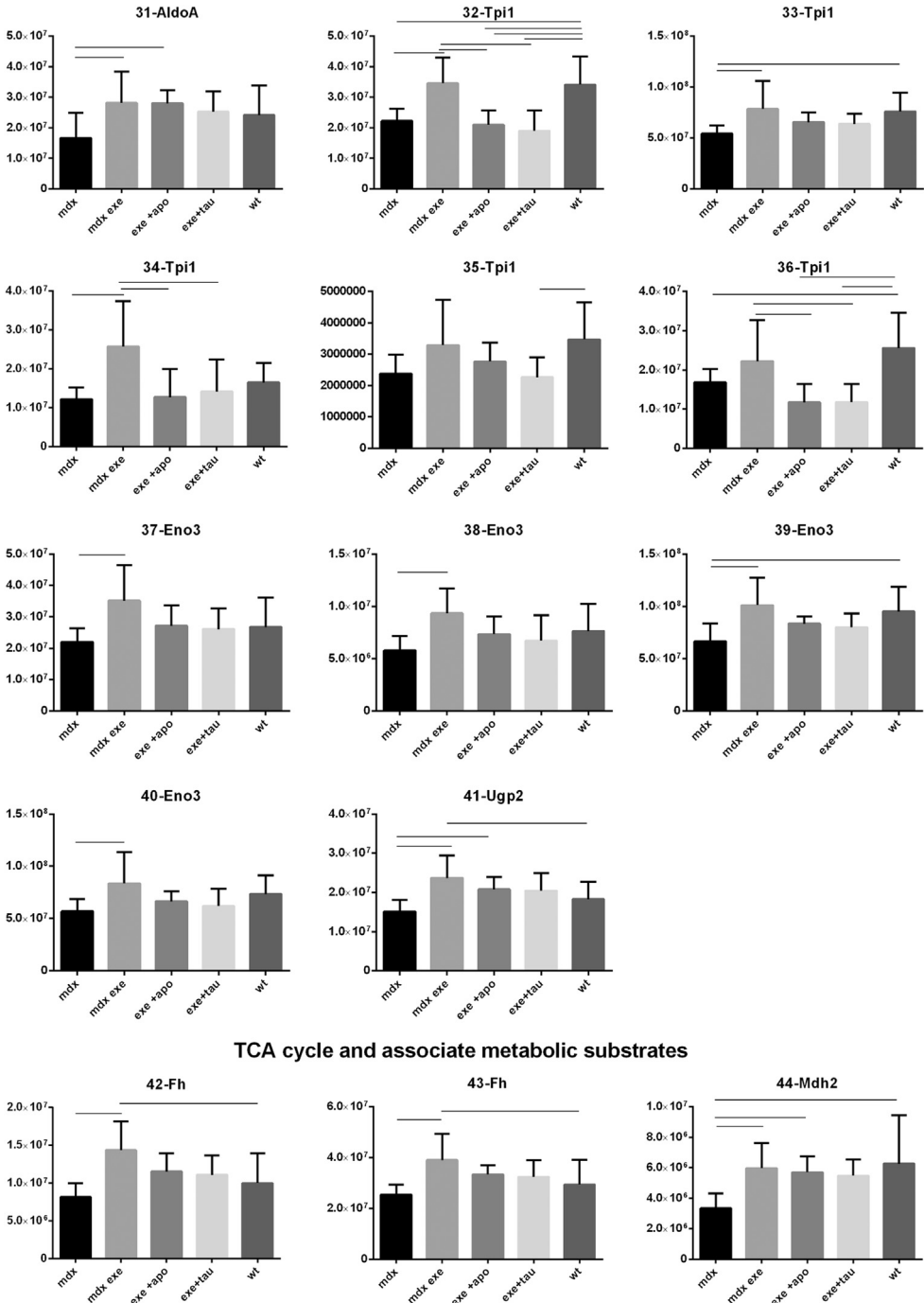


Fig. 1. (continued)

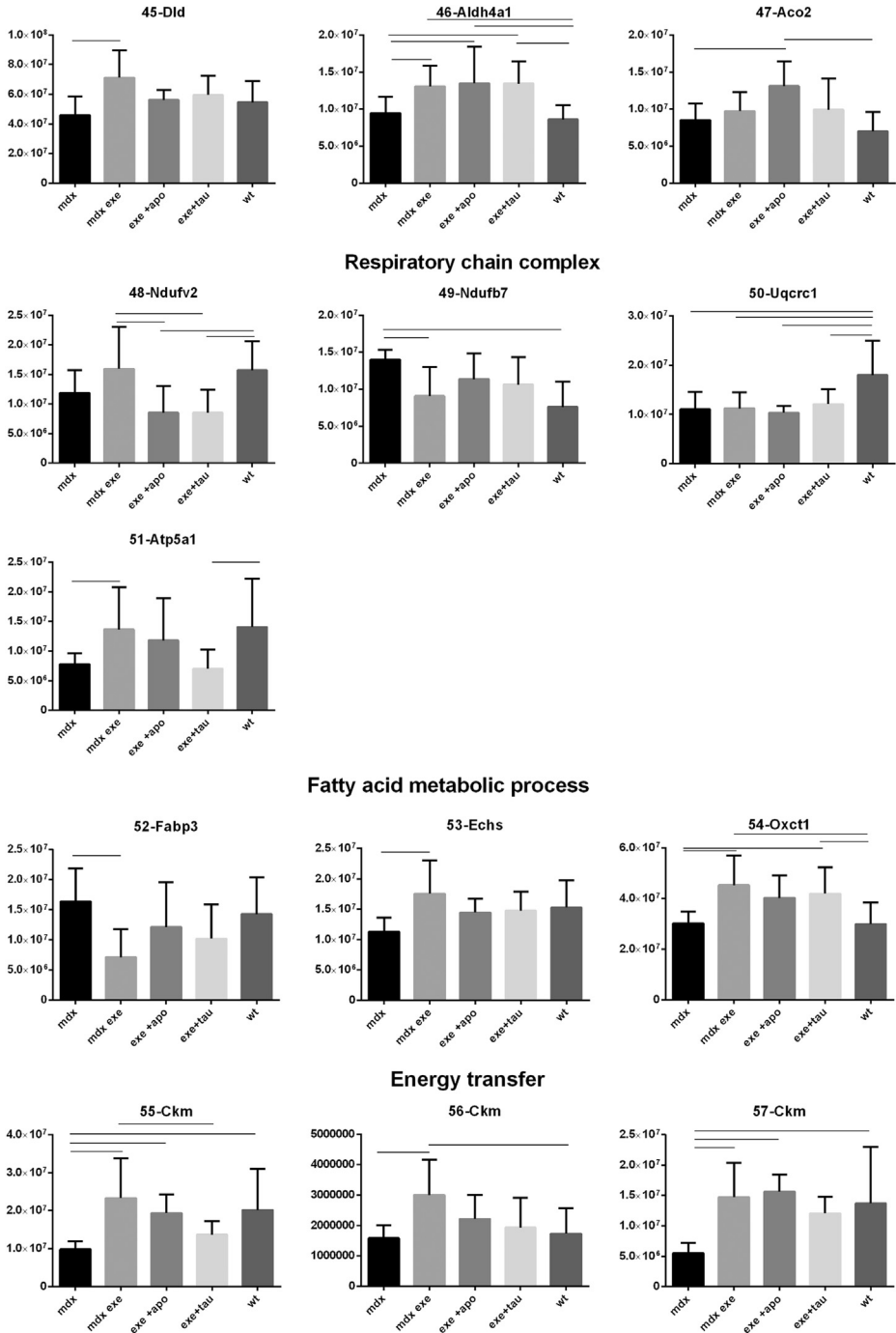


Fig. 1. (continued)

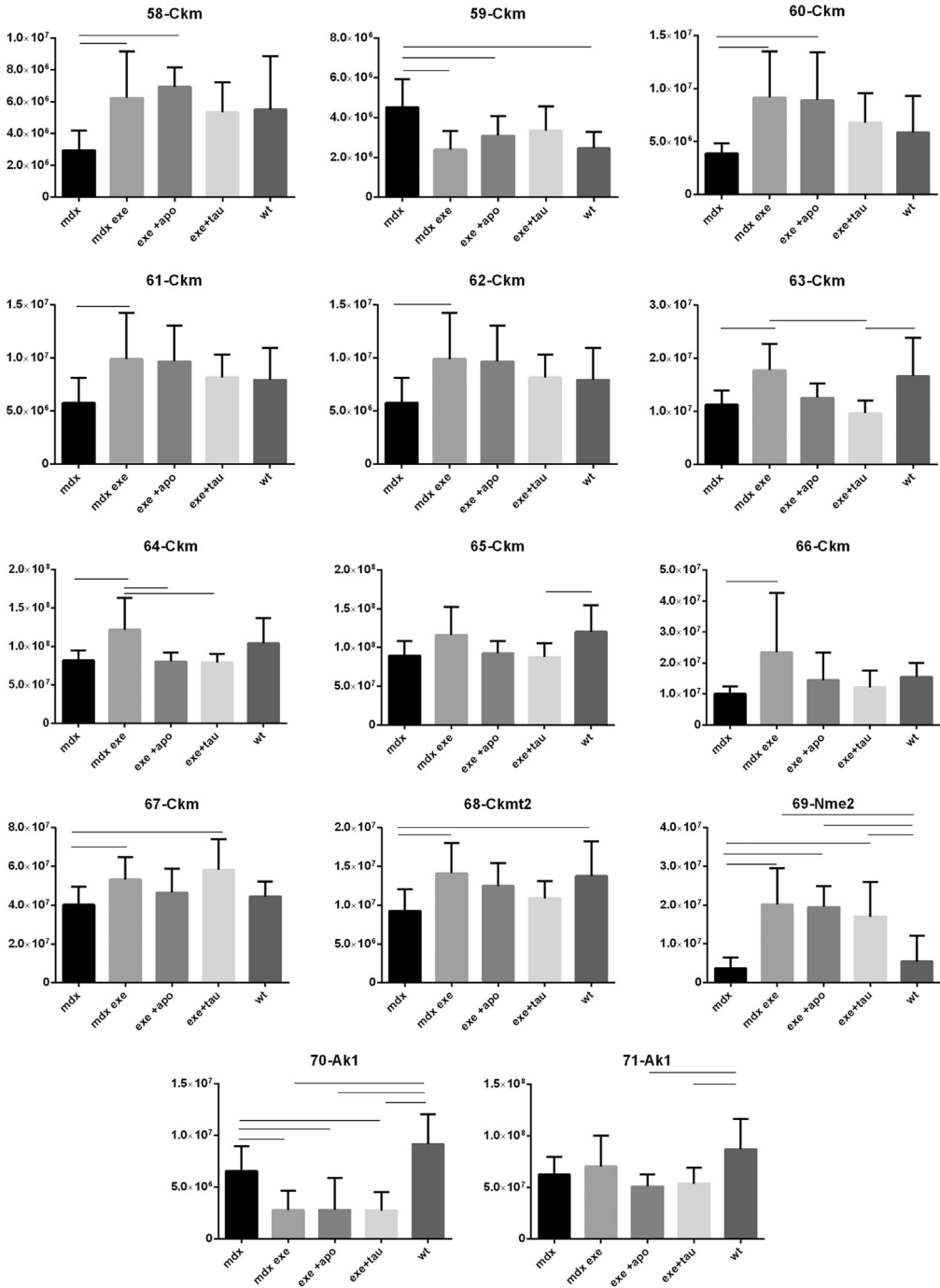


Fig. 1. (continued)

Other metabolic process

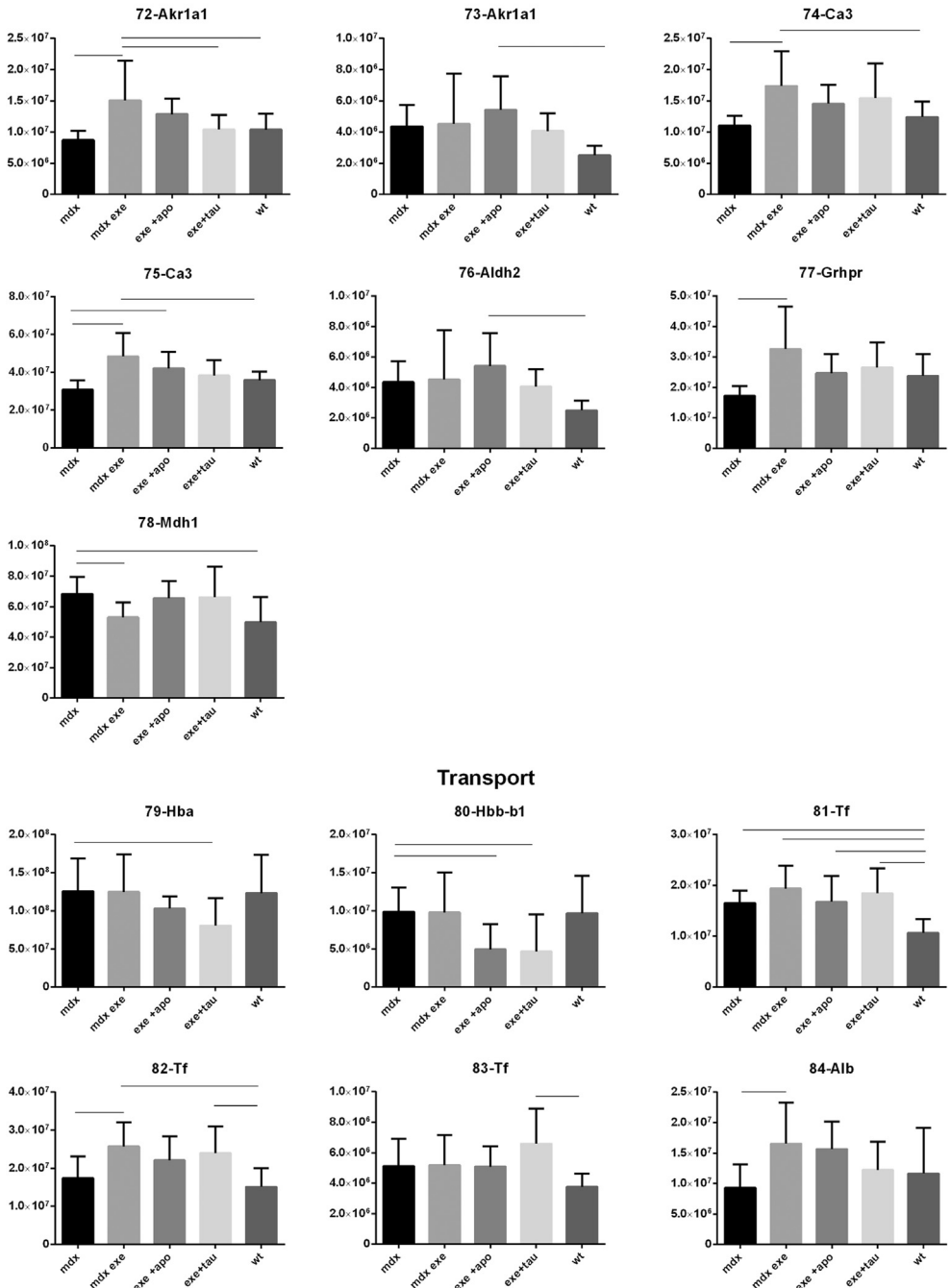


Fig. 1. (continued)

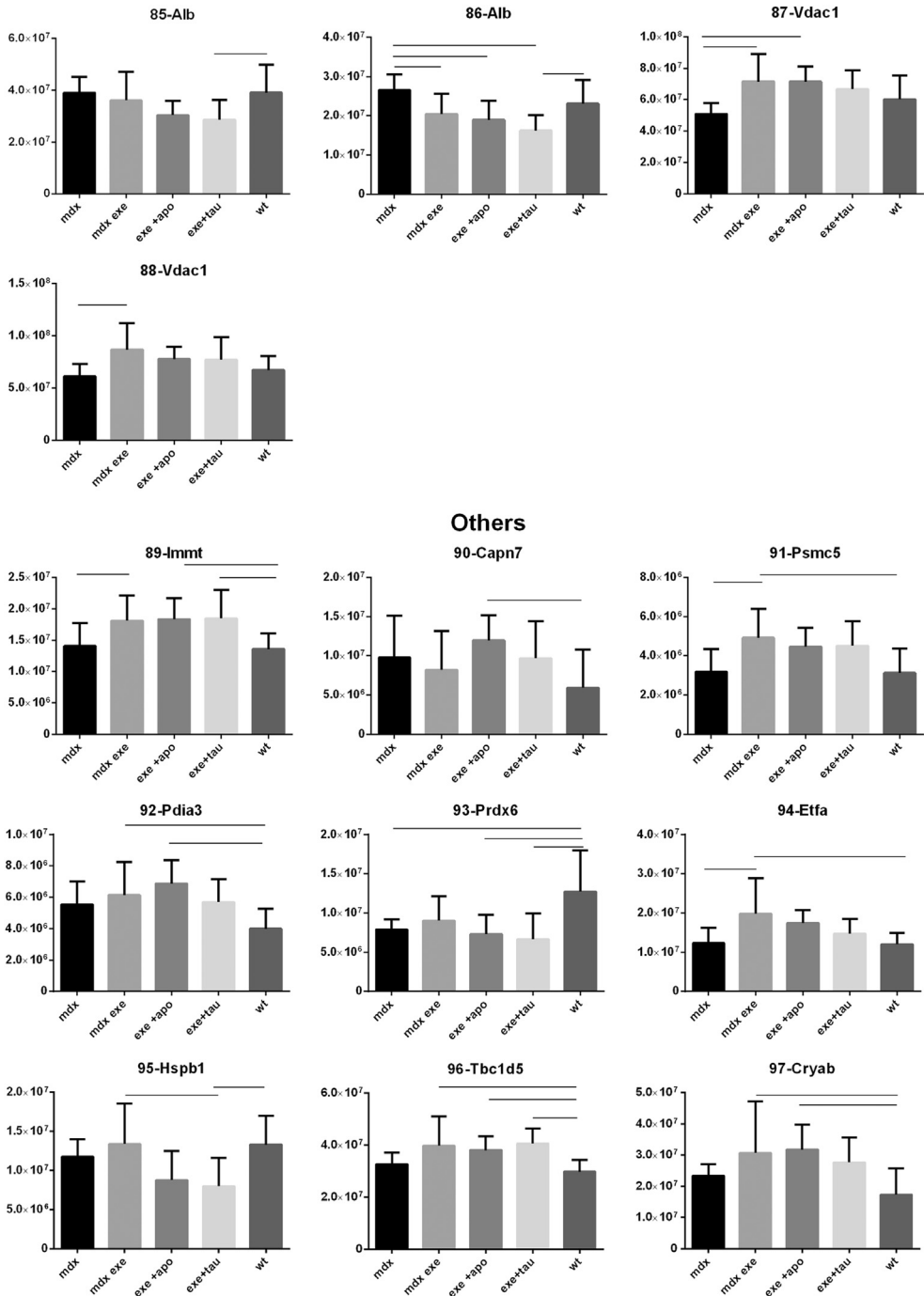


Fig. 1. (continued)

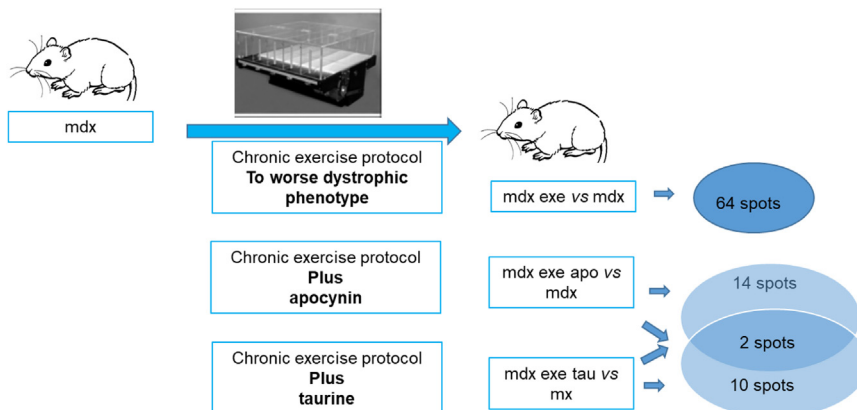


Fig. 2. Picture representing different abundant spots between mdx and mdx exe treated and untreated with compounds. Detailed data on spot differences were reported in table 4 of ref [1].

1. Data

1.1. MS data

97 differentially abundant spots were identified through the study published in [1]. Among these, some spots showing low Mascot (PMF) score value or discrepancy between theoretical and calculated MW or pI, were further analyzed performing peptide sequencing by tandem mass spectrometry. MS/MS analysis was carried out by using an Ultraflex III MALDI-TOF/TOF mass spectrometer (Bruker Daltonics) as described in Materials and Methods, and Table 1 reports detailed MALDI-TOF/TOF data. 12 spots show an experimental Mr different from expected. The sequence coverage of these spots is reported in Table 2. The muscle protein LIM domain-binding protein 3 (LDB3) was found in three different spots showing a Mr lower than expected. This protein belongs to Z-disc proteins whose alteration was correlated with myofibrillar myopathies [2]. Creatin kinase (Ckm) was found in six spots showing a Mr lower than expected.

1.2. Apocynin and taurine modulate the effect of exercise on mdx mice muscle protein abundance

Fig. 1 reports 97 histograms representing the spot abundance, in each group analysed (mdx, mdx exe, mdx exe tau, mdx exe apo) evaluated by gel image analysis with ProgenesisSame Spot. Proteins are divided in categories according to their GO biological process. Protein spot abundance in wt mice was also evaluated as referring phenotype. Fig. 2 summarizes the modulatory effects of taurine and apocynin.

1.3. Comparison with wt strain

Table 3 reports differentially abundant protein spots and relative fold changes, between mdx exe vs wt and mdx vs wt tibialis anterior muscles. In Fig. 3a diagram represents the relationships between these three groups. The protocol used for mdx training consisted of a 30 min running on a horizontal treadmill (Columbus Instruments, USA) at 12 m/min, twice a week for at least 4 weeks. This protocol causes significant weakness in the limb strength as measured by a grip strength meter [3]. The *in vivo* weakness produced by such a protocol is observed exclusively in mdx mice with no similar effects in wild type mice [4,5]. In fact, protocols used to induce training effects in wild types mice usually consist of continuous running at 20 m/min for at least 15 min using a treadmill slope of 10°, five days a week, for eight weeks [6]. To exclude training effects in wt animals we checked the amount of selected proteins in wt animals subjected to the same exercise protocol of mdx mice. In particular, we

Table 3

Differentially abundant protein spots between mdx exe vs wt and mdx vs wt tibialis anterior muscles.

Spot No	Protein name	^a fold change mdx vs wt	^a fold change mdx exe vs wt
<i>Sarcomere structure and muscle contraction</i>			
3	LIM domain-binding protein 3	ns	1.5
4	LIM domain-binding protein 3	ns	1.7
5	LIM domain-binding protein 3	ns	1.8
6	Myozenin-1	ns	1.4
7	Troponin I, fast skeletal muscle	-2.2	ns
8	Troponin I, fast skeletal muscle	-1.6	-1.5
9	Troponin I, fast skeletal muscle	-1.8	ns
14	Myosin regulatory light chain 2, skeletal muscle isoform	ns	-2.1
15	Myosin regulatory light chain 2, skeletal muscle isoform	ns	-3.7
16	Myosin regulatory light chain 2, skeletal muscle isoform	-2.1	-4.1
17	Tropomyosin beta chain	-2.3	-2.8
18	Tropomyosin alpha-1 chain	-1.8	-2.8
20	Myosin light chain 1/3, skeletal muscle isoform	ns	-2.9
23	Actin, alpha skeletal muscle and Actin, alpha cardiac muscle1	-1.4	ns
24	Actin, alpha cardiac muscle 1	-1.4	-1.6
26	Myotilin	ns	1.7
27	Myotilin	ns	1.8
<i>Metabolism and energy transfer</i>			
30	Fructose-bisphosphate aldolase A	ns	1.6
32	Triosephosphate isomerase	-1.53	ns
33	Triosephosphate isomerase	-1.4	ns
36	Triosephosphate isomerase	-1.52	ns
39	Beta-enolase	-1.4	ns
41	UTP–glucose-1-phosphate uridylyltransferase	ns	1.3
42	Fumarate hydratase, mitochondrial	ns	1.4
43	Fumarate hydratase, mitochondrial	ns	1.3
44	Malate dehydrogenase, mitochondrial	-1.8	ns
46	Delta-1-pyrroline-5-carboxylate dehydrogenase, mitochondrial	ns	1.5
49	NADH dehydrogenase [ubiquinone] 1 beta subcomplex subunit 7	1.8	ns
50	Cytochrome b-c1 complex subunit 1, mitochondrial	-1.6	-1.6
54	Succinyl-CoA:3-ketoacid coenzyme A transferase 1, mitochondrial	ns	1.5
55	Creatine kinase M-type	-2	ns
56	Creatine kinase M-type	ns	1.7
57	Creatine kinase M-type	-2.5	ns
59	Creatine kinase M-type	1.8	ns
68	Creatine kinase M-type	-1.5	ns
69	Nucleoside diphosphate kinase B	ns	3.7
70	Adenylate kinase isoenzyme 1	ns	-2.6
<i>Others</i>			
72	Alcohol dehydrogenase [NADP(+)]	ns	1.4

Table 3 (continued)

Spot No	Protein name	^a fold change mdx vs wt	^a fold change mdx exe vs wt
74	Carbonic anhydrase 3	ns	1.4
75	Carbonic anhydrase 3	ns	1.3
78	Malate dehydrogenase, cytoplasmic	1.4	ns
81	Serotransferrin	1.5	1.8
82	Serotransferrin	ns	1.7
91	26S protease regulatory subunit 8	ns	1.6
92	Protein disulfide-isomerase A3	ns	1.5
93	Peroxiredoxin-6	-1.6	ns
94	Electron transfer flavoprotein sub- unit alpha, mitochondrial	ns	1.4
96	TBC1 domain family member 5	ns	1.3
97	Alpha-crystallin B chain	ns	1.8

^a Fold change was calculated dividing the average of %V of mdx or mdx exe by the average of %V of wt (V = volume = integration of the optical density over the spot area; %V = V single spot/V total spots included in the reference gel).

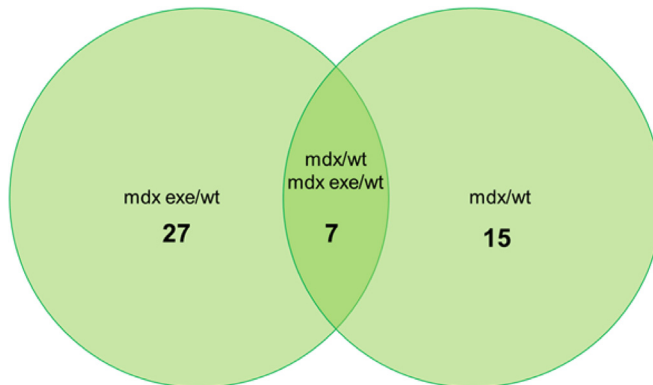


Fig. 3. Diagram representing the distribution of differences in spot abundance between groups: 27 protein spots differ exclusively between mdx exe and wt, 15 protein spots differ exclusively between mdx and wt and 7 spots are different from wt in both mdx and mdx exe.

analysed by western blot the amount of several proteins of glycolysis (all increased in mdx exe mice), oxophos proteins, and PGC-1-alpha and Sirt1 proteins. As shown in Fig. 4 none difference is observed in the expression level of these proteins.

2. Experimental design, materials and methods

The methodologies that allowed the data here presented are described in [1] and in cited references. Here, only the protocol for MS/MS data is described.

Trypsin digests of some spots with low Mascot (PMF) score value or with discrepancy between theoretical and calculated MW or pI were further analyzed performing peptide sequencing by tandem mass spectrometry. MS/MS analysis was performed by using an Ultraflex III MALDI-TOF/TOF mass spectrometer (Bruker Daltonics). Two to four PMF peaks showing a high intensity were CID fragmented using Argon as collision gas, and MALDI-TOF/TOF tandem MS was performed in LIFT mode by software controlled data acquisition. Fragmented ions were analyzed using the Flex Analysis software v.3.0. The MS/MS database searching was carried out in the UniProtKB database using the on-line available MASCOT MS/MS ion search software. The following parameters were applied for database

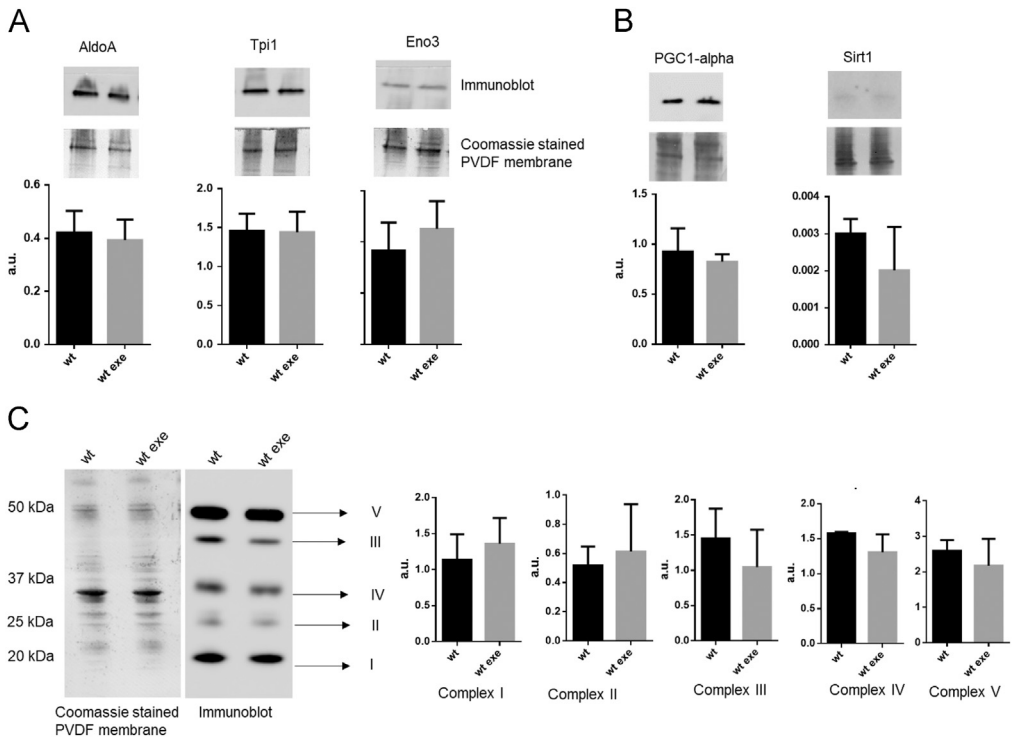


Fig. 4. Histograms and representative immunoblot images of glycolytic enzymes: AldoA, Tpi1 and Eno3 (panel A); PGC1-alpha and Sirt1 (panel B) and Oxphos complexes from wt and wt exe mice. (n=5; mean \pm S.D.; t-test unpaired). Normalization of immunoblot was performed on Coomassie stained gel.

searching: taxonomy: *Mus musculus*, trypsin specificity, one missed cleavage allowed, peptide precursor mass tolerance: ± 100 ppm, fragment mass tolerance: ± 0.6 Da, peptide precursor charge state: +1, carbamidomethylation of cysteine as a fixed modification, oxidation of methionine as a possible modification. For protein identification, Mascot ion score, peptide coverage by “b” and “y” ions, and expected value were considered. We considered as significant, peptides with individual ion scores $-10 * \text{Log}[P]$, where P is the probability that the observed match is a random event, that indicated identity ($p < 0.05$).

Acknowledgements

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Transparency document. Supporting information

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.dib.2018.03.037>.

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