

ADDITIONAL FILE 4

Table S3. Gene microarray analyses of EDL muscles from WT and CASQ1-null mice reveals significant up-regulation of genes involved in autophagy/atrophy signaling.

<i>GB_accession</i>	<i>Gene_Symbol</i>	<i>Description</i>	<i>Fold Change</i> ^A
NM 026346	atrogen-1/MAFbx	E3 ubiquitin ligase	1,82445753
AK010596	Psmc1	proteasome (prosome, macropain) 26S subunit, non-ATPase, 1	1,63895746
X06086	Ctsl	Cathepsin L	1,46843157
AF041054	Bnip	BCL2/adenovirus E1B 19 kDa-interacting protein 1, NIP3	1,61282553

Microarray analyses revealed significant up-regulation of 4 atrogenes involved in autophagy/atrophy signalling. Cathepsin L is a lysosomal enzyme involved in the degradation of membrane proteins that is upregulated during skeletal muscle atrophy [1]; Bnip3 regulates autophagy by inducing mitochondrial damage and removal via autophagosomes (mitophagy) [2]. Atrogen1/MAFbx is an ubiquitin ligase that contributes to muscle atrophy in a variety of catabolic states [3]. Psmc1 is a regulatory subunit of the proteasome that is involved in the ATP-dependent degradation of ubiquitinated proteins [4]. The up-regulation of these genes in EDL muscle from CASQ1-null mice reflects activation of the proteosomal and autophagic pathways during muscle atrophy.

SUPPLEMENTAL REFERENCES

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3. Li YP, Chen Y, John J, Moylan J, Jin B, Mann DL et al. TNF-alpha acts via p38 MAPK to stimulate expression of the ubiquitin ligase atrogin1/MAFbx in skeletal muscle. *Faseb J.* 2005;19(3):362-70. doi:19/3/362 [pii] 10.1096/fj.04-2364com.
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