Shenhav Cohen

List of Publications by Year
in descending order

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21 papers	2,520 citations	623734 14 h-index	794594 19 g-index
25 all docs	25 docs citations	25 times ranked	4775 citing authors

#	Article	IF	Citations
1	New roles for desmin in the maintenance of muscle homeostasis. FEBS Journal, 2022, 289, 2755-2770.	4.7	50
2	Breakdown of Filamentous Myofibrils by the UPSâ€“Step by Step. Biomolecules, 2021, 11, 110.	4.0	13
3	A semiautomated measurement of muscle fiber size using the Imaris software. American Journal of Physiology - Cell Physiology, 2021, 321, C615-C631.	4.6	17
4	Reply to Kissane and Eggington. American Journal of Physiology - Cell Physiology, 2021, 321, C1084-C1085.	4.6	0
5	A signaling hub of insulin receptor, dystrophin glycoprotein complex and plakoglobin regulates muscle size. Nature Communications, 2020, 11, 1381.	12.8	33
6	<scp>USP</scp> 1 deubiquitinates Akt to inhibit <scp>PI</scp> 3Kâ€Aktâ€FoxO signaling in muscle during prolonged starvation. EMBO Reports, 2020, 21, e48791.	4.5	64
7	Role of calpains in promoting desmin filaments depolymerization and muscle atrophy. Biochimica Et Biophysica Acta - Molecular Cell Research, 2020, 1867, 118788.	4.1	24
8	Profiling of the muscle-specific dystroglycan interactome reveals the role of Hippo signaling in muscular dystrophy and age-dependent muscle atrophy. BMC Medicine, 2020, 18, 8.	5.5	20
9	Non-canonical activation of DAPK2 by AMPK constitutes a new pathway linking metabolic stress to autophagy. Nature Communications, 2018, 9, 1759.	12.8	33
10	GSK3-Î² promotes calpain-1â€“mediated desmin filament depolymerization and myofibril loss in atrophy. Journal of Cell Biology, 2018, 217, 3698-3714.	5.2	58
11	Myofibril breakdown during atrophy is a delayed response requiring the transcription factor PAX4 and desmin depolymerization. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E1375-E1384.	7.1	50
12	The extracellular matrix protein agrin promotes heart regeneration in mice. Nature, 2017, 547, 179-184.	27.8	498
13	Muscle wasting in disease: molecular mechanisms and promising therapies. Nature Reviews Drug Discovery, 2015, 14, 58-74.	46.4	792
14	VWA domain of S5a restricts the ability to bind ubiquitin and Ubl to the 26S proteasome. Molecular Biology of the Cell, 2014, 25, 3988-3998.	2.1	15
15	Trim32 reduces PI3Kâ€“Aktâ€“FoxO signaling in muscle atrophy by promoting plakoglobinâ€“PI3K dissociation. Journal of Cell Biology, 2014, 204, 747-758.	5.2	82
16	Ubiquitylation by Trim32 causes coupled loss of desmin, Z-bands, and thin filaments in muscle atrophy. Journal of Cell Biology, 2012, 198, 575-589.	5.2	165
17	During muscle atrophy, thick, but not thin, filament components are degraded by MuRF1-dependent ubiquitylation. Journal of Cell Biology, 2009, 185, 1083-1095.	5.2	499
18	During muscle atrophy, thick, but not thin, filament components are degraded by MuRF1-dependent ubiquitylation. Journal of Experimental Medicine, 2009, 206, i13-i13.	8.5	0

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19	JAKâ€“STAT signaling involved in phorbol 12-myristate 13-acetate- and dimethyl sulfoxide-induced 2â€²-5â€² oligoadenylate synthetase expression in human HL-60 leukemia cells. Leukemia Research, 2005, 29, 923-931.	0.8	12
20	The trans-Golgi network-associated human ubiquitin-protein ligase POSH is essential for HIV type 1 production. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 1478-1483.	7.1	71
21	Expression of a PKR Dominant-Negative Mutant in Myogenic Cells Interferes with the Myogenic Process. Experimental Cell Research, 2000, 254, 45-54.	2.6	24