

Junichi Matsumoto

List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/5450005/publications.pdf>

Version: 2024-02-01

10
papers

304
citations

1040056

9
h-index

1372567

10
g-index

10
all docs

10
docs citations

10
times ranked

462
citing authors

#	ARTICLE	IF	CITATIONS
1	Inhibition of xanthine oxidase in the acute phase of myocardial infarction prevents skeletal muscle abnormalities and exercise intolerance. Cardiovascular Research, 2021, 117, 805-819.	3.8	25
2	Cardiac-specific loss of mitoNEET expression is linked with age-related heart failure. Communications Biology, 2021, 4, 138.	4.4	20
3	Brain-Derived Neurotrophic Factor Improves Impaired Fatty Acid Oxidation Via the Activation of Adenosine Monophosphate-Activated Protein Kinase- ϵ Proliferator-Activated Receptor- α Coactivator-1 β Signaling in Skeletal Muscle of Mice With Heart Failure. Circulation: Heart Failure, 2021, 14, e005890.	3.9	18
4	Empagliflozin restores lowered exercise endurance capacity via the activation of skeletal muscle fatty acid oxidation in a murine model of heart failure. European Journal of Pharmacology, 2020, 866, 172810.	3.5	43
5	Angiotensin-converting-enzyme inhibitor prevents skeletal muscle fibrosis in myocardial infarction mice. Skeletal Muscle, 2020, 10, 11.	4.2	10
6	Brain-Derived Neurotrophic Factor Improves Limited Exercise Capacity in Mice With Heart Failure. Circulation, 2018, 138, 2064-2066.	1.6	32
7	Protein acetylation in skeletal muscle mitochondria is involved in impaired fatty acid oxidation and exercise intolerance in heart failure. Journal of Cachexia, Sarcopenia and Muscle, 2018, 9, 844-859.	7.3	46
8	Deletion of NAD(P)H Oxidase 2 Prevents Angiotensin II-Induced Skeletal Muscle Atrophy. BioMed Research International, 2018, 2018, 1-10.	1.9	13
9	Dipeptidyl peptidase-4 inhibitor improved exercise capacity and mitochondrial biogenesis in mice with heart failure via activation of glucagon-like peptide-1 receptor signalling. Cardiovascular Research, 2016, 111, 338-347.	3.8	64
10	The experimental model of transition from compensated cardiac hypertrophy to failure created by transverse aortic constriction in mice. IJC Heart and Vasculature, 2016, 11, 24-28.	1.1	33