

Saverio Francesco Retta

List of Publications by Year in descending order

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77
papers

3,943
citations

109321

35
h-index

123424

61
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83
all docs

83
docs citations

83
times ranked

4861
citing authors

#	ARTICLE	IF	CITATIONS
1	Next-Generation Sequencing Advances the Genetic Diagnosis of Cerebral Cavernous Malformation (CCM). <i>Antioxidants</i> , 2022, 11, 1294.	5.1	7
2	Towards precision nanomedicine for cerebrovascular diseases with emphasis on Cerebral Cavernous Malformation (CCM). <i>Expert Opinion on Drug Delivery</i> , 2021, 18, 849-876.	5.0	10
3	Protein Kinase C α (PKC α) Regulates the Nucleocytoplasmic Shuttling of KRIT1. <i>FASEB Journal</i> , 2021, 35, .	0.5	0
4	An international call for a new grading system for cerebral and cerebellar cavernomas. <i>Journal of Neurosurgical Sciences</i> , 2021, 65, 239-246.	0.6	5
5	Polymorphisms in genes related to oxidative stress and inflammation: Emerging links with the pathogenesis and severity of Cerebral Cavernous Malformation disease. <i>Free Radical Biology and Medicine</i> , 2021, 172, 403-417.	2.9	22
6	Protein kinase C α (PKC α) regulates the nucleocytoplasmic shuttling of KRIT1. <i>Journal of Cell Science</i> , 2021, 134, .	2.0	8
7	Intracellular Antioxidant Activity of Biocompatible Citrate-Capped Palladium Nanozymes. <i>Nanomaterials</i> , 2020, 10, 99.	4.1	36
8	KRIT1 loss-mediated upregulation of NOX1 in stromal cells promotes paracrine pro-angiogenic responses. <i>Cellular Signalling</i> , 2020, 68, 109527.	3.6	15
9	Dicarbonyl Stress and S-Glutathionylation in Cerebrovascular Diseases: A Focus on Cerebral Cavernous Malformations. <i>Antioxidants</i> , 2020, 9, 124.	5.1	24
10	Vitamin D Deficiency and the Risk of Cerebrovascular Disease. <i>Antioxidants</i> , 2020, 9, 327.	5.1	55
11	From Genes and Mechanisms to Molecular-Targeted Therapies: The Long Climb to the Cure of Cerebral Cavernous Malformation (CCM) Disease. <i>Methods in Molecular Biology</i> , 2020, 2152, 3-25.	0.9	12
12	Next Generation Sequencing (NGS) Strategies for Genetic Testing of Cerebral Cavernous Malformation (CCM) Disease. <i>Methods in Molecular Biology</i> , 2020, 2152, 59-75.	0.9	2
13	Study of CCM Microvascular Endothelial Phenotype by an In Vitro Tubule Differentiation Model. <i>Methods in Molecular Biology</i> , 2020, 2152, 371-375.	0.9	0
14	Detection of p62/SQSTM1 Aggregates in Cellular Models of CCM Disease by Immunofluorescence. <i>Methods in Molecular Biology</i> , 2020, 2152, 417-426.	0.9	0
15	Spectrophotometric Method for Determining Glyoxalase 1 Activity in Cerebral Cavernous Malformation (CCM) Disease. <i>Methods in Molecular Biology</i> , 2020, 2152, 445-449.	0.9	0
16	Production of KRIT1-knockout and KRIT1-knockin Mouse Embryonic Fibroblasts as Cellular Models of CCM Disease. <i>Methods in Molecular Biology</i> , 2020, 2152, 151-167.	0.9	3
17	Generation of CCM Phenotype by a Human Microvascular Endothelial Model. <i>Methods in Molecular Biology</i> , 2020, 2152, 131-137.	0.9	0
18	Fluorescence Analysis of Reactive Oxygen Species (ROS) in Cellular Models of Cerebral Cavernous Malformation Disease. <i>Methods in Molecular Biology</i> , 2020, 2152, 451-465.	0.9	1

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19	KRIT1 Deficiency Promotes Aortic Endothelial Dysfunction. <i>International Journal of Molecular Sciences</i> , 2019, 20, 4930.	4.1	24
20	KRIT1 Loss-Of-Function Associated with Cerebral Cavemous Malformation Disease Leads to Enhanced S-Glutathionylation of Distinct Structural and Regulatory Proteins. <i>Antioxidants</i> , 2019, 8, 27.	5.1	39
21	Data in support of sustained upregulation of adaptive redox homeostasis mechanisms caused by KRIT1 loss-of-function. <i>Data in Brief</i> , 2018, 16, 929-938.	1.0	37
22	KRIT1 loss-of-function induces a chronic Nrf2-mediated adaptive homeostasis that sensitizes cells to oxidative stress: Implication for Cerebral Cavemous Malformation disease. <i>Free Radical Biology and Medicine</i> , 2018, 115, 202-218.	2.9	69
23	Multifunctional Platinum@BSA-Rapamycin Nanocarriers for the Combinatorial Therapy of Cerebral Cavemous Malformation. <i>ACS Omega</i> , 2018, 3, 15389-15398.	3.5	31
24	Biological Activities, Health Benefits, and Therapeutic Properties of Avenanthramides: From Skin Protection to Prevention and Treatment of Cerebrovascular Diseases. <i>Oxidative Medicine and Cellular Longevity</i> , 2018, 2018, 1-17.	4.0	60
25	Yeast-Derived Recombinant Avenanthramides Inhibit Proliferation, Migration and Epithelial Mesenchymal Transition of Colon Cancer Cells. <i>Nutrients</i> , 2018, 10, 1159.	4.1	14
26	Krit1 loss-of-function increases TNF- α -induced apoptosis by inhibiting Notch1 in endothelial cells. <i>Journal of Molecular and Cellular Cardiology</i> , 2018, 120, 48.	1.9	0
27	Up-regulation of NADPH oxidase-mediated redox signaling contributes to the loss of barrier function in KRIT1 deficient endothelium. <i>Scientific Reports</i> , 2017, 7, 8296.	3.3	51
28	Loss of KRIT1 causes a sustained activation of an adaptive cellular allostatic response that counteracts intrinsic oxidative stress but sensitizes cells to further oxidative challenges. <i>Free Radical Biology and Medicine</i> , 2017, 108, S20.	2.9	0
29	Beyond multiple mechanisms and a unique drug: Defective autophagy as pivotal player in cerebral cavemous malformation pathogenesis and implications for targeted therapies. <i>Rare Diseases (Austin)</i> , Tj ETQq1 1 0.784314 zGBT /Over	1.7	0
30	Oxidative stress and inflammation in cerebral cavemous malformation disease pathogenesis: Two sides of the same coin. <i>International Journal of Biochemistry and Cell Biology</i> , 2016, 81, 254-270.	2.8	80
31	Cytochrome P450 and matrix metalloproteinase genetic modifiers of disease severity in Cerebral Cavemous Malformation type 1. <i>Free Radical Biology and Medicine</i> , 2016, 92, 100-109.	2.9	47
32	Platinum nanozymes recover cellular ROS homeostasis in an oxidative stress-mediated disease model. <i>Nanoscale</i> , 2016, 8, 3739-3752.	5.6	203
33	Cellular processes underlying cerebral cavemous malformations: Autophagy as another point of view. <i>Autophagy</i> , 2016, 12, 424-425.	9.1	25
34	The Role of Oxidative Stress in Cerebral Cavemous Malformation (CCM) Pathogenesis: From Disease Mechanisms toward Therapeutic Approaches. <i>Free Radical Biology and Medicine</i> , 2015, 87, S56.	2.9	0
35	Defective autophagy is a key feature of cerebral cavemous malformations. <i>EMBO Molecular Medicine</i> , 2015, 7, 1403-1417.	6.9	109
36	<i>Yersinia enterocolitica</i> exploits different pathways to accomplish adhesion and toxin injection into host cells. <i>Cellular Microbiology</i> , 2015, 17, 1179-1204.	2.1	30

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37	Evaluation of the bioactive properties of avenanthramide analogs produced in recombinant yeast. <i>BioFactors</i> , 2015, 41, 15-27.	5.4	36
38	Strategy for Identifying Repurposed Drugs for the Treatment of Cerebral Cavernous Malformation. <i>Circulation</i> , 2015, 131, 289-299.	1.6	149
39	Cerebral cavernous malformation (CCM) disease: from monogenic forms to genetic susceptibility factors. <i>Journal of Neurosurgical Sciences</i> , 2015, 59, 201-9.	0.6	27
40	KRIT1 loss of function causes a ROS-dependent upregulation of c-Jun. <i>Free Radical Biology and Medicine</i> , 2014, 68, 134-147.	2.9	66
41	The Ras Superfamily of Small GTPases: The Unlocked Secrets. <i>Methods in Molecular Biology</i> , 2014, 1120, 1-18.	0.9	138
42	Fluorescence Microscopy Study of Rap1 Subcellular Localization. <i>Methods in Molecular Biology</i> , 2014, 1120, 197-205.	0.9	5
43	Ras GTPases Are Both Regulators and Effectors of Redox Agents. <i>Methods in Molecular Biology</i> , 2014, 1120, 55-74.	0.9	13
44	Combined Pulldown and Time-Lapse Microscopy Studies for Determining the Role of Rap1 in the Crosstalk Between Integrins and Cadherins. <i>Methods in Molecular Biology</i> , 2014, 1120, 177-195.	0.9	1
45	miR-21 coordinates tumor growth and modulates KRIT1 levels. <i>Biochemical and Biophysical Research Communications</i> , 2013, 438, 90-96.	2.1	27
46	Genetic and cellular basis of cerebral cavernous malformations: implications for clinical management. <i>Clinical Genetics</i> , 2013, 83, 7-14.	2.0	32
47	EndMT contributes to the onset and progression of cerebral cavernous malformations. <i>Nature</i> , 2013, 498, 492-496.	27.8	403
48	A <i>de novo</i> X;8 translocation creates a PTK2-THOC2 gene fusion with THOC2 expression knockdown in a patient with psychomotor retardation and congenital cerebellar hypoplasia. <i>Journal of Medical Genetics</i> , 2013, 50, 543-551.	3.2	42
49	The Interplay between ROS and Ras GTPases: Physiological and Pathological Implications. <i>Journal of Signal Transduction</i> , 2012, 2012, 1-9.	2.0	56
50	Reactive Oxygen Species: Friends and Foes of Signal Transduction. <i>Journal of Signal Transduction</i> , 2012, 2012, 1-1.	2.0	12
51	Molecular Crosstalk between Integrins and Cadherins: Do Reactive Oxygen Species Set the Talk?. <i>Journal of Signal Transduction</i> , 2012, 2012, 1-12.	2.0	55
52	Identification of the Kelch Family Protein Nd1-L as a Novel Molecular Interactor of KRIT1. <i>PLoS ONE</i> , 2012, 7, e44705.	2.5	28
53	Mutation Analysis of CCM1, CCM2 and CCM3 Genes in a Cohort of Italian Patients with Cerebral Cavernous Malformation. <i>Brain Pathology</i> , 2011, 21, 215-224.	4.1	52
54	A Unique Interplay Between Rap1 and E-Cadherin in the Endocytic Pathway Regulates Self-Renewal of Human Embryonic Stem Cells. <i>Stem Cells</i> , 2010, 28, 247-257.	3.2	82

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55	Production of novel antioxidative phenolic amides through heterologous expression of the plantâ€™s chlorogenic acid biosynthesis genes in yeast. <i>Metabolic Engineering</i> , 2010, 12, 223-232.	7.0	35
56	KRIT1 Regulates the Homeostasis of Intracellular Reactive Oxygen Species. <i>PLoS ONE</i> , 2010, 5, e11786.	2.5	106
57	<i>Helicobacter pylori</i> Type IV Secretion Apparatus Exploits Î²1 Integrin in a Novel RGD-Independent Manner. <i>PLoS Pathogens</i> , 2009, 5, e1000684.	4.7	203
58	Structural and functional differences between KRIT1A and KRIT1B isoforms: A framework for understanding CCM pathogenesis. <i>Experimental Cell Research</i> , 2009, 315, 285-303.	2.6	49
59	Inhibition of PI3K induces Rac Activation and Membrane Ruffling in Proto-Dbl Expressing Cells. <i>Cell Cycle</i> , 2006, 5, 2657-2665.	2.6	5
60	Rap1: A turnabout for the crosstalk between cadherins and integrins. <i>European Journal of Cell Biology</i> , 2006, 85, 283-293.	3.6	83
61	Nuclear Translocation of Integrin Cytoplasmic Domain-associated Protein 1 Stimulates Cellular Proliferation. <i>Molecular Biology of the Cell</i> , 2005, 16, 1859-1871.	2.1	35
62	E-cadherin endocytosis regulates the activity of Rap1: a traffic light GTPase at the crossroads between cadherin and integrin function. <i>Journal of Cell Science</i> , 2005, 118, 4765-4783.	2.0	157
63	Identification of Krit1B: a novel alternative splicing isoform of cerebral cavernous malformation gene-1. <i>Gene</i> , 2004, 325, 63-78.	2.2	18
64	Altered expression of integrins in RSV-transformed chick epiphyseal chondrocytes. <i>Biochimie</i> , 2003, 85, 483-492.	2.6	2
65	Disruption of Focal Adhesions by Integrin Cytoplasmic Domain-associated Protein-1Î±. <i>Journal of Biological Chemistry</i> , 2003, 278, 6567-6574.	3.4	79
66	Integrin-induced Epidermal Growth Factor (EGF) Receptor Activation Requires c-Src and p130Cas and Leads to Phosphorylation of Specific EGF Receptor Tyrosines. <i>Journal of Biological Chemistry</i> , 2002, 277, 9405-9414.	3.4	330
67	The integrin cytoplasmic domain-associated protein ICAP-1 binds and regulates Rho family GTPases during cell spreading. <i>Journal of Cell Biology</i> , 2002, 156, 377-388.	5.2	58
68	Cross Talk between Î²1 and Î±V Integrins: Î²1 Affects Î²3 mRNA Stability. <i>Molecular Biology of the Cell</i> , 2001, 12, 3126-3138.	2.1	58
69	Adhesion to Matrix Proteins. , 1999, 96, 125-130.		10
70	Purification of Fibronectin from Human Plasma. , 1999, 96, 119-124.		11
71	The Muscle-Specific Laminin Receptor Î±7Î²1 Integrin Negatively Regulates Î±5Î²1 Fibronectin Receptor Function. <i>Experimental Cell Research</i> , 1999, 246, 421-432.	2.6	27
72	Î±7B Integrin interferes with matrix assembly but not with confluent monolayer polarity, and alters some morphogenetic properties of FRT epithelial cells. <i>European Journal of Cell Biology</i> , 1998, 75, 107-117.	3.6	12

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73	Î²1D Integrin Inhibits Cell Cycle Progression in Normal Myoblasts and Fibroblasts. Journal of Biological Chemistry, 1998, 273, 15234-15240.	3.4	53
74	Muscle Î²1D Integrin Reinforces the Cytoskeletonâ€“Matrix Link: Modulation of Integrin Adhesive Function by Alternative Splicing. Journal of Cell Biology, 1997, 139, 1583-1595.	5.2	126
75	Focal Adhesion and Stress Fiber Formation Is Regulated by Tyrosine Phosphatase Activity. Experimental Cell Research, 1996, 229, 307-317.	2.6	76
76	p125FAK Tyrosine Phosphorylation and Focal Adhesion Assembly: Studies with Phosphotyrosine Phosphatase Inhibitors. Experimental Cell Research, 1995, 221, 141-152.	2.6	74
77	Expression of beta 1B integrin isoform in CHO cells results in a dominant negative effect on cell adhesion and motility.. Journal of Cell Biology, 1994, 127, 557-565.	5.2	69