## Tingting Cui

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/1461719/publications.pdf

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		759233	794594
19	850	12	19
papers	citations	h-index	g-index
19	19	19	1080
all docs	docs citations	times ranked	citing authors

#	Article	lF	CITATIONS
1	Impaired Activation of the Nrf2-ARE Signaling Pathway Undermines H2O2-Induced Oxidative Stress Response: A Possible Mechanism for Melanocyte Degeneration in Vitiligo. Journal of Investigative Dermatology, 2014, 134, 2221-2230.	0.7	145
2	Nrf2 Promotes Keratinocyte Proliferation in Psoriasis through Up-Regulation of Keratin 6, Keratin 16, and Keratin 17. Journal of Investigative Dermatology, 2017, 137, 2168-2176.	0.7	104
3	SIRT3-Dependent Mitochondrial Dynamics Remodeling Contributes to Oxidative Stress-Induced Melanocyte Degeneration in Vitiligo. Theranostics, 2019, 9, 1614-1633.	10.0	92
4	Dysregulated autophagy increased melanocyte sensitivity to H2O2-induced oxidative stress in vitiligo. Scientific Reports, 2017, 7, 42394.	3.3	85
5	Baicalein protects human vitiligo melanocytes from oxidative stress through activation of NF-E2-related factor2 (Nrf2) signaling pathway. Free Radical Biology and Medicine, 2018, 129, 492-503.	2.9	69
6	Oxidative Stress–Induced HMGB1 Release fromÂMelanocytes: A Paracrine Mechanism Underlying the Cutaneous Inflammation inÂVitiligo. Journal of Investigative Dermatology, 2019, 139, 2174-2184.e4.	0.7	64
7	Simvastatin Protects Human Melanocytes from H2O2-Induced Oxidative Stress byÂActivating Nrf2. Journal of Investigative Dermatology, 2017, 137, 1286-1296.	0.7	62
8	Oxidative stress-induced IL-15 trans-presentation in keratinocytes contributes to CD8+ T cells activation via JAK-STAT pathway in vitiligo. Free Radical Biology and Medicine, 2019, 139, 80-91.	2.9	52
9	Ginkgo biloba extract protects human melanocytes from H <sub>2</sub> O <sub>2</sub> â€induced oxidative stress by activating Nrf2. Journal of Cellular and Molecular Medicine, 2019, 23, 5193-5199.	3.6	35
10	HOâ€1 regulates the function of Treg: Association with the immune intolerance in vitiligo. Journal of Cellular and Molecular Medicine, 2018, 22, 4335-4343.	3.6	27
11	Role of the aryl hydrocarbon receptor signaling pathway in promoting mitochondrial biogenesis against oxidative damage in human melanocytes. Journal of Dermatological Science, 2019, 96, 33-41.	1.9	27
12	Oxeiptosis: a novel pathway of melanocytes death in response to oxidative stress in vitiligo. Cell Death Discovery, 2022, 8, 70.	4.7	21
13	Intracellular virus sensor MDA5 exacerbates vitiligo by inducing the secretion of chemokines in keratinocytes under virus invasion. Cell Death and Disease, 2020, 11, 453.	6.3	14
14	Homocysteine induces melanocytes apoptosis via PERK–elF2α–CHOP pathway in vitiligo. Clinical Science, 2020, 134, 1127-1141.	4.3	13
15	RIP1-Mediated Necroptosis Facilitates Oxidative Stressâ€'Induced Melanocyte Death, Offering Insight into Vitiligo. Journal of Investigative Dermatology, 2021, 141, 2921-2931.e6.	0.7	12
16	HSF1-Dependent Autophagy Activation Contributes to the Survival of Melanocytes Under Oxidative Stress in Vitiligo. Journal of Investigative Dermatology, 2022, 142, 1659-1669.e4.	0.7	12
17	Identification of Novel HLA-A*0201-Restricted CTL Epitopes in Chinese Vitiligo Patients. Scientific Reports, 2016, 6, 36360.	3.3	6
18	Identification of the Risk HLA-A Alleles and Autoantigen in Han Chinese Vitiligo Patients and the Association of CD8+T Cell Reactivity with Disease Characteristics. Medical Science Monitor, 2018, 24, 6489-6497.	1.1	6

#	Article	IF	CITATIONS
19	Metabolomics Signature and Potential Application of Serum Polyunsaturated Fatty Acids Metabolism in Patients With Vitiligo. Frontiers in Immunology, 2022, 13, 839167.	4.8	4