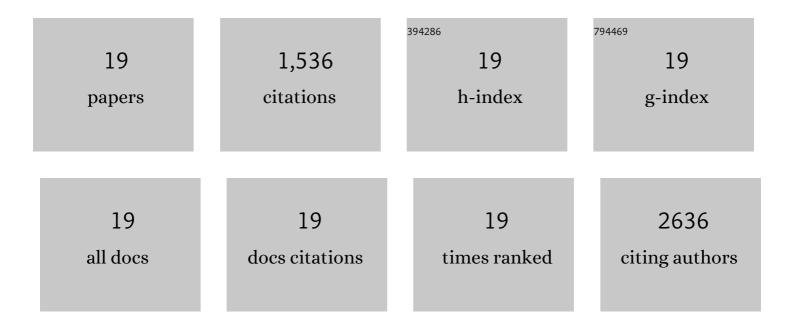
## Shuo Wei

List of Publications by Year in descending order

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SHUO WE

#	Article	IF	CITATIONS
1	The IL-33-PIN1-IRAK-M axis is critical for type 2 immunity in IL-33-induced allergic airway inflammation. Nature Communications, 2018, 9, 1603.	5.8	58
2	Pin1â€Targeted Therapy for Systemic Lupus Erythematosus. Arthritis and Rheumatology, 2016, 68, 2503-2513.	2.9	22
3	Chemical Tools To Decipher Regulation of Phosphatases by Proline Isomerization on Eukaryotic RNA Polymerase II. ACS Chemical Biology, 2015, 10, 2405-2414.	1.6	22
4	The Rab2A GTPase Promotes Breast Cancer Stem Cells and Tumorigenesis via Erk Signaling Activation. Cell Reports, 2015, 11, 111-124.	2.9	80
5	Antibody against early driver of neurodegeneration cis P-tau blocks brain injury and tauopathy. Nature, 2015, 523, 431-436.	13.7	374
6	Active Pin1 is a key target of all-trans retinoic acid in acute promyelocytic leukemia and breast cancer. Nature Medicine, 2015, 21, 457-466.	15.2	220
7	SENP1 deSUMOylates and Regulates Pin1 Protein Activity and Cellular Function. Cancer Research, 2013, 73, 3951-3962.	0.4	68
8	Negative Regulation of the Stability and Tumor Suppressor Function of Fbw7 by the Pin1 Prolyl Isomerase. Molecular Cell, 2012, 46, 771-783.	4.5	128
9	Targeting the Oncogenic E3 Ligase Skp2 in Prostate and Breast Cancer Cells with a Novel Energy Restriction-Mimetic Agent. PLoS ONE, 2012, 7, e47298.	1.1	28
10	Energy Restriction as an Antitumor Target of Thiazolidinediones. Journal of Biological Chemistry, 2010, 285, 9780-9791.	1.6	66
11	Development of Novel Adenosine Monophosphate-Activated Protein Kinase Activators. Journal of Medicinal Chemistry, 2010, 53, 2552-2561.	2.9	43
12	Â-Tocopheryl succinate and derivatives mediate the transcriptional repression of androgen receptor in prostate cancer cells by targeting the PP2A-JNK-Sp1-signaling axis. Carcinogenesis, 2009, 30, 1125-1131.	1.3	40
13	Thiazolidinediones Mimic Glucose Starvation in Facilitating Sp1 Degradation through the Up-Regulation of β-Transducin Repeat-Containing Protein. Molecular Pharmacology, 2009, 76, 47-57.	1.0	48
14	PPARÎ <sup>3</sup> -independent antitumor effects of thiazolidinediones. Cancer Letters, 2009, 276, 119-124.	3.2	99
15	Pharmacological Exploitation of the Peroxisome Proliferator-Activated Receptor Î <sup>3</sup> Agonist Ciglitazone To Develop a Novel Class of Androgen Receptor-Ablative Agents. Journal of Medicinal Chemistry, 2008, 51, 2100-2107.	2.9	23
16	A Novel Mechanism by Which Thiazolidinediones Facilitate the Proteasomal Degradation of Cyclin D1 in Cancer Cells. Journal of Biological Chemistry, 2008, 283, 26759-26770.	1.6	67
17	Peroxisome Proliferator-Activated Receptor γ–Independent Suppression of Androgen Receptor Expression by Troglitazone Mechanism and Pharmacologic Exploitation. Cancer Research, 2007, 67, 3229-3238.	0.4	46
18	Thiazolidinediones Modulate the Expression of β-Catenin and Other Cell-Cycle Regulatory Proteins by Targeting the F-Box Proteins of Skp1-Cul1-F-box Protein E3 Ubiquitin Ligase Independently of Peroxisome Proliferator-Activated Receptor γ. Molecular Pharmacology, 2007, 72, 725-733.	1.0	47

#	Article	IF	CITATIONS
19	Peroxisome Proliferator-Activated Receptor γ-Independent Repression of Prostate-Specific Antigen Expression by Thiazolidinediones in Prostate Cancer Cells. Molecular Pharmacology, 2006, 69, 1564-1570.	1.0	57