Silvia Buonamici

List of Publications by Year
in descending order

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Version: 2024-02-01

42 papers	7,518 citations	101543 36 h-index	265206 42 g-index
44 all docs	44 docs citations	44 times ranked	10557 citing authors

#	Article	IF	Citations
1	Sex-BiasedZRSR2Mutations in Myeloid Malignancies Impair Plasmacytoid Dendritic Cell Activation and Apoptosis. Cancer Discovery, 2022, 12, 522-541.	9.4	44
2	Spliceosome-targeted therapies trigger an antiviral immune response in triple-negative breast cancer. Cell, 2021, 184, 384-403.e21.	28.9	94
3	Intron retention is a hallmark and spliceosome represents a therapeutic vulnerability in aggressive prostate cancer. Nature Communications, 2020, 11, 2089.	12.8	83
4	Sensitivity to splicing modulation of BCL2 family genes defines cancer therapeutic strategies for splicing modulators. Nature Communications, 2019, 10, 137.	12.8	65
5	Splicing modulation as novel therapeutic strategy against diffuse malignant peritoneal mesothelioma. EBioMedicine, 2019, 39, 215-225.	6.1	41
6	Genome-wide CRISPR-Cas9 Screen Identifies Leukemia-Specific Dependence on a Pre-mRNA Metabolic Pathway Regulated by DCPS. Cancer Cell, 2018, 33, 386-400.e5.	16.8	99
7	The cryo-EM structure of the SF3b spliceosome complex bound to a splicing modulator reveals a pre-mRNA substrate competitive mechanism of action. Genes and Development, 2018, 32, 309-320.	5.9	89
8	Structural Basis of Splicing Modulation by Antitumor Macrolide Compounds. Molecular Cell, 2018, 70, 265-273.e8.	9.7	126
9	Somatic Mutational Landscape of Splicing Factor Genes and Their Functional Consequences across 33 Cancer Types. Cell Reports, 2018, 23, 282-296.e4.	6.4	333
10	H3B-8800, an orally available small-molecule splicing modulator, induces lethality in spliceosome-mutant cancers. Nature Medicine, 2018, 24, 497-504.	30.7	391
11	Targeting splicing abnormalities in cancer. Current Opinion in Genetics and Development, 2018, 48, 67-74.	3.3	72
12	SRPK1 maintains acute myeloid leukemia through effects on isoform usage of epigenetic regulators including BRD4. Nature Communications, 2018, 9, 5378.	12.8	60
13	Synthetic Lethal and Convergent Biological Effects of Cancer-Associated Spliceosomal Gene Mutations. Cancer Cell, 2018, 34, 225-241.e8.	16.8	162
14	Discovery of Asciminib (ABL001), an Allosteric Inhibitor of the Tyrosine Kinase Activity of BCR-ABL1. Journal of Medicinal Chemistry, 2018, 61, 8120-8135.	6.4	275
15	Splicing modulation sensitizes chronic lymphocytic leukemia cells to venetoclax by remodeling mitochondrial apoptotic dependencies. JCI Insight, 2018, 3, .	5.0	39
16	Splicing modulators act at the branch point adenosine binding pocket defined by the PHF5Aâ€“SF3b complex. Nature Communications, 2017, 8, 15522.	12.8	113
17	The allosteric inhibitor ABL001 enables dual targeting of BCRâ€“ABL1. Nature, 2017, 543, 733-737.	27.8	389
18	Basal-A Triple-Negative Breast Cancer Cells Selectively Rely on RNA Splicing for Survival. Molecular Cancer Therapeutics, 2017, 16, 2849-2861.	4.1	41

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19	Novel SF3B1 in-frame deletions result in aberrant RNA splicing in CLL patients. Blood Advances, 2017, 1, 995-1000.	5.2	23
20	Modulation of splicing catalysis for therapeutic targeting of leukemia with mutations in genes encoding spliceosomal proteins. Nature Medicine, 2016, 22, 672-678.	30.7	301
21	Physiologic Expression of Sf3b1 K700E Causes Impaired Erythropoiesis, Aberrant Splicing, and Sensitivity to Therapeutic Spliceosome Modulation. Cancer Cell, 2016, 30, 404-417.	16.8	318
22	Transcriptomic Characterization of SF3B1 Mutation Reveals Its Pleiotropic Effects in Chronic Lymphocytic Leukemia. Cancer Cell, 2016, 30, 750-763.	16.8	173
23	Cancer-Associated SF3B1 Hotspot Mutations Induce Cryptic 3â€² Splice Site Selection through Use of a Different Branch Point. Cell Reports, 2015, 13, 1033-1045.	6.4	377
24	SRSF2 Mutations Contribute to Myelodysplasia by Mutant-Specific Effects on Exon Recognition. Cancer Cell, 2015, 27, 617-630.	16.8	449
25	Coordinate activation of Shh and PI3K signaling in PTEN-deficient glioblastoma: new therapeutic opportunities. Nature Medicine, 2013, 19, 1518-1523.	30.7	127
26	Discovery of NVPâ€ŁEQ506, a Secondâ€Generation Inhibitor of Smoothened. ChemMedChem, 2013, 8, 1261-1265.	3.2	80
27	A novel tumour-suppressor function for the Notch pathway in myeloid leukaemia. Nature, 2011, 473, 230-233.	27.8	351
28	A crucial requirement for Hedgehog signaling in small cell lung cancer. Nature Medicine, 2011, 17, 1504-1508.	30.7	224
29	Interfering with Resistance to Smoothened Antagonists by Inhibition of the PI3K Pathway in Medulloblastoma. Science Translational Medicine, 2010, 2, 51ra70.	12.4	416
30	CCR7 signalling as an essential regulator of CNS infiltration in T-cell leukaemia. Nature, 2009, 459, 1000-1004.	27.8	227
31	Hedgehog Signaling Is Dispensable for Adult Hematopoietic Stem Cell Function. Cell Stem Cell, 2009, 4, 548-558.	11.1	174
32	Molecular pathogenesis of T-cell leukaemia and lymphoma. Nature Reviews Immunology, 2008, 8, 380-390.	22.7	396
33	Control of hematopoietic stem cell quiescence by the E3 ubiquitin ligase Fbw7. Journal of Experimental Medicine, 2008, 205, 1395-1408.	8.5	157
34	Knockdown of CCR7 or Its Ligands Causes a Loss of Central Nervous System Involvement in Notch1 Induced T-ALL. Blood, 2008, 112, 199-199.	1.4	4
35	Control of hematopoietic stem cell quiescence by the E3 ubiquitin ligase Fbw7. Journal of Cell Biology, 2008, 181, i16-i16.	5.2	0
36	The SCFFBW7 ubiquitin ligase complex as a tumor suppressor in T cell leukemia. Journal of Experimental Medicine, 2007, 204, 1825-1835.	8.5	427

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37	Targeting the NF-Î¹B signaling pathway in Notch1-induced T-cell leukemia. Nature Medicine, 2007, 13, 70-77.	30.7	315
38	EVI1 Abrogates Interferon-Î± Response by Selectively Blocking PML Induction. Journal of Biological Chemistry, 2005, 280, 428-436.	3.4	35
39	EVI1 induces myelodysplastic syndrome in mice. Journal of Clinical Investigation, 2004, 114, 713-719.	8.2	174
40	The role of EVI1 in normal and leukemic cells. Blood Cells, Molecules, and Diseases, 2003, 31, 206-212.	1.4	71
41	Association of 3q21q26 syndrome with different RPN1/EVI1 fusion transcripts. Haematologica, 2003, 88, 1221-8.	3.5	40
42	Real-time quantitation of minimal residual disease in inv(16)-positive acute myeloid leukemia may indicate risk for clinical relapse and may identify patients in a curable state. Blood, 2002, 99, 443-449.	1.4	133