

## List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Sam68 is required for the growth and survival of nonmelanoma skin cancer. Cancer Medicine, 2019, 8, 6106-6113.	1.3	13
2	Sam68/KHDRBS1 is critical for colon tumorigenesis by regulating genotoxic stress-induced NF-κB activation. ELife, 2016, 5, .	2.8	44
3	Ultrasound imaging of splenomegaly as a proxy to monitor colon tumor development in <i>Apc</i> <sup>min716/+</sup> mice. Cancer Medicine, 2016, 5, 2469-2476.	1.3	14
4	Sam68 Is Required for DNA Damage Responses via Regulating Poly(ADP-ribosyl)ation. PLoS Biology, 2016, 14, e1002543.	2.6	28
5	Sam68/KHDRBS1-dependent NF- $\hat{I}^{9}B$ activation confers radioprotection to the colon epithelium in $\hat{I}^{3}$ -irradiated mice. ELife, 2016, 5, .	2.8	13
6	TopBP1 Governs Hematopoietic Stem/Progenitor Cells Survival in Zebrafish Definitive Hematopoiesis. PLoS Genetics, 2015, 11, e1005346.	1.5	21
7	Metalloprotease NleC Suppresses Host NF-l̂ºB/Inflammatory Responses by Cleaving p65 and Interfering with the p65/RPS3 Interaction. PLoS Pathogens, 2015, 11, e1004705.	2.1	55
8	The PARP1/ARTD1-Mediated Poly-ADP-Ribosylation and DNA Damage Repair in B Cell Diversification. Antibodies, 2014, 3, 37-55.	1.2	2
9	Sam68 modulates the promoter specificity of NF-κB and mediates expression of CD25 in activated T cells. Nature Communications, 2013, 4, 1909.	5.8	40
10	Identification of an N-terminal Truncation of the NF-κB p65 Subunit That Specifically Modulates Ribosomal Protein S3-dependent NF-κB Gene Expression. Journal of Biological Chemistry, 2012, 287, 43019-43029.	1.6	31