

# Rachel E Simmonds

## List of Publications by Year in descending order

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35  
papers

1,888  
citations

346980

22  
h-index

466096

32  
g-index

39  
all docs

39  
docs citations

39  
times ranked

2521  
citing authors

#	ARTICLE	IF	CITATIONS
1	Aberrant stromal tissue factor localisation and mycolactone-driven vascular dysfunction, exacerbated by IL-1 $\beta$ , are linked to fibrin formation in Buruli ulcer lesions. <i>PLoS Pathogens</i> , 2022, 18, e1010280.	2.1	5
2	Synthesis, Biological Evaluation and Docking Studies of Ring-Opened Analogues of Ipomoeassin F. <i>Molecules</i> , 2022, 27, 4419.	1.7	0
3	Inhibition of the SEC61 translocon by mycolactone induces a protective autophagic response controlled by EIF2S1-dependent translation that does not require ULK1 activity. <i>Autophagy</i> , 2021, , 1-19.	4.3	6
4	Mycolactone enhances the Ca <sup>2+</sup> leak from endoplasmic reticulum by trapping Sec61 translocons in a Ca <sup>2+</sup> permeable state. <i>Biochemical Journal</i> , 2021, 478, 4005-4024.	1.7	13
5	The One That Got Away: How Macrophage-Derived IL-1 $\beta$ Escapes the Mycolactone-Dependent Sec61 Blockade in Buruli Ulcer. <i>Frontiers in Immunology</i> , 2021, 12, 788146.	2.2	6
6	Structure of the Inhibited State of the Sec Translocon. <i>Molecular Cell</i> , 2020, 79, 406-415.e7.	4.5	44
7	Ipomoeassin F Binds Sec61 $\beta$ to Inhibit Protein Translocation. <i>Journal of the American Chemical Society</i> , 2019, 141, 8450-8461.	6.6	58
8	Transient up-regulation of miR-155-3p by lipopolysaccharide in primary human monocyte-derived macrophages results in RISC incorporation but does not alter TNF expression. <i>Wellcome Open Research</i> , 2019, 4, 43.	0.9	10
9	Transient up-regulation of miR-155-3p by lipopolysaccharide in primary human monocyte-derived macrophages results in RISC incorporation but does not alter TNF expression. <i>Wellcome Open Research</i> , 2019, 4, 43.	0.9	4
10	Inhibition of Sec61-dependent translocation by mycolactone uncouples the integrated stress response from ER stress, driving cytotoxicity via translational activation of ATF4. <i>Cell Death and Disease</i> , 2018, 9, 397.	2.7	59
11	Buruli Ulcer: a Review of the Current Knowledge. <i>Current Tropical Medicine Reports</i> , 2018, 5, 247-256.	1.6	65
12	Buruli Ulcer: Case Study of a Neglected Tropical Disease. <i>Advances in Environmental Microbiology</i> , 2017, , 105-149.	0.1	0
13	Mycolactone reveals substrate-driven complexity of Sec61-dependent transmembrane protein biogenesis. <i>Journal of Cell Science</i> , 2017, 130, 1307-1320.	1.2	51
14	Mechanistic insights into the inhibition of Sec61-dependent co- and post-translational translocation by mycolactone. <i>Journal of Cell Science</i> , 2016, 129, 1404-15.	1.2	77
15	Recent advances: role of mycolactone in the pathogenesis and monitoring of <i>Mycobacterium ulcerans</i> infection/Buruli ulcer disease. <i>Cellular Microbiology</i> , 2016, 18, 17-29.	1.1	74
16	Mycolactone-Dependent Depletion of Endothelial Cell Thrombomodulin Is Strongly Associated with Fibrin Deposition in Buruli Ulcer Lesions. <i>PLoS Pathogens</i> , 2015, 11, e1005011.	2.1	38
17	The Pathogenic Mechanism of the <i>Mycobacterium ulcerans</i> Virulence Factor, Mycolactone, Depends on Blockade of Protein Translocation into the ER. <i>PLoS Pathogens</i> , 2014, 10, e1004061.	2.1	129
18	Pleiotropic molecular effects of the <i>Mycobacterium ulcerans</i> virulence factor mycolactone underlying the cell death and immunosuppression seen in Buruli ulcer. <i>Biochemical Society Transactions</i> , 2014, 42, 177-183.	1.6	51

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19	TLR Signalling and Adapter Utilization in Primary Human <i>in vitro</i> Differentiated Adipocytes. <i>Scandinavian Journal of Immunology</i> , 2012, 76, 359-370.	1.3	20
20	Mycolactone Inhibits Monocyte Cytokine Production by a Posttranscriptional Mechanism. <i>Journal of Immunology</i> , 2009, 182, 2194-2202.	0.4	89
21	Signalling, inflammation and arthritis: NF- $\kappa$ B and its relevance to arthritis and inflammation. <i>Rheumatology</i> , 2008, 47, 584-590.	0.9	309
22	Inhibitors of TLR8 Reduce TNF Production from Human Rheumatoid Synovial Membrane Cultures. <i>Journal of Immunology</i> , 2008, 181, 8002-8009.	0.4	85
23	Molecular diversity and thrombotic risk in protein S deficiency: The PROSIT study. <i>Human Mutation</i> , 2005, 25, 259-269.	1.1	48
24	Efficient isolation of peptide ligands for the endothelial cell protein C receptor (EPCR) using candidate receptor phage display biopanning. <i>Peptides</i> , 2005, 26, 1264-1269.	1.2	8
25	Coagulation, inflammation, and apoptosis: different roles for protein S and the protein S-C4b binding protein complex. <i>Blood</i> , 2004, 103, 1192-1201.	0.6	185
26	Regulation of the human endothelial cell protein C receptor gene promoter by multiple Sp1 binding sites. <i>Blood</i> , 2003, 101, 4393-4401.	0.6	12
27	Deletion or replacement of the second EGF-like domain of protein S results in loss of APC cofactor activity. <i>Blood</i> , 2003, 101, 1416-1418.	0.6	26
28	<i>In vitro</i> high level protein S expression after modification of protein S cDNA. <i>Thrombosis and Haemostasis</i> , 2003, 90, 1214-5.	1.8	3
29	Protein S Gla-domain mutations causing impaired Ca <sup>2+</sup> -induced phospholipid binding and severe functional protein S deficiency. <i>Blood</i> , 2002, 100, 2812-2819.	0.6	26
30	Genetic and Phenotypic Variability between Families with Hereditary Protein S Deficiency. <i>Thrombosis and Haemostasis</i> , 2002, 87, 258-265.	1.8	32
31	Haemostatic Genetic Risk Factors in Arterial Thrombosis. <i>Thrombosis and Haemostasis</i> , 2001, 86, 374-385.	1.8	51
32	Structural and Functional Implications of the Intron/Exon Organization of the Human Endothelial Cell Protein C/Activated Protein C Receptor (EPCR) Gene: Comparison With the Structure of CD1/Major Histocompatibility Complex $\alpha$ 1 and $\alpha$ 2 Domains. <i>Blood</i> , 1999, 94, 632-641.	0.6	114
33	Structural and Functional Implications of the Intron/Exon Organization of the Human Endothelial Cell Protein C/Activated Protein C Receptor (EPCR) Gene: Comparison With the Structure of CD1/Major Histocompatibility Complex $\alpha$ 1 and $\alpha$ 2 Domains. <i>Blood</i> , 1999, 94, 632-641.	0.6	0
34	Clarification of the Risk for Venous Thrombosis Associated with Hereditary Protein S Deficiency by Investigation of a Large Kindred with a Characterized Gene Defect. <i>Annals of Internal Medicine</i> , 1998, 128, 8.	2.0	91
35	Genetic and Phenotypic Analysis of a Large (122-Member) Protein S-Deficient Kindred Provides an Explanation for the Familial Coexistence of Type I and Type III Plasma Phenotypes. <i>Blood</i> , 1997, 89, 4364-4370.	0.6	98