## Elaine A Dunlop

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Mechanistic target of rapamycin inhibitors: successes and challenges as cancer therapeutics. , 2019, 2, 1069-1085.		11
2	Rab35-dependent extracellular nanovesicles are required for induction of tumour supporting stroma. Nanoscale, 2018, 10, 8547-8559.	5.6	20
3	Energy Stress-Mediated Cytotoxicity in Tuberous Sclerosis Complex 2-Deficient Cells with Nelfinavir and Mefloquine Treatment. Cancers, 2018, 10, 375.	3.7	5
4	Loss of tuberous sclerosis complex 2 sensitizes tumors to nelfinavirâ^'bortezomib therapy to intensify endoplasmic reticulum stress-induced cell death. Oncogene, 2018, 37, 5913-5925.	5.9	10
5	The lysosome: a crucial hub for AMPK and mTORC1 signalling. Biochemical Journal, 2017, 474, 1453-1466.	3.7	143
6	Targeting protein homeostasis with nelfinavir/salinomycin dual therapy effectively induces death of mTORC1 hyperactive cells. Oncotarget, 2017, 8, 48711-48724.	1.8	13
7	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	9.1	4,701
8	Tuberous sclerosis—A model for tumour growth. Seminars in Cell and Developmental Biology, 2016, 52, 3-11.	5.0	18
9	Control of TSC2-Rheb signaling axis by arginine regulates mTORC1 activity. ELife, 2016, 5, .	6.0	147
10	FLCN, a novel autophagy component, interacts with GABARAP and is regulated by ULK1 phosphorylation. Autophagy, 2014, 10, 1749-1760.	9.1	64
11	mTOR and autophagy: A dynamic relationship governed by nutrients and energy. Seminars in Cell and Developmental Biology, 2014, 36, 121-129.	5.0	382
12	The tumor suppressor folliculin regulates AMPK-dependent metabolic transformation. Journal of Clinical Investigation, 2014, 124, 2640-2650.	8.2	124
13	A tuberous sclerosis complex signalling node at the peroxisome regulates mTORC1 and autophagy in response to ROS. Nature Cell Biology, 2013, 15, 1186-1196.	10.3	218
14	The kinase triad, AMPK, mTORC1 and ULK1, maintains energy and nutrient homoeostasis. Biochemical Society Transactions, 2013, 41, 939-943.	3.4	109
15	Assessment of the potential pathogenicity of missense mutations identified in the GTPase-activating protein (GAP)-related domain of the neurofibromatosis type-1 ( <i>NF1</i> ) gene. Human Mutation, 2012, 33, 1687-1696.	2.5	21
16	Absence of the Birt–Hogg–Dubé gene product is associated with increased hypoxia-inducible factor transcriptional activity and a loss of metabolic flexibility. Oncogene, 2011, 30, 1159-1173.	5.9	69
17	Determining the pathogenicity of patient-derived TSC2 mutations by functional characterization and clinical evidence. European Journal of Human Genetics, 2011, 19, 789-795.	2.8	9
18	ULK1 inhibits mTORC1 signaling, promotes multisite Raptor phosphorylation and hinders substrate binding. Autophagy, 2011, 7, 737-747.	9.1	177

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19	mTOR Ser-2481 Autophosphorylation Monitors mTORC-specific Catalytic Activity and Clarifies Rapamycin Mechanism of Action. Journal of Biological Chemistry, 2010, 285, 7866-7879.	3.4	189
20	Erythropoietin-Induced Activation of the JAK2/STAT5, PI3K/Akt, and Ras/ERK Pathways Promotes Malignant Cell Behavior in a Modified Breast Cancer Cell Line. Molecular Cancer Research, 2010, 8, 615-626.	3.4	61
21	Mammalian target of rapamycin complex 1: Signalling inputs, substrates and feedback mechanisms. Cellular Signalling, 2009, 21, 827-835.	3.6	220
22	Mammalian target of rapamycin complex 1-mediated phosphorylation of eukaryotic initiation factor 4E-binding protein 1 requires multiple protein–protein interactions for substrate recognition. Cellular Signalling, 2009, 21, 1073-1084.	3.6	72
23	Impaired Downregulation Following Erythropoietin Receptor Activation in Non-Small Cell Lung Carcinoma. Stem Cells, 2007, 25, 380-384.	3.2	18
24	Erythropoietin Receptor Expression in Non-Small Cell Lung Carcinoma: A Question of Antibody Specificity. Stem Cells, 2007, 25, 718-722.	3.2	86
25	Induction of Signalling in Non-Erythroid Cells by Pharmacological Levels of Erythropoietin. Neurodegenerative Diseases, 2006, 3, 94-100.	1.4	52