

Donald E Ayer

List of Publications by Year in descending order

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46
papers

7,832
citations

117625

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docs citations

54
times ranked

8189
citing authors

#	ARTICLE	IF	CITATIONS
1	Age-associated decline of MondoA drives cellular senescence through impaired autophagy and mitochondrial homeostasis. <i>Cell Reports</i> , 2022, 38, 110444.	6.4	27
2	The glucose-sensing transcription factor MLX balances metabolism and stress to suppress apoptosis and maintain spermatogenesis. <i>PLoS Biology</i> , 2021, 19, e3001085.	5.6	7
3	Protein synthesis inhibitors stimulate MondoA transcriptional activity by driving an accumulation of glucose 6-phosphate. <i>Cancer & Metabolism</i> , 2020, 8, 27.	5.0	9
4	MondoA drives muscle lipid accumulation and insulin resistance. <i>JCI Insight</i> , 2019, 4, .	5.0	22
5	Cellular acidosis triggers human MondoA transcriptional activity by driving mitochondrial ATP production. <i>ELife</i> , 2019, 8, .	6.0	41
6	Pan-cancer Alterations of the MYC Oncogene and Its Proximal Network across the Cancer Genome Atlas. <i>Cell Systems</i> , 2018, 6, 282-300.e2.	6.2	284
7	Extracellular Matrix Remodeling Regulates Glucose Metabolism through TXNIP Destabilization. <i>Cell</i> , 2018, 175, 117-132.e21.	28.9	180
8	Ras Suppresses TXNIP Expression by Restricting Ribosome Translocation. <i>Molecular and Cellular Biology</i> , 2018, 38, .	2.3	12
9	Deregulated Myc Requires MondoA/Mlx for Metabolic Reprogramming and Tumorigenesis. <i>Cancer Cell</i> , 2015, 27, 271-285.	16.8	172
10	MondoA-Mlx Transcriptional Activity Is Limited by mTOR-MondoA Interaction. <i>Molecular and Cellular Biology</i> , 2015, 35, 101-110.	2.3	34
11	Metabolic reprogramming in triple-negative breast cancer through Myc suppression of TXNIP. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2015, 112, 5425-5430.	7.1	190
12	Interactions between Myc and MondoA transcription factors in metabolism and tumourigenesis. <i>British Journal of Cancer</i> , 2015, 113, 1529-1533.	6.4	50
13	Foxk proteins repress the initiation of starvation-induced atrophy and autophagy programs. <i>Nature Cell Biology</i> , 2014, 16, 1202-1214.	10.3	120
14	Response of <i>BRAF</i> -Mutant Melanoma to BRAF Inhibition Is Mediated by a Network of Transcriptional Regulators of Glycolysis. <i>Cancer Discovery</i> , 2014, 4, 423-433.	9.4	242
15	Response to Acidity: The MondoA-TXNIP Checkpoint Couples the Acidic Tumor Microenvironment to Cell Metabolism. , 2014, , 69-100.		0
16	Adaptive metabolic response to 4 weeks of sugar-sweetened beverage consumption in healthy, lightly active individuals and chronic high glucose availability in primary human myotubes. <i>European Journal of Nutrition</i> , 2013, 52, 937-948.	3.9	12
17	MondoA senses adenine nucleotides: transcriptional induction of thioredoxin-interacting protein. <i>Biochemical Journal</i> , 2013, 453, 209-218.	3.7	13
18	Coordination of Nutrient Availability and Utilization by MAX- and MLX-Centered Transcription Networks. <i>Cold Spring Harbor Perspectives in Medicine</i> , 2013, 3, a014258-a014258.	6.2	43

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19	An extended Myc network contributes to glucose homeostasis in cancer and diabetes. <i>Frontiers in Bioscience - Landmark</i> , 2011, 16, 2206.	3.0	43
20	MondoA Senses Non-glucose Sugars. <i>Journal of Biological Chemistry</i> , 2011, 286, 38027-38034.	3.4	60
21	Glucose Controls Nuclear Accumulation, Promoter Binding, and Transcriptional Activity of the MondoA-Mlx Heterodimer. <i>Molecular and Cellular Biology</i> , 2010, 30, 2887-2895.	2.3	84
22	Myc, Mondo, and Metabolism. <i>Genes and Cancer</i> , 2010, 1, 587-596.	1.9	35
23	Transcriptional and Translational Downregulation of Thioredoxin Interacting Protein Is Required for Metabolic Reprogramming during G1. <i>Genes and Cancer</i> , 2010, 1, 893-907.	1.9	67
24	Coordination of glucose and glutamine utilization by an expanded Myc network. <i>Transcription</i> , 2010, 1, 36-40.	3.1	28
25	Glutamine-dependent anapleurosis dictates glucose uptake and cell growth by regulating MondoA transcriptional activity. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2009, 106, 14878-14883.	7.1	142
26	Over-expression of the BRMS1 family member SUDS3 does not suppress metastasis of human cancer cells. <i>Cancer Letters</i> , 2009, 276, 32-37.	7.2	17
27	Glucose sensing by MondoA:Mlx complexes: A role for hexokinases and direct regulation of thioredoxin-interacting protein expression. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2008, 105, 6912-6917.	7.1	238
28	A <i>C. elegans</i> Myc-like network cooperates with semaphorin and Wnt signaling pathways to control cell migration. <i>Developmental Biology</i> , 2007, 310, 226-239.	2.0	37
29	MondoA-Mlx Heterodimers Are Candidate Sensors of Cellular Energy Status: Mitochondrial Localization and Direct Regulation of Glycolysis. <i>Molecular and Cellular Biology</i> , 2006, 26, 4863-4871.	2.3	101
30	ING2 PHD domain links histone H3 lysine 4 methylation to active gene repression. <i>Nature</i> , 2006, 442, 96-99.	27.8	851
31	The Polybasic Region That Follows the Plant Homeodomain Zinc Finger 1 of Pf1 Is Necessary and Sufficient for Specific Phosphoinositide Binding. <i>Journal of Biological Chemistry</i> , 2006, 281, 28831-28836.	3.4	65
32	Identification and Characterization of Three New Components of the mSin3A Corepressor Complex. <i>Molecular and Cellular Biology</i> , 2003, 23, 3456-3467.	2.3	164
33	Role for the Mortality Factors MORF4, MRCX, and MRC15 in Transcriptional Repression via Associations with Pf1, mSin3A, and Transducin-Like Enhancer of Split. <i>Molecular and Cellular Biology</i> , 2002, 22, 7868-7876.	2.3	89
34	A Novel Heterodimerization Domain, CRM1, and 14-3-3 Control Subcellular Localization of the MondoA-Mlx Heterocomplex. <i>Molecular and Cellular Biology</i> , 2002, 22, 8514-8526.	2.3	45
35	Pf1, a Novel PHD Zinc Finger Protein That Links the TLE Corepressor to the mSin3A-Histone Deacetylase Complex. <i>Molecular and Cellular Biology</i> , 2001, 21, 4110-4118.	2.3	80
36	MondoA, a Novel Basic Helix-Loop-Helix/Leucine Zipper Transcriptional Activator That Constitutes a Positive Branch of a Max-Like Network. <i>Molecular and Cellular Biology</i> , 2000, 20, 8845-8854.	2.3	118

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37	Î²-Cateninâ€™ Histone Deacetylase Interactions Regulate the Transition of LEF1 from a Transcriptional Repressor to an Activator. <i>Molecular and Cellular Biology</i> , 2000, 20, 6882-6890.	2.3	200
38	Solution Structure of the Interacting Domains of the Madâ€™Sin3 Complex. <i>Cell</i> , 2000, 103, 655-665.	28.9	95
39	Mlx, a Novel Max-like BHLHZip Protein That Interacts with the Max Network of Transcription Factors. <i>Journal of Biological Chemistry</i> , 1999, 274, 36344-36350.	3.4	89
40	A 13-Amino Acid Amphipathic Î±-Helix Is Required for the Functional Interaction between the Transcriptional Repressor Mad1 and mSin3A. <i>Journal of Biological Chemistry</i> , 1999, 274, 32750-32756.	3.4	58
41	Histone deacetylases: transcriptional repression with SINers and NuRDs. <i>Trends in Cell Biology</i> , 1999, 9, 193-198.	7.9	257
42	SAP30, a Component of the mSin3 Corepressor Complex Involved in N-CoR-Mediated Repression by Specific Transcription Factors. <i>Molecular Cell</i> , 1998, 2, 33-42.	9.7	196
43	Histone Deacetylase Activity Is Required for Full Transcriptional Repression by mSin3A. <i>Cell</i> , 1997, 89, 341-347.	28.9	705
44	Nuclear Receptor Repression Mediated by a Complex Containing SMRT, mSin3A, and Histone Deacetylase. <i>Cell</i> , 1997, 89, 373-380.	28.9	1,206
45	Mad-max transcriptional repression is mediated by ternary complex formation with mammalian homologs of yeast repressor Sin3. <i>Cell</i> , 1995, 80, 767-776.	28.9	585
46	Mad: A heterodimeric partner for Max that antagonizes Myc transcriptional activity. <i>Cell</i> , 1993, 72, 211-222.	28.9	717