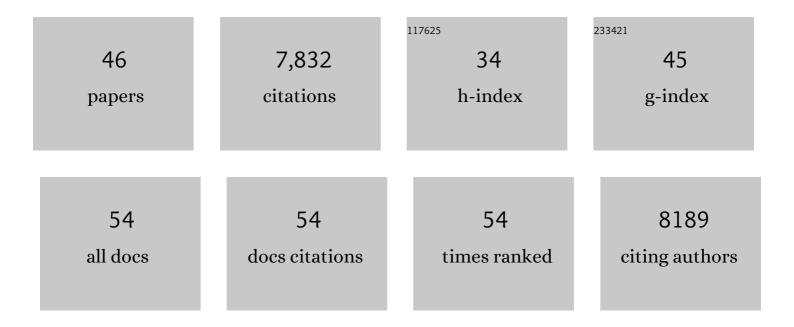
## Donald E Ayer

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Nuclear Receptor Repression Mediated by a Complex Containing SMRT, mSin3A, and Histone Deacetylase. Cell, 1997, 89, 373-380.	28.9	1,206
2	ING2 PHD domain links histone H3 lysine 4 methylation to active gene repression. Nature, 2006, 442, 96-99.	27.8	851
3	Mad: A heterodimeric partner for Max that antagonizes Myc transcriptional activity. Cell, 1993, 72, 211-222.	28.9	717
4	Histone Deacetylase Activity Is Required for Full Transcriptional Repression by mSin3A. Cell, 1997, 89, 341-347.	28.9	705
5	Mad-max transcriptional repression is mediated by ternary complex formation with mammalian homologs of yeast repressor Sin3. Cell, 1995, 80, 767-776.	28.9	585
6	Pan-cancer Alterations of the MYC Oncogene and Its Proximal Network across the Cancer Genome Atlas. Cell Systems, 2018, 6, 282-300.e2.	6.2	284
7	Histone deacetylases: transcriptional repression with SINers and NuRDs. Trends in Cell Biology, 1999, 9, 193-198.	7.9	257
8	Response of <i>BRAF</i> -Mutant Melanoma to BRAF Inhibition Is Mediated by a Network of Transcriptional Regulators of Glycolysis. Cancer Discovery, 2014, 4, 423-433.	9.4	242
9	Glucose sensing by MondoA:Mlx complexes: A role for hexokinases and direct regulation of thioredoxin-interacting protein expression. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 6912-6917.	7.1	238
10	β-Catenin–Histone Deacetylase Interactions Regulate the Transition of LEF1 from a Transcriptional Repressor to an Activator. Molecular and Cellular Biology, 2000, 20, 6882-6890.	2.3	200
11	SAP30, a Component of the mSin3 Corepressor Complex Involved in N-CoR-Mediated Repression by Specific Transcription Factors. Molecular Cell, 1998, 2, 33-42.	9.7	196
12	Metabolic reprogramming in triple-negative breast cancer through Myc suppression of TXNIP. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, 5425-5430.	7.1	190
13	Extracellular Matrix Remodeling Regulates Glucose Metabolism through TXNIP Destabilization. Cell, 2018, 175, 117-132.e21.	28.9	180
14	Deregulated Myc Requires MondoA/Mlx for Metabolic Reprogramming and Tumorigenesis. Cancer Cell, 2015, 27, 271-285.	16.8	172
15	Identification and Characterization of Three New Components of the mSin3A Corepressor Complex. Molecular and Cellular Biology, 2003, 23, 3456-3467.	2.3	164
16	Glutamine-dependent anapleurosis dictates glucose uptake and cell growth by regulating MondoA transcriptional activity. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 14878-14883.	7.1	142
17	Foxk proteins repress the initiation of starvation-induced atrophy and autophagy programs. Nature Cell Biology, 2014, 16, 1202-1214.	10.3	120
18	MondoA, a Novel Basic Helix-Loop-Helix–Leucine Zipper Transcriptional Activator That Constitutes a Positive Branch of a Max-Like Network. Molecular and Cellular Biology, 2000, 20, 8845-8854.	2.3	118

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19	MondoA-Mlx Heterodimers Are Candidate Sensors of Cellular Energy Status: Mitochondrial Localization and Direct Regulation of Glycolysis. Molecular and Cellular Biology, 2006, 26, 4863-4871.	2.3	101
20	Solution Structure of the Interacting Domains of the Mad–Sin3 Complex. Cell, 2000, 103, 655-665.	28.9	95
21	Mlx, a Novel Max-like BHLHZip Protein That Interacts with the Max Network of Transcription Factors. Journal of Biological Chemistry, 1999, 274, 36344-36350.	3.4	89
22	Role for the Mortality Factors MORF4, MRGX, and MRG15 in Transcriptional Repression via Associations with Pf1, mSin3A, and Transducin-Like Enhancer of Split. Molecular and Cellular Biology, 2002, 22, 7868-7876.	2.3	89
23	Glucose Controls Nuclear Accumulation, Promoter Binding, and Transcriptional Activity of the MondoA-Mlx Heterodimer. Molecular and Cellular Biology, 2010, 30, 2887-2895.	2.3	84
24	Pf1, a Novel PHD Zinc Finger Protein That Links the TLE Corepressor to the mSin3A-Histone Deacetylase Complex. Molecular and Cellular Biology, 2001, 21, 4110-4118.	2.3	80
25	Transcriptional and Translational Downregulation of Thioredoxin Interacting Protein Is Required for Metabolic Reprogramming during G1. Genes and Cancer, 2010, 1, 893-907.	1.9	67
26	The Polybasic Region That Follows the Plant Homeodomain Zinc Finger 1 of Pf1 Is Necessary and Sufficient for Specific Phosphoinositide Binding. Journal of Biological Chemistry, 2006, 281, 28831-28836.	3.4	65
27	MondoA Senses Non-glucose Sugars. Journal of Biological Chemistry, 2011, 286, 38027-38034.	3.4	60
28	A 13-Amino Acid Amphipathic α-Helix Is Required for the Functional Interaction between the Transcriptional Repressor Mad1 and mSin3A. Journal of Biological Chemistry, 1999, 274, 32750-32756.	3.4	58
29	Interactions between Myc and MondoA transcription factors in metabolism and tumourigenesis. British Journal of Cancer, 2015, 113, 1529-1533.	6.4	50
30	A Novel Heterodimerization Domain, CRM1, and 14-3-3 Control Subcellular Localization of the MondoA-Mlx Heterocomplex. Molecular and Cellular Biology, 2002, 22, 8514-8526.	2.3	45
31	An extended Myc network contributes to glucose homeostasis in cancer and diabetes. Frontiers in Bioscience - Landmark, 2011, 16, 2206.	3.0	43
32	Coordination of Nutrient Availability and Utilization by MAX- and MLX-Centered Transcription Networks. Cold Spring Harbor Perspectives in Medicine, 2013, 3, a014258-a014258.	6.2	43
33	Cellular acidosis triggers human MondoA transcriptional activity by driving mitochondrial ATP production. ELife, 2019, 8, .	6.0	41
34	A C. elegans Myc-like network cooperates with semaphorin and Wnt signaling pathways to control cell migration. Developmental Biology, 2007, 310, 226-239.	2.0	37
35	Myc, Mondo, and Metabolism. Genes and Cancer, 2010, 1, 587-596.	1.9	35
36	MondoA-Mlx Transcriptional Activity Is Limited by mTOR-MondoA Interaction. Molecular and Cellular Biology, 2015, 35, 101-110.	2.3	34

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37	Coordination of glucose and glutamine utilization by an expanded Myc network. Transcription, 2010, 1, 36-40.	3.1	28
38	Age-associated decline of MondoA drives cellular senescence through impaired autophagy and mitochondrial homeostasis. Cell Reports, 2022, 38, 110444.	6.4	27
39	MondoA drives muscle lipid accumulation and insulin resistance. JCI Insight, 2019, 4, .	5.0	22
40	Over-expression of the BRMS1 family member SUDS3 does not suppress metastasis of human cancer cells. Cancer Letters, 2009, 276, 32-37.	7.2	17
41	MondoA senses adenine nucleotides: transcriptional induction of thioredoxin-interacting protein. Biochemical Journal, 2013, 453, 209-218.	3.7	13
42	Adaptive metabolic response to 4Âweeks of sugar-sweetened beverage consumption in healthy, lightly active individuals and chronic high glucose availability in primary human myotubes. European Journal of Nutrition, 2013, 52, 937-948.	3.9	12
43	Ras Suppresses TXNIP Expression by Restricting Ribosome Translocation. Molecular and Cellular Biology, 2018, 38, .	2.3	12
44	Protein synthesis inhibitors stimulate MondoA transcriptional activity by driving an accumulation of glucose 6-phosphate. Cancer & Metabolism, 2020, 8, 27.	5.0	9
45	The glucose-sensing transcription factor MLX balances metabolism and stress to suppress apoptosis and maintain spermatogenesis. PLoS Biology, 2021, 19, e3001085.	5.6	7
46	Response to Acidity: The MondoA–TXNIP Checkpoint Couples the Acidic Tumor Microenvironment to Cell Metabolism. , 2014, , 69-100.		0