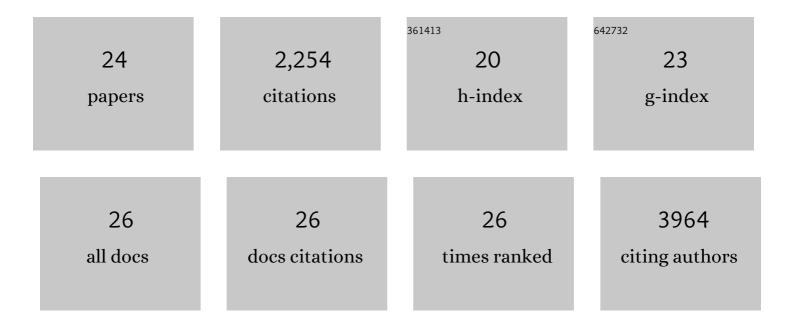
Yann-Gaël Gangloff

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	The ESCRT-0 subcomplex component Hrs/Hgs is a master regulator of myogenesis via modulation of signaling and degradation pathways. BMC Biology, 2021, 19, 153.	3.8	4
2	H2A.Z is dispensable for both basal and activated transcription in post-mitotic mouse muscles. Nucleic Acids Research, 2020, 48, 4601-4613.	14.5	18
3	Lack of muscle mTOR kinase activity causes early onset myopathy and compromises wholeâ€body homeostasis. Journal of Cachexia, Sarcopenia and Muscle, 2019, 10, 35-53.	7.3	24
4	Increased Serpina3n release into circulation during glucocorticoidâ€mediated muscle atrophy. Journal of Cachexia, Sarcopenia and Muscle, 2018, 9, 929-946.	7.3	53
5	Resistance exercise initiates mechanistic target of rapamycin (mTOR) translocation and protein complex co-localisation in human skeletal muscle. Scientific Reports, 2017, 7, 5028.	3.3	86
6	mTOR inactivation in myocardium from infant mice rapidly leads to dilated cardiomyopathy due to translation defects and p53/JNK-mediated apoptosis. Journal of Molecular and Cellular Cardiology, 2016, 97, 213-225.	1.9	43
7	S6K1 controls pancreatic \hat{l}^2 cell size independently of intrauterine growth restriction. Journal of Clinical Investigation, 2015, 125, 2736-2747.	8.2	23
8	The metabolic checkpoint kinase mTOR is essential for IL-15 signaling during the development and activation of NK cells. Nature Immunology, 2014, 15, 749-757.	14.5	484
9	Arrest of Myelination and Reduced Axon Growth When Schwann Cells Lack mTOR. Journal of Neuroscience, 2012, 32, 1817-1825.	3.6	125
10	Myopathy caused by mammalian target of rapamycin complex 1 (mTORC1) inactivation is not reversed by restoring mitochondrial function. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 20808-20813.	7.1	38
11	Muscle inactivation of mTOR causes metabolic and dystrophin defects leading to severe myopathy. Journal of Cell Biology, 2009, 187, 859-874.	5.2	320
12	Muscle inactivation of mTOR causes metabolic and dystrophin defects leading to severe myopathy. Journal of Experimental Medicine, 2009, 206, i33-i33.	8.5	0
13	Disruption of the Mouse mTOR Gene Leads to Early Postimplantation Lethality and Prohibits Embryonic Stem Cell Development. Molecular and Cellular Biology, 2004, 24, 9508-9516.	2.3	427
14	Crystal Structure of a Subcomplex of Human Transcription Factor TFIID Formed by TATA Binding Protein-associated Factors hTAF4 (hTAFII135) and hTAF12 (hTAFII20). Journal of Biological Chemistry, 2002, 277, 45502-45509.	3.4	56
15	Distinct Mutations in Yeast TAF II 25 Differentially Affect the Composition of TFIID and SAGA Complexes as Well as Global Gene Expression Patterns. Molecular and Cellular Biology, 2002, 22, 3178-3193.	2.3	31
16	Functional Analysis of the TFIID-specific Yeast TAF4 (yTAFII48) Reveals an Unexpected Organization of Its Histone-fold Domain. Journal of Biological Chemistry, 2002, 277, 45510-45517.	3.4	25
17	Dissecting the interaction network of multiprotein complexes by pairwise coexpression of subunits in E. coli11Edited by K. Nagai. Journal of Molecular Biology, 2001, 306, 363-373.	4.2	64
18	The histone fold is a key structural motif of transcription factor TFIID. Trends in Biochemical Sciences, 2001, 26, 250-257.	7.5	127

#	Article	IF	CITATIONS
19	Histone Folds Mediate Selective Heterodimerization of Yeast TAF II 25 with TFIID Components yTAF II 47 and yTAF II 65 and with SAGA Component ySPT7. Molecular and Cellular Biology, 2001, 21, 1841-1853.	2.3	66
20	The TFIID Components Human TAF II 140 and Drosophila BIP2 (TAF II 155) Are Novel Metazoan Homologues of Yeast TAF II 47 Containing a Histone Fold and a PHD Finger. Molecular and Cellular Biology, 2001, 21, 5109-5121.	2.3	62
21	The Human TFIID Components TAF _{II} 135 and TAF _{II} 20 and the Yeast SAGA Components ADA1 and TAF _{II} 68 Heterodimerize to Form Histone-Like Pairs. Molecular and Cellular Biology, 2000, 20, 340-351.	2.3	86
22	The Human Transcription Factor IID Subunit Human TATA-binding Protein-associated Factor 28 Interacts in a Ligand-reversible Manner with the Vitamin D3 and Thyroid Hormone Receptors. Journal of Biological Chemistry, 2000, 275, 10064-10071.	3.4	20
23	Human TAF _{II} 55 Interacts with the Vitamin D ₃ and Thyroid Hormone Receptors and with Derivatives of the Retinoid X Receptor That Have Altered Transactivation Properties. Molecular and Cellular Biology, 1999, 19, 5486-5494.	2.3	47
24	Synergistic Transcriptional Activation by TATA-Binding Protein and hTAF _{II} 28 Requires Specific Amino Acids of the hTAF _{II} 28 Histone Fold. Molecular and Cellular Biology, 1999, 19, 5050-5060.	2.3	23