Ricardo Mouro Pinto

List of Publications by Year
in descending order

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21 papers	2,139 citations	516681 16 h-index	713444 21 g-index
26 all docs	26 docs citations	26 times ranked	2612 citing authors

#	Article	IF	Citations
1	Modifiers of Somatic Repeat Instability in Mouse Models of Friedreich Ataxia and the Fragile X-Related Disorders: Implications for the Mechanism of Somatic Expansion in Huntingtonâ€™s Disease. Journal of Huntington's Disease, 2021, 10, 149-163.	1.9	15
2	Approaches to Sequence the HTT CAG Repeat Expansion and Quantify Repeat Length Variation. Journal of Huntington's Disease, 2021, 10, 53-74.	1.9	16
3	Somatic CAG expansion in Huntington's disease is dependent on the MLH3 endonuclease domain, which can be excluded via splice redirection. Nucleic Acids Research, 2021, 49, 3907-3918.	14.5	20
4	Genetic Risk Underlying Psychiatric and Cognitive Symptoms in Huntingtonâ€™s Disease. Biological Psychiatry, 2020, 87, 857-865.	1.3	29
5	Patterns of CAG repeat instability in the central nervous system and periphery in Huntingtonâ€™s disease and in spinocerebellar ataxia type 1. Human Molecular Genetics, 2020, 29, 2551-2567.	2.9	69
6	Promotion of somatic CAG repeat expansion by Fan1 knock-out in Huntingtonâ€™s disease knock-in mice is blocked by Mlh1 knock-out. Human Molecular Genetics, 2020, 29, 3044-3053.	2.9	48
7	Histone deacetylase knockouts modify transcription, CAG instability and nuclear pathology in Huntington disease mice. ELife, 2020, 9, .	6.0	9
8	CAG Repeat Not Polyglutamine Length Determines Timing of Huntingtonâ€™s Disease Onset. Cell, 2019, 178, 887-900.e14.	28.9	301
9	Heterozygous mutations cause genetic instability in a yeast model of cancer evolution. Nature, 2019, 566, 275-278.	27.8	27
10	Assessing average somatic CAG repeat instability at the protein level. Scientific Reports, 2019, 9, 19152.	3.3	17
11	Genetic Contributors to Intergenerational CAG Repeat Instability in Huntingtonâ€™s Disease Knock-In Mice. Genetics, 2017, 205, 503-516.	2.9	17
12	Identification of Genetic Factors that Modify Clinical Onset of Huntingtonâ€™s Disease. Cell, 2015, 162, 516-526.	28.9	514
13	Mismatch Repair Genes Mlh1 and Mlh3 Modify CAG Instability in Huntington's Disease Mice: Genome-Wide and Candidate Approaches. PLoS Genetics, 2013, 9, e1003930.	3.5	175
14	Friedreich Ataxia Patient Tissues Exhibit Increased 5-Hydroxymethylcytosine Modification and Decreased CTCF Binding at the FXN Locus. PLoS ONE, 2013, 8, e74956.	2.5	29
15	The mismatch repair system protects against intergenerational GAA repeat instability in a Friedreich ataxia mouse model. Neurobiology of Disease, 2012, 46, 165-171.	4.4	53
16	Pms2 Suppresses Large Expansions of the (GAAÂ·TTC)n Sequence in Neuronal Tissues. PLoS ONE, 2012, 7, e47085.	2.5	43
17	Quantification of Age-Dependent Somatic CAG Repeat Instability in Hdh CAG Knock-In Mice Reveals Different Expansion Dynamics in Striatum and Liver. PLoS ONE, 2011, 6, e23647.	2.5	62
18	Prolonged treatment with pimelic o-aminobenzamide HDAC inhibitors ameliorates the disease phenotype of a Friedreich ataxia mouse model. Neurobiology of Disease, 2011, 42, 496-505.	4.4	109

#	Article	IF	Citations
19	The Friedreich ataxia GAA repeat expansion mutation induces comparable epigenetic changes in human and transgenic mouse brain and heart tissues. Human Molecular Genetics, 2007, 17, 735-746.	2.9	229
20	GAA repeat expansion mutation mouse models of Friedreich ataxia exhibit oxidative stress leading to progressive neuronal and cardiac pathology. Genomics, 2006, 88, 580-590.	2.9	222
21	GAA repeat instability in Friedreich ataxia YAC transgenic mice. Genomics, 2004, 84, 301-310.	2.9	84