## Ricardo Mouro Pinto

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Identification of Genetic Factors that Modify Clinical Onset of Huntington's Disease. Cell, 2015, 162, 516-526.	28.9	514
2	CAG Repeat Not Polyglutamine Length Determines Timing of Huntington's Disease Onset. Cell, 2019, 178, 887-900.e14.	28.9	301
3	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
4	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
5	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
6	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
7	GAA repeat instability in Friedreich ataxia YAC transgenic mice. Genomics, 2004, 84, 301-310.	2.9	84
8	Patterns of CAG repeat instability in the central nervous system and periphery in Huntington's disease and in spinocerebellar ataxia type 1. Human Molecular Genetics, 2020, 29, 2551-2567.	2.9	69
9	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
10	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
11	Promotion of somatic CAG repeat expansion by Fan1 knock-out in Huntington's disease knock-in mice is blocked by Mlh1 knock-out. Human Molecular Genetics, 2020, 29, 3044-3053.	2.9	48
12	Pms2 Suppresses Large Expansions of the (GAA·TTC)n Sequence in Neuronal Tissues. PLoS ONE, 2012, 7, e47085.	2.5	43
13	Genetic Risk Underlying Psychiatric and Cognitive Symptoms in Huntington's Disease. Biological Psychiatry, 2020, 87, 857-865.	1.3	29
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	Heterozygous mutations cause genetic instability in a yeast model of cancer evolution. Nature, 2019, 566, 275-278.	27.8	27
16	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
17	Genetic Contributors to Intergenerational CAG Repeat Instability in Huntington's Disease Knock-In Mice. Genetics, 2017, 205, 503-516.	2.9	17
18	Assessing average somatic CAG repeat instability at the protein level. Scientific Reports, 2019, 9, 19152.	3.3	17

#	Article	IF	CITATIONS
19	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
20	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 Huntington's Disease. Journal of Huntington's Disease, 2021, 10, 149-163.	1.9	15
21	Histone deacetylase knockouts modify transcription, CAG instability and nuclear pathology in Huntington disease mice. ELife, 2020, 9, .	6.0	9