Maryam Neishabury

List of Publications by Year
in descending order

18	270	933447 10	996975 15
papers	citations	h-index	g-index

18	18	18	377
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	The First Case of BENTA Disease (B Cell Expansion with NF-Î³B and T Cell Anergy) from Iran. Journal of Clinical Immunology, 2021, 41, 811-813.	3.8	2
2	Novel variants in Iranian individuals suspected to have inherited red blood cell disorders, including bone marrow failure syndromes. Haematologica, 2020, 105, e1-e4.	3.5	3
3	Novel mutations in mitochondrial carrier family gene SLC25A38, causing congenital sideroblastic anemia in Iranian families, identified by whole exome sequencing. Blood Cells, Molecules, and Diseases, 2018, 71, 39-44.	1.4	7
4	The Impact ofXmnI-HBG2, BCL11Aand HBS1L-MYB Single Nucleotide Polymorphisms on Hb F Variation of Hematologically Normal Iranian Individuals. Hemoglobin, 2016, 40, 198-201.	0.8	0
5	Electromobility Shift Assay Reveals Evidence in Favor of Allele-Specific Binding of RUNX1 to the 5â€² Hypersensitive Site 4-Locus Control Region. Hemoglobin, 2016, 40, 236-239.	0.8	1
6	The influence of the BCL11A polymorphism on the phenotype of patients with beta thalassemia could be affected by the beta globin locus control region and/or the Xmn1-HBG2 genotypic background. Blood Cells, Molecules, and Diseases, 2013, 51, 80-84.	1.4	11
7	The modifying effect of Xmn1-HBG2 on thalassemic phenotype is associated with its linked elements in the beta globin locus control region, including the palindromic site at 5â€²HS4. Blood Cells, Molecules, and Diseases, 2012, 48, 1-5.	1.4	10
8	Commentary on â€œThe modifying effect of Xmn1-HBG2 on thalassemic phenotype is associated with its linked elements in the beta globin locus control region, including the palindromic site at 5â€² HS4â€•by M. Neishabury et al.. Blood Cells, Molecules, and Diseases, 2012, 48, 6.	1.4	5
9	The XmnI andBCL11ASingle Nucleotide Polymorphisms May Help Predict Hydroxyurea Response in Iranian Î²-Thalassemia Patients. Hemoglobin, 2012, 36, 371-380.	0.8	35
10	Analyzing 5â€²HS3 and 5â€²HS4 LCR core regions and NF-E2 in Iranian thalassemia intermedia patients with normal or carrier status for beta-globin mutations. Blood Cells, Molecules, and Diseases, 2011, 46, 201-205.	1.4	3
11	A REPORT OF 8 CASES WITH HEMOGLOBIN H DISEASE IN AN IRANIAN FAMILY. Pediatric Hematology and Oncology, 2010, 27, 405-412.	0.8	4
12	Frequency of Positive XmnI GÎ³ polymorphism and coinheritance of common alpha thalassemia mutations do not show statistically significant difference between thalassemia major and intermedia cases with homozygous IVSII-1 mutation. Blood Cells, Molecules, and Diseases, 2010, 44, 95-99.	1.4	20
13	Molecular Mechanisms Underlying Thalassemia Intermedia in Iran. Genetic Testing and Molecular Biomarkers, 2008, 12, 549-556.	1.7	21
14	The Iranian Human Mutation Gene Bank: A data and sample resource for worldwide collaborative genetics research. Human Mutation, 2003, 21, 146-150.	2.5	25
15	High Prevalence of the â^'Î±3.7Deletion Among Thalassemia Patients in Iran. Hemoglobin, 2003, 27, 53-55.	0.8	21
16	alpha-globin gene deletion and point mutation analysis among in Iranian patients with microcytic hypochromic anemia. Haematologica, 2003, 88, 1196-7.	3.5	19
17	Rare and unexpected mutations among Iranian beta-thalassemia patients and prenatal samples discovered by reverse-hybridization and DNA sequencing. Haematologica, 2002, 87, 1113-4.	3.5	26
18	Spontaneous mutation, oxidative DNA damage, and the roles of base and nucleotide excision repair in the yeastSaccharomyces cerevisiae. Yeast, 1999, 15, 205-218.	1.7	57