Samira Kalayinia

List of Publications by Year in descending order

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1478280 1058333 20 212 14 6 citations h-index g-index papers 20 20 20 266 docs citations times ranked citing authors all docs

#	Article	IF	CITATIONS
1	MicroRNAs: roles in cardiovascular development and disease. Cardiovascular Pathology, 2021, 50, 107296.	0.7	89
2	Next generation sequencing applications for cardiovascular disease. Annals of Medicine, 2018, 50, 91-109.	1.5	29
3	A novel de novo dominant mutation of <i>NOTCH1</i> gene in an Iranian family with nonâ€syndromic congenital heart disease. Journal of Clinical Laboratory Analysis, 2020, 34, e23147.	0.9	13
4	Association between the atrial natriuretic peptide rs5065 gene polymorphism and the presence and severity of coronary artery disease in an Iranian population. Coronary Artery Disease, 2014, 25, 242-246.	0.3	10
5	The impact of vascular endothelial growth factor +405 C/G polymorphism on long-term outcome and severity of coronary artery disease. Journal of Clinical Laboratory Analysis, 2017, 31, e22066.	0.9	10
6	A comprehensive in silico analysis, distribution and frequency of human Nkx2-5 mutations; A critical gene in congenital heart disease. Journal of Cardiovascular and Thoracic Research, 2019, 11, 287-299.	0.3	8
7	⟨i>GATA4⟨ i⟩ screening in Iranian patients of various ethnicities affected with congenital heart disease: Coâ€occurrence of a novel de novo translocation (5;7) and a likely pathogenic heterozygous ⟨i>GATA4⟨ i⟩ mutation in a family with autosomal dominant congenital heart disease. Journal of Clinical Laboratory Analysis. 2019, 33, e22923.	0.9	7
8	Chromosome 9 Inversion: Pathogenic or Benign? A Comprehensive Systematic Review of all Clinical Reports. Current Molecular Medicine, 2022, 22, 385-400.	0.6	7
9	Dilated cardiomyopathy caused by a pathogenic nucleotide variant in RBM20 in an Iranian family. BMC Medical Genomics, 2022, 15, 106.	0.7	7
10	Mosaic trisomy 22 in a 4â€yearâ€old boy with congenital heart disease and general hypotrophy: A case report. Journal of Clinical Laboratory Analysis, 2019, 33, e22663.	0.9	6
11	Whole-exome sequencing identified compound heterozygous variants in the <i>TTN</i> gene causing Salih myopathy with dilated cardiomyopathy in an Iranian family. Cardiology in the Young, 2022, 32, 1462-1467.	0.4	6
12	Extracellular matrix protein 1 gene (ECM1) mutations in nine Iranian families with lipoid proteinosis. Indian Journal of Medical Research, 2016, 143, 303.	0.4	5
13	Whole-exome sequencing reveals a rare missense variant in DTNA in an Iranian pedigree with early-onset atrial fibrillation. BMC Cardiovascular Disorders, 2022, 22, 37.	0.7	5
14	Whole-Exome Sequencing Reveals a Novel Mutation of <i>FLNA</i> Gene in an Iranian Family with Nonsyndromic Tetralogy of Fallot. Laboratory Medicine, 2021, 52, 614-618.	0.8	4
15	9P21.3 locus; An Important Region in Coronary Artery Disease: A Panel Approach to Investigation of the Coronary Artery Disease Etiology. International Journal of Cardiovascular Practice, 2019, 4, 21-35.	0.2	3
16	<i>In silico</i> analysis of <i>GATA4</i> variants demonstrates main contribution to congenital heart disease. Journal of Cardiovascular and Thoracic Research, 2021, 13, 336-354.	0.3	2
17	The association between inÂvitro fertilization and intracytoplasmic sperm injection treatment and the risk of congenital heart defects. Journal of Maternal-Fetal and Neonatal Medicine, 2022, 35, 7471-7485.	0.7	1
18	Whole-Exome Sequencing Revealed a Pathogenic Nonsense Variant in the <i> SLC19A2 < /i > Gene in an Iranian Family with Thiamine-Responsive Megaloblastic Anemia. Laboratory Medicine, 2022, 53, 640-650.</i>	0.8	0

#	Article	IF	CITATIONS
19	Whole-exome sequencing identified compound heterozygous variants in the TTN gene causing Salih myopathy with dilated cardiomyopathy in an Iranian family – CORRIGENDUM. Cardiology in the Young, 2022, 32, 1468-1468.	0.4	O
20	An Iranian Congenital Adrenal Hypoplasia Patient with Elevated Testosterone in Infancy due to a Novel Pathogenic Frameshift Variant in NROB1. International Journal of Endocrinology, 2021, 2021, 1-5.	0.6	0