Lilia M Iakoucheva

List of Publications by Year
in descending order

56 papers	16,500 citations	81743 39 h-index	149479 56 g-index
66 all docs	66 docs citations	66 times ranked	23515 citing authors

#	Article	IF	Citations
1	A phenotypic spectrum of autism is attributable to the combined effects of rare variants, polygenic risk and sex. Nature Genetics, 2022, 54, 1284-1292.	9.4	66
2	Autism-linked Cullin3 germline haploinsufficiency impacts cytoskeletal dynamics and cortical neurogenesis through RhoA signaling. Molecular Psychiatry, 2021, 26, 3586-3613.	4.1	26
3	Full-length isoform transcriptome of the developing human brain provides further insights into autism. Cell Reports, 2021, 36, 109631.	2.9	23
4	Cortical organoids model early brain development disrupted by 16p11.2 copy number variants in autism. Molecular Psychiatry, 2021, 26, 7560-7580.	4.1	61
5	Prioritizing de novo autism risk variants with calibrated gene- and variant-scoring models. Human Genetics, 2021, , 1.	1.8	1
6	Inferring the molecular and phenotypic impact of amino acid variants with MutPred2. Nature Communications, 2020, 11, 5918.	5.8	305
7	Getting to the Cores of Autism. Cell, 2019, 178, 1287-1298.	13.5	204
8	Oligogenic Effects of 16p11.2 Copy-Number Variation on Craniofacial Development. Cell Reports, 2019, 28, 3320-3328.e4.	2.9	34
9	Pathogenicity and functional impact of non-frameshifting insertion/deletion variation in the human genome. PLoS Computational Biology, 2019, 15, e1007112.	1.5	34
10	Paternally inherited cis-regulatory structural variants are associated with autism. Science, 2018, 360, 327-331.	6.0	174
11	Transcriptome-wide isoform-level dysregulation in ASD, schizophrenia, and bipolar disorder. Science, 2018, 362, .	6.0	805
12	Comprehensive Analyses of Tissue-Specific Networks with Implications to Psychiatric Diseases. Methods in Molecular Biology, 2017, 1613, 371-402.	0.4	5
13	When loss-of-function is loss of function: assessing mutational signatures and impact of loss-of-function genetic variants. Bioinformatics, 2017, 33, i389-i398.	1.8	53
14	Frequency and Complexity of De Novo Structural Mutation in Autism. American Journal of Human Genetics, 2016, 98, 667-679.	2.6	88
15	Widespread Expansion of Protein Interaction Capabilities by Alternative Splicing. Cell, 2016, 164, 805-817.	13.5	479
16	Spatiotemporal 16p11.2 Protein Network Implicates Cortical Late Mid-Fetal Brain Development and KCTD13-Cul3-RhoA Pathway in Psychiatric Diseases. Neuron, 2015, 85, 742-754.	3.8	139
17	Protein interaction network of alternatively spliced isoforms from brain links genetic risk factors for autism. Nature Communications, 2014, 5, 3650.	5.8	131
18	Predicted disorder-to-order transition mutations in Ĩº BÎ± disrupt function. Physical Chemistry Chemical Physics, 2014, 16, 6480.	1.3	21

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19	Pathological Unfoldomics of Uncontrolled Chaos: Intrinsically Disordered Proteins and Human Diseases. Chemical Reviews, 2014, 114, 6844-6879.	23.0	231
20	A Proteome-Scale Map of the Human Interactome Network. Cell, 2014, 159, 1212-1226.	13.5	1,199
21	Disease-Associated Mutations Disrupt Functionally Important Regions of Intrinsic Protein Disorder. PLoS Computational Biology, 2012, 8, e1002709.	1.5	123
22	Whole-Genome Sequencing in Autism Identifies Hot Spots for De Novo Germline Mutation. Cell, 2012, 151, 1431-1442.	13.5	501
23	A Protein Domain-Based Interactome Network for C.Âelegans Early Embryogenesis. Cell, 2012, 151, 1633.	13.5	4
24	Disease mutations in disordered regionsâ€”exception to the rule?. Molecular BioSystems, 2012, 8, 27-32.	2.9	93
25	Duplications of the neuropeptide receptor gene VIPR2 confer significant risk for schizophrenia. Nature, 2011, 471, 499-503.	13.7	296
26	Mapping copy number variation by population-scale genome sequencing. Nature, 2011, 470, 59-65.	13.7	991
27	Identification, analysis, and prediction of protein ubiquitination sites. Proteins: Structure, Function and Bioinformatics, 2010, 78, 365-380.	1.5	513
28	Graphlet Kernels for Prediction of Functional Residues in Protein Structures. Journal of Computational Biology, 2010, 17, 55-72.	0.8	44
29	LOSS OF POST-TRANSLATIONAL MODIFICATION SITES IN DISEASE. , 2009, , 337-347.		56
30	Unfoldomics of human diseases: linking protein intrinsic disorder with diseases. BMC Genomics, 2009, 10, S7.	1.2	236
31	Microduplications of 16p11.2 are associated with schizophrenia. Nature Genetics, 2009, 41, 1223-1227.	9.4	646
32	Prioritizing Disease Genes and Understanding Disease Pathways. , 2009, , 239-256.		0
33	RNA association or phosphorylation of the RS domain prevents aggregation of RS domain-containing proteins. Biochimica Et Biophysica Acta - General Subjects, 2008, 1780, 214-225.	1.1	34
34	A Protein Domain-Based Interactome Network for C. elegans Early Embryogenesis. Cell, 2008, 134, 534-545.	13.5	196
35	Functional Anthology of Intrinsic Disorder. 1. Biological Processes and Functions of Proteins with Long Disordered Regions. Journal of Proteome Research, 2007, 6, 1882-1898.	1.8	525
36	Functional Anthology of Intrinsic Disorder. 3. Ligands, Post-Translational Modifications, and Diseases Associated with Intrinsically Disordered Proteins. Journal of Proteome Research, 2007, 6, 1917-1932.	1.8	369

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37	Functional Anthology of Intrinsic Disorder. 2. Cellular Components, Domains, Technical Terms, Developmental Processes, and Coding Sequence Diversities Correlated with Long Disordered Regions. Journal of Proteome Research, 2007, 6, 1899-1916.	1.8	244
38	Intrinsic Disorder and Functional Proteomics. Biophysical Journal, 2007, 92, 1439-1456.	0.2	643
39	Prediction of Intrinsic Disorder and Its Use in Functional Proteomics. Methods in Molecular Biology, 2007, 408, 69-92.	0.4	37
40	Serine/arginine-rich splicing factors belong to a class of intrinsically disordered proteins. Nucleic Acids Research, 2006, 34, 305-312.	6.5	102
41	Intrinsic Disorder Is a Common Feature of Hub Proteins from Four Eukaryotic Interactomes. PLoS Computational Biology, 2006, 2, e100.	1.5	512
42	Two Sample Logo: a graphical representation of the differences between two sets of sequence alignments. Bioinformatics, 2006, 22, 1536-1537.	1.8	468
43	Flexible nets. The roles of intrinsic disorder in protein interaction networks. FEBS Journal, 2005, 272, 5129-5148.	2.2	1,052
44	DisProt: a database of protein disorder. Bioinformatics, 2005, 21, 137-140.	1.8	231
45	Combining prediction, computation and experiment for the characterization of protein disorder. Current Opinion in Structural Biology, 2004, 14, 570-576.	2.6	125
46	The importance of intrinsic disorder for protein phosphorylation. Nucleic Acids Research, 2004, 32, 1037-1049.	6.5	1,230
47	Order, Disorder, and Flexibility. Structure, 2003, 11, 1316-1317.	1.6	55
48	Intrinsic Disorder and Protein Functionâ€. Biochemistry, 2002, 41, 6573-6582.	1.2	1,605
49	Equilibrium and Stop-Flow Kinetic Studies of Fluorescently Labeled DNA Substrates with DNA Repair Proteins XPA and Replication Protein A. Biochemistry, 2002, 41, 131-143.	1.2	26
50	Intrinsic Disorder in Cell-signaling and Cancer-associated Proteins. Journal of Molecular Biology, 2002, 323, 573-584.	2.0	1,077
51	Single-Molecule Conformational Dynamics of Fluctuating Noncovalent DNAâ˜'Protein Interactions in DNA Damage Recognition. Journal of the American Chemical Society, 2001, 123, 9184-9185.	6.6	46
52	Identification of intrinsic order and disorder in the DNA repair protein XPA. Protein Science, 2001, 10, 560-571.	3.1	108
53	Aberrant mobility phenomena of the DNA repair protein XPA. Protein Science, 2001, 10, 1353-1362.	3.1	67
54	Nucleotide Excision Repair in Oocyte Nuclear Extracts from Xenopus laevis. Methods, 2000, 22, 188-193.	1.9	6

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55	Extended X-Ray Absorption Fine Structure Evidence for a Single Metal Binding Domain inXenopus laevisNucleotide Excision Repair Protein XPA. Biochemical and Biophysical Research Communications, 1999, 254, 109-113.	1.0	15
56	Oligogenic Effects of 16p11.2 Copy Number Variation on Craniofacial Development. SSRN Electronic Journal, 0, , .	0.4	1