Sarah L Shammas

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Binding and folding in transcriptional complexes. Current Opinion in Structural Biology, 2021, 66, 156-162.	5.7	12
2	Charge Interactions Modulate the Encounter Complex Ensemble of Two Differently Charged Disordered Protein Partners of KIX. Journal of Chemical Theory and Computation, 2020, 16, 3856-3868.	5.3	13
3	Hsp70 Inhibits the Nucleation and Elongation of Tau and Sequesters Tau Aggregates with High Affinity. ACS Chemical Biology, 2018, 13, 636-646.	3.4	96
4	Two Differential Binding Mechanisms of FG-Nucleoporins and Nuclear Transport Receptors. Cell Reports, 2018, 22, 3660-3671.	6.4	41
5	Stopped-Flow Kinetic Techniques for Studying Binding Reactions of Intrinsically Disordered Proteins. Methods in Enzymology, 2018, 611, 423-457.	1.0	2
6	Conserved Helix-Flanking Prolines Modulate Intrinsically Disordered Protein:Target Affinity by Altering the Lifetime of the Bound Complex. Biochemistry, 2017, 56, 2379-2384.	2.5	40
7	Mechanistic roles of protein disorder within transcription. Current Opinion in Structural Biology, 2017, 42, 155-161.	5 .7	56
8	Affinity of IDPs to their targets is modulated by ion-specific changes in kinetics and residual structure. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 9882-9887.	7.1	67
9	Phosphorylation of the IDP KID Modulates Affinity for KIX by Increasing the Lifetime of the Complex. Biophysical Journal, 2017, 113, 2706-2712.	0.5	22
10	pKID Binds to KIX via an Unstructured Transition State with Nonnative Interactions. Biophysical Journal, 2017, 113, 2713-2722.	0.5	33
11	Role of non-native electrostatic interactions in the coupled folding and binding of PUMA with Mcl-1. PLoS Computational Biology, 2017, 13, e1005468.	3.2	32
12	Insights into Coupled Folding and Binding Mechanisms from Kinetic Studies. Journal of Biological Chemistry, 2016, 291, 6689-6695.	3.4	141
13	GADIS: Algorithm for designing sequences to achieve target secondary structure profiles of intrinsically disordered proteins. Protein Engineering, Design and Selection, 2016, 29, 339-346.	2.1	26
14	A mechanistic model of tau amyloid aggregation based on direct observation of oligomers. Nature Communications, 2015, 6, 7025.	12.8	179
15	Plasticity of an Ultrafast Interaction between Nucleoporins and Nuclear Transport Receptors. Cell, 2015, 163, 734-745.	28.9	255
16	Transient misfolding dominates multidomain protein folding. Nature Communications, 2015, 6, 8861.	12.8	97
17	Allostery within a transcription coactivator is predominantly mediated through dissociation rate constants. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 12055-12060.	7.1	76
18	Mechanism of Assembly of the Non-Covalent Spectrin Tetramerization Domain from Intrinsically Disordered Partners. Journal of Molecular Biology, 2014, 426, 21-35.	4.2	31

#	Article	IF	CITATIONS
19	Interplay between partner and ligand facilitates the folding and binding of an intrinsically disordered protein. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 15420-15425.	7.1	144
20	Remarkably Fast Coupled Folding and Binding of the Intrinsically Disordered Transactivation Domain of cMyb to CBP KIX. Journal of Physical Chemistry B, 2013, 117, 13346-13356.	2.6	84
21	Perturbation of the Stability of Amyloid Fibrils through Alteration of Electrostatic Interactions. Biophysical Journal, 2011, 100, 2783-2791.	0.5	121
22	Binding of the Molecular Chaperone αB-Crystallin to Aβ Amyloid Fibrils Inhibits Fibril Elongation. Biophysical Journal, 2011, 101, 1681-1689.	0.5	143