## Harish Kumar

List of Publications by Year in descending order

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430874 526287 27 947 18 27 h-index citations g-index papers 28 28 28 1206 docs citations times ranked citing authors all docs

| #  | Article  | IF   | CITATIONS |
|----|--|------|-----------|
| 1  | Understanding the Kinetic Roles of the Inducer Heparin and of Rod-like Protofibrils during Amyloid Fibril Formation by Tau Protein. Journal of Biological Chemistry, 2011, 286, 38948-38959.                               | 3.4  | 122       |
| 2  | Salt-Induced Modulation of the Pathway of Amyloid Fibril Formation by the Mouse Prion Protein. Biochemistry, 2010, 49, 7615-7624.  | 2.5  | 101       |
| 3  | Evidence for the Existence of a Secondary Pathway for Fibril Growth during the Aggregation of Tau.<br>Journal of Molecular Biology, 2012, 421, 296-314.  | 4.2  | 57        |
| 4  | Development of the Structural Core and of Conformational Heterogeneity during the Conversion of Oligomers of the Mouse Prion Protein to Worm-like Amyloid Fibrils. Journal of Molecular Biology, 2012, 423, 217-231.       | 4.2  | 54        |
| 5  | Rational Stabilization of Helix 2 of the Prion Protein Prevents Its Misfolding and Oligomerization.<br>Journal of the American Chemical Society, 2014, 136, 16704-16707.   | 13.7 | 53        |
| 6  | Molecular Mechanism of the Misfolding and Oligomerization of the Prion Protein: Current Understanding and Its Implications. Biochemistry, 2015, 54, 4431-4442.   | 2.5  | 53        |
| 7  | Mechanistic Studies Unravel the Complexity Inherent in Tau Aggregation Leading to Alzheimer's<br>Disease and the Tauopathies. Biochemistry, 2013, 52, 4107-4126.   | 2.5  | 51        |
| 8  | Unfolding Rates of Barstar Determined in Native and Low Denaturant Conditions Indicate the Presence of Intermediates. Biochemistry, 2002, 41, 1568-1578.   | 2.5  | 48        |
| 9  | Partially Unfolded Forms of the Prion Protein Populated under Misfolding-promoting Conditions. Journal of Biological Chemistry, 2015, 290, 25227-25240.  | 3.4  | 42        |
| 10 | Mechanism of aggregation and membrane interactions of mammalian prion protein. Biochimica Et Biophysica Acta - Biomembranes, 2018, 1860, 1927-1935.  | 2.6  | 37        |
| 11 | Unraveling the Molecular Mechanism of pH-Induced Misfolding and Oligomerization of the Prion Protein. Journal of Molecular Biology, 2016, 428, 1345-1355.  | 4.2  | 36        |
| 12 | Resonance Raman Spectroscopic Measurements Delineate the Structural Changes that Occur during Tau Fibril Formation. Biochemistry, 2014, 53, 6550-6565.   | 2.5  | 34        |
| 13 | Structural Effects of Multiple Pathogenic Mutations Suggest a Model for the Initiation of Misfolding of the Prion Protein. Angewandte Chemie - International Edition, 2015, 54, 7529-7533.                                 | 13.8 | 34        |
| 14 | NMR Identification and Characterization of the Flexible Regions in the 160 kDa Molten Globule-Like Aggregate of Barstar at Low pHâ€. Biochemistry, 2002, 41, 9885-9899.  | 2.5  | 29        |
| 15 | Modulation of the extent of structural heterogeneity in α-synuclein fibrils by the small molecule thioflavin T. Journal of Biological Chemistry, 2017, 292, 16891-16903.   | 3.4  | 28        |
| 16 | Salt-Mediated Oligomerization of the Mouse Prion Protein Monitored by Real-Time NMR. Journal of Molecular Biology, 2017, 429, 1852-1872.   | 4.2  | 26        |
| 17 | Pathogenic Mutations within the Disordered Palindromic Region of the Prion Protein Induce<br>Structure Therein and Accelerate the Formation of Misfolded Oligomers. Journal of Molecular<br>Biology, 2016, 428, 3935-3947. | 4.2  | 21        |
| 18 | The Pathogenic Mutation T182A Converts the Prion Protein into a Molten Globule-like Conformation Whose Misfolding to Oligomers but Not to Fibrils Is Drastically Accelerated. Biochemistry, 2016, 55, 459-469.             | 2.5  | 20        |

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|----|---|--------------|-----------|
| 19 | The G126V Mutation in the Mouse Prion Protein Hinders Nucleation-Dependent Fibril Formation by Slowing Initial Fibril Growth and by Increasing the Critical Concentration. Biochemistry, 2017, 56, 5931-5942. | 2.5          | 20        |
| 20 | Structural mechanisms of oligomer and amyloid fibril formation by the prion protein. Chemical Communications, 2018, 54, 6230-6242.  | 4.1          | 20        |
| 21 | Mechanistic and Structural Origins of the Asymmetric Barrier to Prion-like Cross-Seeding between Tau-3R and Tau-4R. Journal of Molecular Biology, 2018, 430, 5304-5312.                                       | 4.2          | 18        |
| 22 | Ruggedness in the Free Energy Landscape Dictates Misfolding of the Prion Protein. Journal of Molecular Biology, 2019, 431, 807-824.   | 4.2          | 16        |
| 23 | Mechanistic approaches to understand the prion-like propagation of aggregates of the human tau protein. Biochimica Et Biophysica Acta - Proteins and Proteomics, 2019, 1867, 922-932.                         | 2.3          | 8         |
| 24 | Destabilization of polar interactions in the prion protein triggers misfolding and oligomerization. Protein Science, 2021, 30, 2258-2271.   | 7.6          | 5         |
| 25 | The Lys 280 â†' Gln mutation mimicking diseaseâ€linked acetylation of Lys 280 in tau extends the struct core of fibrils and modulates their catalytic properties. Protein Science, 2021, 30, 785-803.         | tural<br>7.6 | 4         |
| 26 | Microsecond Dynamics During the Binding-induced Folding of an Intrinsically Disordered Protein. Journal of Molecular Biology, 2021, 433, 167254.  | 4.2          | 3         |
| 27 | Elongation of Fibrils Formed by a Tau Fragment is Inhibited by a Transient Dimeric Intermediate. Journal of Physical Chemistry B, 2022, 126, 3385-3397.   | 2.6          | 1         |