

BIBLIOGRAPHY

BIBLIOGRAPHY

- [1] Ambrose, C.T. A therapeutic approach for senile dementias: neuroangiogenesis. *Journal of Alzheimer's Disease*, 43(1): 1-17, 2015.
- [2] Satizabal, C.L., Beiser, A.S., Chouraki, V., Chêne, G., Dufouil, C., and Seshadri, S. Incidence of dementia over three decades in the Framingham Heart Study. *New England Journal of Medicine*, 374(6): 523-532, 2016.
- [3] Selkoe, D.J. and Hardy, J. The amyloid hypothesis of Alzheimer's disease at 25 years. *EMBO molecular medicine*, 8(6): 595-608, 2016.
- [4] Wilson, E.N., Abela, A.R., Do Carmo, S., Allard, S., Marks, A.R., Welikovitch, L.A., and Cuello, A.C. Intraneuronal amyloid beta accumulation disrupts hippocampal CRTC1-dependent gene expression and cognitive function in a rat model of Alzheimer disease. *Cerebral Cortex*, 27(2): 1501-1511, 2017.
- [5] Kuruva, C.S. and Reddy, P.H. Amyloid beta modulators and neuroprotection in Alzheimer's disease: a critical appraisal. *Drug discovery today*, 22(2): 223-233, 2017.
- [6] Retrieved on 15 Aug. 2017 from <http://www.alz.org/facts/>
- [7] Retrieved on 25 Aug. 2017 from <http://www.nytimes.com/2010/06/02/health/02alzheimers.html>
- [8] Zhou, Z.D., Chan, C.H., Ma, Q.H., Xu, X.H., Xiao, Z.C., and Tan, E.K. The roles of amyloid precursor protein (APP) in neurogenesis: Implications to pathogenesis and therapy of Alzheimer disease. *Cell adhesion & migration*, 5(4): 280-292, 2011.
- [9] Tabaton, M., Zhu, X., Perry, G., Smith, M.A., and Giliberto, L. Signaling effect of amyloid- β 42 on the processing of A β PP. *Experimental neurology*, 221(1): 18-25, 2010.
- [10] Zou, K., Gong, J. S., Yanagisawa, K., and Michikawa, M. A novel functions of monomeric amyloid β -protein serving as an antioxidant molecule against metal-induced oxidative damage. *Journal of Neuroscience*, 22(12): 4833-4841, 2002.
- [11] Igba, U., Sun, G.Y., Weisman, G.A., He, Y., and Wood, W.G. Amyloid β -protein stimulates trafficking of cholesterol and caveolin-1 from the plasma membrane to the Golgi complex in mouse primary astrocytes. *Neuroscience*, 162(2): 328-338, 2009.

BIBLIOGRAPHY

- [12] Stewart, K.L. and Radford, S.E. Amyloid plaques beyond A β : a survey of the diverse modulators of amyloid aggregation. *Biophysical Reviews*, 9(35): 1-15, 2017.
- [13] Pickett, E.K., Koffie, R.M., Wegmann, S., Henstridge, C.M., Herrmann, A.G., Colom-Cadena, M., and Walsh, D.M. Non-fibrillar oligomeric amyloid- β within synapses. *Journal of Alzheimer's Disease*, 53(3): 787-800, 2016.
- [14] Anfinsen, C.B. Principles that govern the folding of protein chains. *Science*, 181(4096): 223-230, 1973.
- [15] Hershko, A. and Ciechanover, A. The ubiquitin system. *Annual review of biochemistry*, 67(1): 425-479, 1998.
- [16] Serpell, L.C., Sunde, M., and Blake, C.C. The molecular basis of amyloidosis. *Cellular and Molecular Life Sciences*, 53(11): 871-887, 1997.
- [17] Fitzpatrick, A.W., Debelouchina, G.T., Bayro, M. J., Clare, D.K., Caporini, M. A., Bajaj, V.S., and MacPhee, C.E. Atomic structure and hierarchical assembly of a cross- β amyloid fibril. *Proceedings of the National Academy of Sciences*, 110(14): 5468-5473, 2013.
- [18] Westermark, P., Benson, M.D., Buxbaum, J.N., Cohen, A.S., Frangione, B., Ikeda, S.I., and Sipe, J.D. A primer of amyloid nomenclature. *Amyloid*, 14(3): 179-183, 2007.
- [19] Kumar, S. and Walter, J. Phosphorylation of amyloid beta (A β) peptides—A trigger for formation of toxic aggregates in Alzheimer's disease. *Aging (Albany NY)*, 3(8): 803-812, 2011.
- [20] Mathuranath, P. S., George, A., Ranjith, N., Justus, S., Kumar, M. S., Menon, R., and Verghese, J. Incidence of Alzheimer's disease in India: A 10 years follow-up study. *Neurology India*, 60(6): 625-630, 2012.
- [21] Van Cauwenbergh, C., Van Broeckhoven, C., and Sleegers, K. The genetic landscape of Alzheimer disease: clinical implications and perspectives. *Genetics in Medicine*, 18(5): 421-430, 2016.
- [22] Bateman, R.J., Xiong, C., Benzinger, T.L., Fagan, A.M., Goate, A., Fox, N.C., and Holtzman, D.M. Clinical and biomarker changes in dominantly inherited Alzheimer's disease. *The New England Journal of Medicine*, 2012(367): 795-804, 2012.
- [23] Braak, H. and Braak, E. Frequency of stages of Alzheimer-related lesions in different age categories. *Neurobiology of aging*, 18(4): 351-357, 1997.

BIBLIOGRAPHY

- [24] Braak, H. and Braak, E. Neuropathological stageing of Alzheimer-related changes. *Acta neuropathologica*, 82(4): 239-259, 1991.
- [25] Khachaturian, Z.S. Diagnosis of Alzheimer's disease. *Archives of neurology*, 42(11): 1097-1105, 1985.
- [26] Alzheimer, A. Über einen eigenartigen schweren Erkrankungsprozeß der Hirnrinde. *Neurologisches Centralblatt*, 23: 1129–1136, 1906.
- [27] Blessed, G., Tomlinson, B.E. and Roth, M. The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. *The British Journal of Psychiatry*, 114 (512): 797-811, 1968.
- [28] Grachev, I.D. Alzheimer's Disease Dementia, Amyloid Imaging and Underpinning Fibrillar A β Plaque Associated Pathology: Are They Aligned. *Journal of Neurology & Stroke*, 2(2): 00050, 2015.
- [29] Kay, D.W.K., Beamish, P. and Roth, M. Old age mental disorders in Newcastle upon Tyne. *The British Journal of Psychiatry*, 110(468): 668-682, 1964.
- [30] Tanzi, R., Kovacs, D., Kim, T., Moir, K., Guenette, S., and Wasco, W. The gene defects responsible for familial Alzheimer's disease. *Neurobiology of Disease*, 3(3): 159-168, 1996.
- [31] Glenner, G.G. and Wong, C.W. Alzheimer's disease and Down's syndrome: sharing of a unique cerebrovascular amyloid fibril protein. *Biochemical and biophysical research communications*, 122(3): 1131-1135, 1984.
- [32] Ihara, Y., Nukina, N., Miura, R., and Ogawara, M. Phosphorylated tau protein is integrated into paired helical filaments in Alzheimer's disease. *The Journal of Biochemistry*, 99(6): 1807-1810, 1986.
- [33] Sperling, R.A., Aisen, P. S., Beckett, L.A., Bennett, D.A., Craft, S., Fagan, A. M., and Park, D.C. Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & dementia*, 7(3): 280-292, 2011.
- [34] Retrieved on 27 Jul. 2017 from <http://www.brightfocus.org/alzheimers/infographic/amyloid-plaques-and-neurofibrillary-tangles>.

BIBLIOGRAPHY

- [35] Van Cauwenbergh, C., Van Broeckhoven, C., and Sleegers, K. The genetic landscape of Alzheimer disease: clinical implications and perspectives. *Genetics in Medicine*, 18(5): 421-430, 2016.
- [36] Gomar, J.J., Conejero-Goldberg, C., Huey, E.D., Davies, P., Goldberg, T.E., and Alzheimer's Disease Neuroimaging Initiative. Lack of neural compensatory mechanisms of BDNF val66met met carriers and APOE E4 carriers in healthy aging, mild cognitive impairment, and Alzheimer's disease. *Neurobiology of aging*, 39: 165-173, 2016.
- [37] Veugelen, S., Saito, T., Saido, T.C., Chávez-Gutiérrez, L., and De Strooper, B. Familial Alzheimer's disease mutations in Presenilin generate Amyloidogenic A β peptide seeds. *Neuron*, 90(2): 410-416, 2016.
- [38] Kuller, L.H., Lopez, O.L., Mackey, R.H., Rosano, C., Edmundowicz, D., Becker, J.T., and Newman, A.B. Subclinical cardiovascular disease and death, dementia, and coronary heart disease in patients 80+ years. *Journal of the American College of Cardiology*, 67(9): 1013-1022, 2016.
- [39] Altman, R. and Rutledge, J.C. The vascular contribution to Alzheimer's disease. *Clinical science*, 119(10): 407-421, 2010.
- [40] Nazareth, A.M.D. Type 2 diabetes mellitus in the pathophysiology of Alzheimer's disease. *Dementia & Neuropsychologia*, 11(2): 105-113, 2017.
- [41] Kandimalla, R., Thirumala, V., and Reddy, P. H. Is Alzheimer's disease a type 3 diabetes? A critical appraisal. *Biochimica et Biophysica Acta (BBA)-Molecular Basis of Disease*, 1863(5): 1078-1089, 2017.
- [42] Kroner, Z. The relationship between Alzheimer's disease and diabetes: Type 3 diabetes. *Alternative Medicine Review*, 14(4): 373-379, 2009.
- [43] Beeri, M.S., Schmeidler, J., Silverman, J.M., Gandy, S., Wysocki, M., Hannigan, C.M., and Haroutunian, V. Insulin in combination with other diabetes medication is associated with less Alzheimer neuropathology. *Neurology*, 71(10): 750-757, 2008.
- [44] Coulthard, E. and Knight, M. Refining Alzheimer's disease diagnosis with MRI. *Brain*, 140(3): 524-526, 2017.
- [45] Johnson, K.A., Schultz, A., Betensky, R.A., Becker, J.A., Sepulcre, J., Rentz, D., and Marshall, G. Tau positron emission tomographic imaging in aging and early Alzheimer disease. *Annals of neurology*, 79(1): 110-119, 2016.

BIBLIOGRAPHY

- [46] Retrieved on 29 Jul. 2017 from <https://www.nia.nih.gov/health/alzheimers/causes>.
- [47] Rozpędek, W., Nowak, A., Pytel, D., Lewko, D., Diehl, J.A., and Majsterek, I. The role of the Amyloid Precursor Protein mutations and PERK-dependent signaling pathways in the pathogenesis of Alzheimer's disease. *Folia Biologica et Oecologica*, 12(1): 48-59, 2016.
- [48] Marik, S.A., Olsen, O., Tessier-Lavigne, M., and Gilbert, C.D. Physiological role for amyloid precursor protein in adult experience-dependent plasticity. *Proceedings of the National Academy of Sciences*, 113(28): 7912-7917, 2016.
- [49] Nixon, R.A. Amyloid precursor protein and endosomal–lysosomal dysfunction in Alzheimer's disease: inseparable partners in a multifactorial disease. *The FASEB Journal*, 31(7): 2729-2743, 2017.
- [50] Allsop, D., Landon, M., and Kidd, M. The isolation and amino acid composition of senile plaque core protein. *Brain research*, 259(2): 348-352, 1983.
- [51] Hardy, J.A. and Higgins, G.A. Alzheimer's disease: the amyloid cascade hypothesis. *Science*, 256(5054): 184, 1992.
- [52] Selkoe, D.J. The molecular pathology of Alzheimer's disease. *Neuron*, 6(4): 487-498, 1991.
- [53] Hardy, J. and Allsop, D. Amyloid deposition as the central event in the aetiology of Alzheimer's disease. *Trends in pharmacological sciences*, 12(10): 383-388, 1991.
- [54] Chow, V.W., Mattson, M. P., Wong, P.C., and Gleichmann, M. An overview of APP processing enzymes and products. *Neuromolecular medicine*, 12(1): 1-12, 2010.
- [55] Zhang, Y., McLaughlin, R., Goodyer, C., and LeBlanc, A. Selective cytotoxicity of intracellular amyloid β peptide1–42 through p53 and Bax in cultured primary human neurons. *The Journal of cell biology*, 156(3): 519-529, 2002.
- [56] Liu, L., Niu, L., Xu, M., Han, Q., Duan, H., Dong, M., and Yang, Y. Molecular tethering effect of C-terminus of amyloid peptide A β 42. *ACS nano*, 8(9): 9503-9510, 2014.

BIBLIOGRAPHY

- [57] Chen, Q.S., Kagan, B.L., Hirakura, Y., and Xie, C.W. Impairment of hippocampal long-term potentiation by Alzheimer amyloid β -peptides. *Journal of neuroscience research*, 60(1): 65-72, 2000.
- [58] Miñano-Molina, A. J., España, J., Martín, E., Barneda-Zahonero, B., Fadó, R., Solé, M., and Rodríguez-Alvarez, J. Soluble oligomers of amyloid- β peptide disrupt membrane trafficking of α -amino-3-hydroxy-5-methylisoxazole-4-propionic acid receptor contributing to early synapse dysfunction. *Journal of Biological Chemistry*, 286(31): 27311-27321, 2011.
- [59] Caccamo, A., Shepherd, J. D., Murphy, M. P., Golde, T. E., Kayed, R., Metherate, R., and LaFerla, F. M. Triple-transgenic model of Alzheimer's Disease with plaques and tangles. *Neuron*, 39(3): 409-421, 2003.
- [60] Tu, S., Okamoto, S. I., Lipton, S. A., and Xu, H. Oligomeric A β -induced synaptic dysfunction in Alzheimer's disease. *Molecular neurodegeneration*, 9(1): 48, 2014.
- [61] Côté, S., Laghaei, R., Derreumaux, P., and Mousseau, N. Distinct dimerization for various alloforms of the amyloid-beta protein: A β 1-40, A β 1-42, and A β 1-40 (d23n). *The journal of physical chemistry B*, 116(13): 4043-4055, 2012.
- [62] Knowles, T. P., Vendruscolo, M., and Dobson, C. M. The amyloid state and its association with protein misfolding diseases. *Nature reviews. Molecular cell biology*, 15(6): 384-396, 2014.
- [63] Zhang, S., Iwata, K., Lachenmann, M. J., Peng, J. W., Li, S., Stimson, E. R., and Lee, J. P. The Alzheimer's peptide A β adopts a collapsed coil structure in water. *Journal of structural biology*, 130(2): 130-141, 2000.
- [64] Cruz, L., Rao, J. S., Teplow, D. B., and Urbanc, B. Dynamics of metastable β -hairpin structures in the folding nucleus of amyloid β -protein. *The Journal of Physical Chemistry B*, 116(22): 6311-6325, 2012.
- [65] Rosenman, D. J., Connors, C. R., Chen, W., Wang, C., and García, A. E. A β monomers transiently sample oligomer and fibril-like configurations: ensemble characterization using a combined MD/NMR approach. *Journal of molecular biology*, 425(18): 3338-3359, 2013.
- [66] Nasica-Labouze, J., Nguyen, P. H., Sterpone, F., Berthoumieu, O., Buchete, N. V., Coté, S., and Laio, A. Amyloid β protein and Alzheimer's

BIBLIOGRAPHY

- disease: When computer simulations complement experimental studies. *Chemical reviews*, 115(9): 3518-3563, 2015.
- [67] Nagarajan, S., Rajadas, J., and Malar, E. P. Density functional theory analysis and spectral studies on amyloid peptide A β (28–35) and its mutants A30G and A30I. *Journal of structural biology*, 170(3): 439-450, 2010.
- [68] Rosenman, D. J., Wang, C., and García, A. E. Characterization of A β monomers through the convergence of ensemble properties among simulations with multiple force fields. *The Journal of Physical Chemistry B*, 120(2): 259-277, 2015.
- [69] Komatsu, H. and Axelsen, P. H. Amyloid Beta-Protein Fibrils from Human Alzheimer's Brain Tissue and from Mouse Models of Alzheimer's Differ in Structures. *Biophysical Journal*, 110(3): 219a, 2016.
- [70] Lyubchenko, Y. L. Amyloid misfolding, aggregation, and the early onset of protein deposition diseases: insights from AFM experiments and computational analyses. *AIMS molecular science*, 2(3): 190, 2015.
- [71] Nichols, M. R., Colvin, B. A., Hood, E. A., Paranjape, G. S., Osborn, D. C., and Terrill-Usery, S. E. Biophysical comparison of soluble amyloid- β (1–42) protofibrils, oligomers, and protofilaments. *Biochemistry*, 54(13): 2193-2204, 2015.
- [72] DaRocha-Souto, B., Scotton, T. C., Coma, M., Serrano-Pozo, A., Hashimoto, T., Serenó, L., and Gómez-Isla, T. Brain oligomeric β -amyloid but not total amyloid plaque burden correlates with neuronal loss and astrocyte inflammatory response in amyloid precursor protein/tau transgenic mice. *Journal of Neuropathology & Experimental Neurology*, 70(5): 360-376, 2011.
- [73] Schmidt, M., Rohou, A., Lasker, K., Yadav, J. K., Schiene-Fischer, C., Fändrich, M., and Grigorieff, N. Peptide dimer structure in an A β (1–42) fibril visualized with cryo-EM. *Proceedings of the National Academy of Sciences*, 112(38): 11858-11863, 2015.
- [74] Rousseau, F., Schymkowitz, J., and Serrano, L. Protein aggregation and amyloidosis: confusion of the kinds. *Current opinion in structural biology*, 16(1): 118-126, 2006.

BIBLIOGRAPHY

- [75] Eskici, G. and Axelsen, P. H. Amyloid Beta Peptide Folding in Reverse Micelles. *Journal of the American Chemical Society*, 139(28): 9566-9575, 2017.
- [76] Hayden, E. Y. and Teplow, D. B. Amyloid β -protein oligomers and Alzheimer's disease. *Alzheimer's research & therapy*, 5(6): 60, 2013.
- [77] Okamoto, A., Yano, A., Nomura, K., Higai, S. I., and Kurita, N. Effect of D23N mutation on the dimer conformation of amyloid β -proteins: Ab initio molecular simulations in water. *Journal of Molecular Graphics and Modelling*, 50: 113-124, 2014.
- [78] Šarić, A., Chebaro, Y. C., Knowles, T. P., and Frenkel, D. Crucial role of nonspecific interactions in amyloid nucleation. *Proceedings of the National Academy of Sciences*, 111(50): 17869-17874, 2014.
- [79] Itoh, S. G. and Okumura, H. Dimerization Process of Amyloid- β (29–42) Studied by the Hamiltonian Replica-Permutation Molecular Dynamics Simulations. *The Journal of Physical Chemistry B*, 118(39): 11428-11436, 2014.
- [80] Shigemitsu, Y., Iwaya, N., Goda, N., Matsuzaki, M., Tenno, T., Narita, A., and Hiroaki, H. Nuclear magnetic resonance evidence for the dimer formation of beta amyloid peptide 1-42 in 1,1,1,3,3,3-hexafluoro-2-propanol. *Analytical biochemistry*, 498: 59-67, 2016.
- [81] Tarus, B., Tran, T. T., Nasica-Labouze, J., Sterpone, F., Nguyen, P. H., and Derreumaux, P. Structures of the Alzheimer's wild-type A β 1-40 dimer from atomistic simulations. *The Journal of Physical Chemistry B*, 119(33): 10478-10487, 2015.
- [82] Barz, B. and Urbanc, B. Dimer formation enhances structural differences between amyloid β -protein (1–40) and (1–42): an explicit-solvent molecular dynamics study. *PloS one*, 7(4): e34345 2012.
- [83] Zhu, X., Bora, R. P., Barman, A., Singh, R., and Prabhakar, R. Dimerization of the full-length Alzheimer amyloid β -peptide (A β 42) in explicit aqueous solution: A molecular dynamics study. *The Journal of Physical Chemistry B*, 116(15): 4405-4416, 2012.
- [84] Buée, L., Bussière, T., Buée-Scherrer, V., Delacourte, A., and Hof, P. R. Tau protein isoforms, phosphorylation and role in neurodegenerative disorders. *Brain Research Reviews*, 33(1): 95-130, 2000.

BIBLIOGRAPHY

- [85] Spires-Jones, T. L. and Hyman, B. T. The intersection of amyloid beta and tau at synapses in Alzheimer's disease. *Neuron*, 82(4): 756-771, 2014.
- [86] Spillantini, M. G. and Goedert, M. Tau pathology and neurodegeneration. *The Lancet Neurology*, 12(6): 609-622, 2013.
- [87] Zempel, H. and Mandelkow, E. Lost after translation: missorting of Tau protein and consequences for Alzheimer disease. *Trends in neurosciences*, 37(12): 721-732, 2014.
- [88] Götz, J., Chen, F., Van Dorpe, J., and Nitsch, R. M. Formation of neurofibrillary tangles in P301L tau transgenic mice induced by A β 42 fibrils. *Science*, 293(5534): 1491-1495, 2001.
- [89] Bloom, G. S. Amyloid- β and tau: the trigger and bullet in Alzheimer disease pathogenesis. *JAMA neurology*, 71(4): 505-508, 2014.
- [90] Héraud, C., Goufak, D., Ando, K., Leroy, K., Suain, V., Yilmaz, Z., and Brion, J. P. Increased misfolding and truncation of tau in APP/PS1/tau transgenic mice compared to mutant tau mice. *Neurobiology of disease*, 62: 100-112, 2014.
- [91] Bennett, R. E., DeVos, S. L., Dujardin, S., Corjuc, B., Gor, R., Gonzalez, J., and Hyman, B. T. Enhanced Tau Aggregation in the Presence of Amyloid β . *The American journal of pathology*, 2017. DOI:10.1016/j.ajpath.2017.03.011.
- [92] Pianu, B., Lefort, R., Thuiliere, L., Tabourier, E., and Bartolini, F. The A β 1-42 peptide regulates microtubule stability independently of tau. *Journal of Cell Science*, 127(5): 1117-1127, 2014.
- [93] Blurton-Jones, M. and LaFerla, F. M. Pathways by which A β facilitates tau pathology. *Current Alzheimer Research*, 3(5): 437-448, 2006.
- [94] LaFerla, F. M. Pathways linking A β and tau pathologies. *Biochemical Society Transactions*, 38(4):993-995, 2010.
- [95] Jost, K., Varga, J., Pence, B., and Zarándi, M. In Vitro Degradation Of beta-Amyloid [25-35] Peptide. *Protein and Peptide Letters*, 8(6): 423-428, 2001.
- [96] Takashima, A., Honda, T., Yasutake, K., Michel, G., Murayama, O., Murayama, M., and Yamaguchi, H. Activation of tau protein kinase I/glycogen synthase kinase-3 β by amyloid β peptide (25–35) enhances phosphorylation of tau in hippocampal neurons. *Neuroscience research*, 31(4): 317-323, 1998.

BIBLIOGRAPHY

- [97] Miller, Y., Ma, B., and Nussinov, R. Synergistic interactions between repeats in tau protein and A β amyloids may be responsible for accelerated aggregation via polymorphic states. *Biochemistry*, 50(23): 5172-5181, 2011.
- [98] Wei, G., Jewett, A. I., and Shea, J. E. Structural diversity of dimers of the Alzheimer amyloid- β (25–35) peptide and polymorphism of the resulting fibrils. *Physical Chemistry Chemical Physics*, 12(14): 3622-3629, 2010.
- [99] Larini, L., Gessel, M. M., LaPointe, N. E., Do, T. D., Bowers, M. T., Feinstein, S. C., and Shea, J. E. Initiation of assembly of tau (273-284) and its Δ K280 mutant: An experimental and computational study. *Physical Chemistry Chemical Physics*, 15(23): 8916-8928, 2013.
- [100] Larini, L. and Shea, J. E. Role of β -hairpin formation in aggregation: The self-assembly of the amyloid- β (25–35) peptide. *Biophysical journal*, 103(3): 576-586, 2012.
- [101] Bleiholder, C., Do, T. D., Wu, C., Economou, N. J., Bernstein, S. S., Buratto, S. K., and Bowers, M. T. Ion mobility spectrometry reveals the mechanism of amyloid formation of A β (25–35) and its modulation by inhibitors at the molecular level: epigallocatechin gallate and scyllo-inositol. *Journal of the American Chemical Society*, 135(45): 16926-16937, 2013.
- [102] Oldfield, C. J. and Dunker, A. K. Intrinsically disordered proteins and intrinsically disordered protein regions. *Annual review of biochemistry*, 83: 553-584, 2014.
- [103] Van Der Lee, R., Buljan, M., Lang, B., Weatheritt, R. J., Daughdrill, G. W., Dunker, A. K., and Kim, P. M. Classification of intrinsically disordered regions and proteins. *Chemical reviews*, 114(13): 6589-6631, 2014.
- [104] Wright, P. E. and Dyson, H. J. Intrinsically unstructured proteins: re-assessing the protein structure-function paradigm. *Journal of molecular biology*, 293(2): 321-331, 1999.
- [105] Xie, H., Vucetic, S., Iakoucheva, L. M., Oldfield, C. J., Dunker, A. K., Uversky, V. N., and Obradovic, Z. Functional anthology of intrinsic disorder. 1. Biological processes and functions of proteins with long disordered regions. *Journal of proteome research*, 6(5): 1882-1898, 2007.

BIBLIOGRAPHY

- [106] Uversky, V. N., Oldfield, C. J., and Dunker, A. K. Intrinsically disordered proteins in human diseases: introducing the D2 concept. *Annual Review of Biophysics*, 37: 215-246, 2008.
- [107] Metallo, S. J. Intrinsically disordered proteins are potential drug targets. *Current opinion in chemical biology*, 14(4): 481-488, 2010.
- [108] Tsolis, A. C., Papandreou, N. C., Iconomidou, V. A., and Hamodrakas, S. J. A consensus method for the prediction of ‘aggregation-prone’ peptides in globular proteins. *PloS one*, 8(1): e54175, 2013.
- [109] Linding, R., Jensen, L. J., Diella, F., Bork, P., Gibson, T. J., and Russell, R. B. Protein disorder prediction: implications for structural proteomics. *Structure*, 11(11): 1453-1459, 2003.
- [110] Leahy, C. T., Murphy, R. D., Hummer, G., Rosta, E., and Buchete, N. V. Coarse master equations for binding kinetics of amyloid peptide dimers. *The journal of physical chemistry letters*, 7(14): 2676-2682, 2016.
- [111] Hefti, F., Goure, W. F., Jerecic, J., Iverson, K. S., Walicke, P. A., and Krafft, G. A. The case for soluble A β oligomers as a drug target in Alzheimer's disease. *Trends in pharmacological sciences*, 34(5): 261-266, 2013.
- [112] Ferreira, S. T., Lourenco, M. V., Oliveira, M. M., and De Felice, F. G. Soluble amyloid- β oligomers as synaptotoxins leading to cognitive impairment in Alzheimer's disease. *Frontiers in cellular neuroscience*, 9:191, 2015.
- [113] Salahuddin, P., Fatima, M. T., Abdelhameed, A. S., Nusrat, S., and Khan, R. H. Structure of amyloid oligomers and their mechanisms of toxicities: targeting amyloid oligomers using novel therapeutic approaches. *European Journal of medicinal chemistry*, 114:41-58, 2016.
- [114] Tarczyluk, M. A., Nagel, D. A., Parri, H. R., Tse, E. H., Brown, J. E., Coleman, M. D., and Hill, E. J. Amyloid β 1-42 induces hypometabolism in human stem cell-derived neuron and astrocyte networks. *Journal of Cerebral Blood Flow & Metabolism*, 35(8): 1348-1357, 2015.
- [115] Kakio, A., Nishimoto, S. I., Yanagisawa, K., Kozutsumi, Y., and Matsuzaki, K. Interactions of amyloid β -protein with various gangliosides in raft-like membranes: importance of GM1 ganglioside-bound form as an endogenous seed for Alzheimer amyloid. *Biochemistry*, 41(23): 7385-7390, 2002.

BIBLIOGRAPHY

- [116] Breydo, L., Kuroski, D., Rasool, S., Milton, S., Wu, J. W., Uversky, V. N., and Glabe, C. G. Structural differences between amyloid beta oligomers. *Biochemical and biophysical research communications*, 477(4): 700-705, 2016.
- [117] Huang, D., Zimmerman, M. I., Martin, P. K., Nix, A. J., Rosenberry, T. L., and Paravastu, A. K. Antiparallel β -sheet structure within the C-terminal region of 42-residue Alzheimer's amyloid- β peptides when they form 150-kDa oligomers. *Journal of molecular biology*, 427(13): 2319-2328, 2015.
- [118] Yu, X. and Zheng, J. Polymorphic structures of Alzheimer's β -amyloid globulomers. *PloS one*, 6(6): e20575, 2011.
- [119] Stroud, J. C., Liu, C., Teng, P. K., and Eisenberg, D. Toxic fibrillar oligomers of amyloid- β have cross- β structure. *Proceedings of the National Academy of Sciences*, 109(20): 7717-7722, 2012.
- [120] Kuperstein, I., Broersen, K., Benilova, I., Rozenski, J., Jonckheere, W., Debulpaep, M., and Braeken, D. Neurotoxicity of Alzheimer's disease A β peptides is induced by small changes in the A β 42 to A β 40 ratio. *The EMBO journal*, 29(19): 3408-3420, 2010.
- [121] Wälti, M. A., Ravotti, F., Arai, H., Glabe, C. G., Wall, J. S., Böckmann, A., and Riek, R. Atomic-resolution structure of a disease-relevant A β (1-42) amyloid fibril. *Proceedings of the National Academy of Sciences*, 113(34): E4976-E4984, 2016.
- [122] Garvey, M., Baumann, M., Wulff, M., Kumar, S. T., Markx, D., Morgado, I., and Balbach, J. Molecular architecture of A β fibrils grown in cerebrospinal fluid solution and in a cell culture model of A β plaque formation. *Amyloid*, 23(2): 76-85, 2016.
- [123] Jahn, T. R., Makin, O. S., Morris, K. L., Marshall, K. E., Tian, P., Sikorski, P., and Serpell, L. C. The common architecture of cross- β amyloid. *Journal of molecular biology*, 395(4): 717-727, 2010.
- [124] Schmidt, A., Annamalai, K., Schmidt, M., Grigorjeff, N., and Fändrich, M. Cryo-EM reveals the steric zipper structure of a light chain-derived amyloid fibril. *Proceedings of the National Academy of Sciences*, 113(22): 6200-6205, 2016

BIBLIOGRAPHY

- [125] Lührs, T., Ritter, C., Adrian, M., Riek-Loher, D., Bohrmann, B., Döbeli, H., and Riek, R. 3D structure of Alzheimer's amyloid- β (1–42) fibrils. *Proceedings of the National Academy of Sciences of the United States of America*, 102(48): 17342-17347, 2005.
- [126] Nilsson, M. R. Techniques to study amyloid fibril formation in vitro. *Methods*, 34(1): 151-160, 2004.
- [127] Sawaya, M. R., Sambashivan, S., Nelson, R., Ivanova, M. I., Sievers, S. A., Apostol, M. I., and Madsen, A. Ø. Atomic structures of amyloid cross- β spines reveal varied steric zippers. *Nature*, 447(7143): 453-457, 2007.
- [128] Fitzpatrick, A. W., Debelouchina, G. T., Bayro, M. J., Clare, D. K., Caporini, M. A., Bajaj, V. S., and MacPhee, C. E. Atomic structure and hierarchical assembly of a cross- β amyloid fibril. *Proceedings of the National Academy of Sciences*, 110(14): 5468-5473, 2013.
- [129] Vugmeyster, L., Clark, M. A., Falconer, I. B., Ostrovsky, D., Gantz, D., Qiang, W., and Hoatson, G. L. Flexibility and solvation of amyloid- β hydrophobic core. *Journal of Biological Chemistry*, 291(35): 18484-18495, 2016.
- [130] Qiang, W., Yau, W. M., Luo, Y., Mattson, M. P., and Tycko, R. Antiparallel β -sheet architecture in Iowa-mutant β -amyloid fibrils. *Proceedings of the National Academy of Sciences*, 109(12): 4443-4448, 2012.
- [131] Antzutkin, O. N., Balbach, J. J., Leapman, R. D., Rizzo, N. W., Reed, J., and Tycko, R. Multiple quantum solid-state NMR indicates a parallel, not antiparallel, organization of β -sheets in Alzheimer's β -amyloid fibrils. *Proceedings of the National Academy of Sciences*, 97(24): 13045-13050, 2000.
- [132] Tycko, R. Amyloid polymorphism: structural basis and neurobiological relevance. *Neuron*, 86(3): 632-645, 2015.
- [133] Jiménez, J. L., Nettleton, E. J., Bouchard, M., Robinson, C. V., Dobson, C. M., and Saibil, H. R. The protofilament structure of insulin amyloid fibrils. *Proceedings of the National Academy of Sciences*, 99(14): 9196-9201, 2002.
- [134] Fändrich, M., Meinhardt, J., and Grigorieff, N. Structural polymorphism of Alzheimer Abeta and other amyloid fibrils. *Prion*, 3(2): 89-93, 2009.

BIBLIOGRAPHY

- [135] Christopeit, T., Hortschansky, P., Schroeckh, V., Gührs, K., Zandomeneghi, G., and Fändrich, M. Mutagenic analysis of the nucleation propensity of oxidized Alzheimer's β -amyloid peptide. *Protein science*, 14(8): 2125-2131, 2005.
- [136] Auer, S. Nucleation of polymorphic amyloid fibrils. *Biophysical journal*, 108(5): 1176-1186, 2015.
- [137] Mullard, A. Sting of Alzheimer's failures offset by upcoming prevention trials. *Nature reviews Drug discovery*, 11(9): 657-660, 2012.
- [138] Mohamed, T., Shakeri, A., and Rao, P. P. Amyloid cascade in Alzheimer's disease: Recent advances in medicinal chemistry. *European Journal of medicinal chemistry*, 113: 258-272, 2016.
- [139] Svennerholm, L. Gangliosides--a new therapeutic agent against stroke and Alzheimer's disease. *Life sciences*, 55(25): 2125-2134, 1994.
- [140] Castillo, G. M., Ngo, C., Cummings, J., Wight, T. N., and Snow, A. D. Perlecan Binds to the β -Amyloid Proteins ($A\beta$) of Alzheimer's Disease, Accelerates $A\beta$ Fibril Formation, and Maintains $A\beta$ Fibril Stability. *Journal of neurochemistry*, 69(6): 2452-2465, 1997.
- [141] Yatin, S. M., Yatin, M., Varadarajan, S., Ain, K. B., and Butterfield, D. A. Role of spermine in amyloid β -peptide-associated free radical-induced neurotoxicity. *Journal of neuroscience research*, 63(5): 395-401, 2001.
- [142] Dolphin, G. T., Chierici, S., Ouberai, M., Dumy, P., and Garcia, J. A Multimeric Quinacrine Conjugate as a Potential Inhibitor of Alzheimer's β -Amyloid Fibril Formation. *Chembiochem*, 9(6): 952-963, 2008.
- [143] Evans, C. G., Wisén, S., and Gestwicki, J. E. Heat shock proteins 70 and 90 inhibit early stages of amyloid β -(1-42) aggregation in vitro. *Journal of Biological Chemistry*, 281(44): 33182-33191, 2006.
- [144] Bush, A. I. Metal complexing agents as therapies for Alzheimer's disease. *Neurobiology of aging*, 23(6): 1031-1038, 2002.
- [145] Nitz, M., Fenili, D., Darabie, A. A., Wu, L., Cousins, J. E., and McLaurin, J. Modulation of amyloid- β aggregation and toxicity by inosose stereoisomers. *The FEBS journal*, 275(8): 1663-1674, 2008.

BIBLIOGRAPHY

- [146] Takahashi, T., Tada, K., and Mihara, H. RNA aptamers selected against amyloid β -peptide (A β) inhibit the aggregation of A β . *Molecular Biosystems*, 5(9): 986-991, 2009.
- [147] Galimberti, D. and Scarpini, E. Emerging amyloid disease-modifying drugs for Alzheimer's disease. *Expert Opinion on Emerging Drugs*, 21(1): 5-7, 2016.
- [148] Bellucci, L., Ardèvol, A., Parrinello, M., Lutz, H., Lu, H., Weidner, T., and Corni, S. The interaction with gold suppresses fiber-like conformations of the amyloid β (16–22) peptide. *Nanoscale*, 8(16): 8737-8748, 2016.
- [149] Li, H., Luo, Y., Derreumaux, P., and Wei, G. Carbon nanotube inhibits the formation of β -sheet-rich oligomers of the Alzheimer's amyloid- β (16–22) peptide. *Biophysical Journal*, 101(9): 2267-2276, 2011.
- [150] Yang, Z., Ge, C., Liu, J., Chong, Y., Gu, Z., Jimenez-Cruz, C. A., Chai, Z., and Zhou, R. Destruction of amyloid fibrils by graphene through penetration and extraction of peptides. *Nanoscale*, 7(44): 18725-18737, 2015.
- [151] Dhanavade, M. J., Parulekar, R. S., Kamble, S. A., and Sonawane, K. D. Molecular modeling approach to explore the role of cathepsin B from *Hordeum vulgare* in the degradation of A β peptides. *Molecular BioSystems*, 12(1): 162-168, 2016.
- [152] Jalkute, C. B., Barage, S. H., and Sonawane, K. D. Insight into molecular interactions of A β peptide and gelatinase from *Enterococcus faecalis*: a molecular modeling approach. *Royal Society of Chemistry Advances*, 5(14): 10488-10496, 2015.
- [153] Wang, X., Sun, X., Kuang, G., Ågren, H., and Tu, Y. A theoretical study on the molecular determinants of the affibody protein ZA β 3 bound to an amyloid β peptide. *Physical Chemistry Chemical Physics*, 17(26): 16886-16893, 2015.
- [154] Romero, A., Cacabelos, R., Oset-Gasque, M. J., Samadi, A., and Marco-Contelles, J. Novel tacrine-related drugs as potential candidates for the treatment of Alzheimer's disease. *Bioorganic & medicinal chemistry letters*, 23(7): 1916-1922, 2013.
- [155] Arai, T., Araya, T., Sasaki, D., Taniguchi, A., Sato, T., Sohma, Y., and Kanai, M. Rational Design and Identification of a Non-Peptidic Aggregation

BIBLIOGRAPHY

- Inhibitor of Amyloid- β Based on a Pharmacophore Motif Obtained from cyclo [-Lys-Leu-Val-Phe-Phe-]. *Angewandte Chemie International Edition*, 53(31): 8236-8239, 2014.
- [156] Kroth, H., Ansaloni, A., Varisco, Y., Jan, A., Sreenivasachary, N., Rezaei-Ghaleh, N., and Pihlgren, M. Discovery and structure activity relationship of small molecule inhibitors of toxic β -amyloid-42 fibril formation. *Journal of Biological Chemistry*, 287(41): 34786-34800, 2012.
- [157] Veloso, A. J., Dhar, D., Chow, A. M., Zhang, B., Tang, D. W., Ganesh, H. V., and Kerman, K. sym-Triazines for directed multitarget modulation of cholinesterases and amyloid- β in Alzheimer's disease. *ACS chemical neuroscience*, 4(2): 339-349, 2012.
- [158] Lu, C., Guo, Y., Yan, J., Luo, Z., Luo, H. B., Yan, M., and Li, X. Design, synthesis, and evaluation of multitarget-directed resveratrol derivatives for the treatment of Alzheimer's disease. *Journal of medicinal chemistry*, 56(14): 5843-5859, 2013.
- [159] Alder, B. J. and Wainwright, T. E. Studies in molecular dynamics. I. General method. *The Journal of Chemical Physics*, 31(2): 459-466, 1959.
- [160] Rahman, A. Correlations in the motion of atoms in liquid argon. *Physical Review*, 136(2A): A405-A410, 1964.
- [161] Stillinger, F. H. and Rahman, A. Improved simulation of liquid water by molecular dynamics. *The Journal of Chemical Physics*, 60(4): 1545-1557, 1974.
- [162] McCammon, J. A., Gelin, B. R., and Karplus, M. Dynamics of folded proteins. *Nature*, 267(5612): 585-590, 1977.
- [163] Van Gunsteren, W. F. and Berendsen, H. J. C. A leap-frog algorithm for stochastic dynamics. *Molecular Simulation*, 1(3): 173-185, 1988.
- [164] Retrieved on 12 March 2018 from https://cmm.cit.nih.gov/intro_simulation/node/15.html
- [165] Kittel, C. Wave diffraction and the reciprocal lattice. In Johnson, S., McFadden, P., Batey, M., editors, *Introduction to solid state physics*, pages 23-41, ISBN 0-471-41526-X. Wiley Publication, 2005.
- [166] Fincham, D. Optimisation of the Ewald sum for large systems. *Molecular Simulation*, 13(1): 1-9, 1994.

BIBLIOGRAPHY

- [167] Ryckaert, J. P., Ciccotti, G., and Berendsen, H. J. Numerical integration of the cartesian equations of motion of a system with constraints: molecular dynamics of n-alkanes. *Journal of Computational Physics*, 23(3): 327-341, 1977.
- [168] Berendsen, H. J., Postma, J. V., van Gunsteren, W. F., DiNola, A. R. H. J., and Haak, J. R. Molecular dynamics with coupling to an external bath. *The Journal of chemical physics*, 81(8): 3684-3690, 1984.
- [169] Case, D.A., Darden, T.A., Cheatham III, T.E., Simmerling, C.L., Wang, J., Duke, R.E., Luo, R., Walker, R.C., Zhang, W., Merz, K.M., Roberts, B., Hayik, S., Roitberg, A., Seabra, G., Swails, J., Götz, A.W., Kolossváry, I., Wong, K.F., Paesani, F., Vanicek, J., Wolf, R.M., Liu, J., Wu, X., Brozell, S.R., Steinbrecher, T., Gohlke, H., Cai, Q., Ye, X., Wang, J., Hsieh, M.J., Cui, G., Roe, D.R., Mathews, D.H., Seetin, M.G., Salomon-Ferrer, R., Sagui, C., Babin, V., Luchko, T., Gusarov, S., Kovalenko, A., and Kollman P.A. AMBER 12, University of California, San Francisco, 2012.
- [170] Jorgensen, W. L. and Jenson, C. Temperature dependence of TIP3P, SPC, and TIP4P water from NPT Monte Carlo simulations: Seeking temperatures of maximum density. *Journal of Computational Chemistry*, 19(10): 1179-1186, 1998.
- [171] Andrew, R. L. *Molecular modeling principles and applications*. Dorling Kindersley (India) Pvt. Ltd., U.P. India, 2nd edition, 2001.
- [172] Kirkwood, J. G. Statistical mechanics of fluid mixtures. *The Journal of Chemical Physics*, 3(5): 300-313, 1935.
- [173] Torrie, G. M. and Valleau, J. P. Nonphysical sampling distributions in Monte Carlo free-energy estimation: Umbrella sampling. *Journal of Computational Physics*, 23(2): 187-199, 1977.
- [174] Kumar, S., Rosenberg, J. M., Bouzida, D., Swendsen, R. H., and Kollman, P. A. The weighted histogram analysis method for free-energy calculations on biomolecules. I. The method. *Journal of computational chemistry*, 13(8): 1011-1021, 1992.
- [175] Duhovny, D., Nussinov, R., and Wolfson, H. J. Efficient unbound docking of rigid molecules. *Lecture notes in computer science*, 2452: 185-200, 2002.

BIBLIOGRAPHY

- [176] Kuntz, I. D., Blaney, J. M., Oatley, S. J., Langridge, R., and Ferrin, T. E. A geometric approach to macromolecule-ligand interactions. *Journal of molecular biology*, 161(2): 269-288, 1982.
- [177] Kollman, P. A., Massova, I., Reyes, C., Kuhn, B., Huo, S., Chong, L., and Donini, O. Calculating structures and free energies of complex molecules: combining molecular mechanics and continuum models. *Accounts of chemical research*, 33(12): 889-897, 2000.
- [178] Sobolev, V., Sorokine, A., Prilusky, J., Abola, E. E., and Edelman, M. Automated analysis of interatomic contacts in proteins. *Bioinformatics (Oxford, England)*, 15(4): 327-332, 1999.
- [179] Sobolev, V., Wade, R. C., Vriend, G., and Edelman, M. Molecular docking using surface complementarity. *Proteins: Structure, Function, and Bioinformatics*, 25(1): 120-129, 1996.
- [180] Laskowski, R. A., Hutchinson, E. G., Michie, A. D., Wallace, A. C., Jones, M. L., and Thornton, J. M. PDBsum: a Web-based database of summaries and analyses of all PDB structures. *Trends in biochemical sciences*, 22(12): 488-490, 1997.
- [181] Wallace, A. C., Laskowski, R. A., and Thornton, J. M. LIGPLOT: a program to generate schematic diagrams of protein-ligand interactions. *Protein Engineering, Design and Selection*, 8(2): 127-134, 1995.
- [182] Laskowski, R. A. and Swindells, M. B. LigPlot+: multiple ligand–protein interaction diagrams for drug discovery. *Journal of Chemical Information and Modeling*, 51 (10): 2778-2786, 2011.
- [183] Kabsch, W. and Sander, C. Dictionary of protein secondary structure: pattern recognition of hydrogen-bonded and geometrical features. *Biopolymers*, 22(12): 2577-2637, 1983.
- [184] Lee, C. and Ham, S. Characterizing amyloid-beta protein misfolding from molecular dynamics simulations with explicit water. *Journal of Computational Chemistry*, 32(2): 349-5515, 2011.
- [185] Kotler, S. A., Walsh, P., Brender, J. R., and Ramamoorthy, A. Differences between amyloid- β aggregation in solution and on the membrane: insights into elucidation of the mechanistic details of Alzheimer's disease. *Chemical Society Reviews*, 43(19): 6692-6700, 2014.

BIBLIOGRAPHY

- [186] Parthasarathy, S., Inoue, M., Xiao, Y., Matsumura, Y., Nabeshima, Y. I., Hoshi, M., and Ishii, Y. Structural insight into an Alzheimer's brain-derived spherical assembly of amyloid β by solid-state NMR. *Journal of the American Chemical Society*, 137(20): 6480-6483, 2015.
- [187] Simmerling, C., Strockbine, B., and Roitberg, A.E. All-atom structure prediction and folding simulations of a stable protein. *Journal of the American Chemical Society*, 124(38): 11258-11259, 2002.
- [188] Hornak, V., Abel, R., Okur, A., Strockbine, B., Roitberg, A., and Simmerling, C. Comparison of multiple Amber force fields and development of improved protein backbone parameters. *Proteins: Structure, Function, and Bioinformatics*, 65(3): 712-725, 2006.
- [189] Onufriev, A., Bashford, D., and Case, D. A. Exploring protein native states and large-scale conformational changes with a modified generalized born model. *Proteins: Structure, Function, and Bioinformatics*, 55(2): 383-394, 2004.
- [190] Darden, T., York, D., and Pedersen, L. Particle mesh Ewald: An $N \cdot \log(N)$ method for Ewald sums in large systems. *The Journal of chemical physics*, 98(12):10089-10092, 1993.
- [191] Roe, D. R. and Cheatham III, T. E. PTraJ and CPPTRAJ: software for processing and analysis of molecular dynamics trajectory data. *Journal of chemical theory and computation*, 9(7): 3084-3095, 2013.
- [192] Humphrey, W., Dalke, A., and Schulten, K. VMD: visual molecular dynamics. *Journal of molecular graphics*, 14(1):33-38, 1996.
- [193] Hou, L., Shao, H., Zhang, Y., Li, H., Menon, N. K., Neuhaus, E. B., and Iwashita, T. Solution NMR studies of the A β (1– 40) and A β (1– 42) peptides establish that the Met35 oxidation state affects the mechanism of amyloid formation. *Journal of the American Chemical Society*, 126(7): 1992-2005, 2004.
- [194] Yan, Y., McCallum, S. A., and Wang, C. M35 oxidation induces A β 40-like structural and dynamical changes in A β 42. *Journal of the American Chemical Society*, 130(16): 5394-5395, 2008.
- [195] Rosenman, D. J., Connors, C. R., Chen, W., Wang, C., and García, A. EA β monomers transiently sample oligomer and fibril-like configurations: Ensemble characterization using a combined MD/NMR approach. *Journal of molecular biology*, 425(18): 3338-3359, 2013.

BIBLIOGRAPHY

- [196] Reddy, G., Straub, J. E., and Thirumalai, D. Influence of preformed asp23–lys28 salt bridge on the conformational fluctuations of monomers and dimers of A β peptides with implications for rates of fibril formation. *The Journal of Physical Chemistry B*, 113(4): 1162-1172, 2009.
- [197] Roychaudhuri, R., Yang, M., Condron, M.M., and Teplow, D.B. Structural dynamics of the amyloid β -protein monomer folding nucleus. *Biochemistry*, 51(19): 3957-3959, 2012.
- [198] Lee, J., Dubey, V. K., Longo, L. M., and Blaber, M. A logical OR redundancy within the Asx-Pro-Asx-Gly type I β -turn motif. *Journal of molecular biology*, 377(4): 1251-1264, 2008.
- [199] Morimoto, A., Irie, K., Murakami, K., Masuda, Y., Ohigashi, H., Nagao, M., and Shirasawa, T. Analysis of the secondary structure of β -amyloid (A β 42) fibrils by systematic proline replacement. *Journal of Biological Chemistry*, 279(50): 52781-52788, 2004.
- [200] Kowalewski, T. and Holtzman, D. M. In situ atomic force microscopy study of Alzheimer's β -amyloid peptide on different substrates: New insights into mechanism of β -sheet formation. *Proceedings of the National Academy of Sciences*, 96(7): 3688-3693, 1999.
- [201] Fink, A. L. Protein aggregation: folding aggregates, inclusion bodies and amyloid. *Folding and design*, 3(1): R9-R23, 1998.
- [202] Wei, W., Norton, D. D., Wang, X., and Kusiak, J. W. A β 17–42 in Alzheimer's disease activates JNK and caspase-8 leading to neuronal apoptosis. *Brain*, 125(9): 2036-2043, 2002.
- [203] Miller, Y., Ma, B., and Nussinov, R. Polymorphism of Alzheimer's A β 17-42 (p3) Oligomers: The Importance of the Turn Location and Its Conformation. *Biophysical journal*, 97(4): 1168-1177, 2009.
- [204] Feig, M., Karanicolas, J., and Brooks, C. L. MMTSB Tool Set: enhanced sampling and multiscale modeling methods for applications in structural biology. *Journal of Molecular Graphics and Modelling*, 22(5): 377-395, 2004.
- [205] Urbanc, B., Cruz, L., Ding, F., Sammond, D., Khare, S., Buldyrev, S.V., and Dokholyan, N.V. Molecular dynamics simulation of amyloid β dimer formation. *Biophysical Journal*, 87(4): 2310-2321, 2004.

BIBLIOGRAPHY

- [206] Gursky, O. and Aleshkov, S. Temperature-dependent β -sheet formation in β -amyloid A β 1–40 peptide in water: uncoupling β -structure folding from aggregation. *Biochimica et Biophysica Acta (BBA)-Protein Structure and Molecular Enzymology*, 1476(1): 93-102, 2000.
- [207] Li, J., Hoop, C.L., Kodali, R., Sivanandam, V.N., and Van der Wel, P.C. Amyloid-like fibrils from a domain-swapping protein feature a parallel, in-register conformation without native-like interactions. *Journal of Biological Chemistry*, 286(33): 28988-28995, 2011.
- [208] Schmidt, M., Rohou, A., Lasker, K., Yadav, J. K., Schiene-Fischer, C., Fändrich, M., and Grigorieff, N. Peptide dimer structure in an A β (1–42) fibril visualized with cryo-EM. *Proceedings of the National Academy of Sciences*, 112(38): 11858-11863, 2015.
- [209] Raz, Y. and Miller, Y. Interactions between A β and mutated Tau lead to polymorphism and induce aggregation of A β -mutated tau oligomeric complexes. *PloS one*, 8(8): e73303, 2013.
- [210] Vasconcelos, B., Stancu, I. C., Buist, A., Bird, M., Wang, P., Vanoosthuyse, A., and Baatsen, P. Heterotypic seeding of Tau fibrillization by pre-aggregated Abeta provides potent seeds for prion-like seeding and propagation of Tau-pathology in vivo. *Acta neuropathologica*, 131(4): 549-569, 2016.
- [211] Crescenzi, O., Tomaselli, S., Guerrini, R., Salvadori, S., D'Ursi, A.M., Temussi, P.A., and Picone, D. Solution structure of the Alzheimer amyloid β -peptide (1–42) in an apolar microenvironment. *European Journal of Biochemistry*, 269(22):5642-5648, 2002.
- [212] Berman, H.M., Westbrook, J., Feng, Z., Gilliland, G., Bhat, T.N., Weissig, H., Shindyalov, I.N., and Bourne, P.E. The protein data bank. *Nucleic acids research*, 28(1):235-242, 2000.
- [213] Kadavath, H., Jaremko, M., Jaremko, Ł., Biernat, J., Mandelkow, E., and Zweckstetter, M. Folding of the tau protein on microtubules. *Angewandte Chemie International Edition*, 54(35): 10347-10351, 2015.
- [214] Do, T. D., Economou, N. J., Chamas, A., Buratto, S. K., Shea, J. E., and Bowers, M. T. Interactions between amyloid- β and Tau fragments promote

BIBLIOGRAPHY

- aberrant aggregates: implications for amyloid toxicity. *The Journal of Physical Chemistry B*, 118(38): 11220-11230, 2014.
- [215] Guo, J. P., Arai, T., Miklossy, J., and McGeer, P. L. A β and Tau form soluble complexes that may promote self-aggregation of both into the insoluble forms observed in Alzheimer's disease. *Proceedings of the National Academy of Sciences*, 103(6): 1953-1958, 2006.
- [216] de Groot, N. S., Castillo, V., Grana-Montes, R., and Ventura, S. AGGRESCAN: method, application, and perspectives for drug design. *Computational Drug Discovery and Design*, 819: 199-220, 2012.
- [217] Pierce, B., Tong, W., and Weng, Z. M-ZDOCK: A Grid-based Approach for Cn Symmetric Multimer Docking. *Bioinformatics*, 21(8): 1472-1476, 2005.
- [218] Colvin, M. T., Silvers, R., Ni, Q. Z., Can, T. V., Sergeyev, I., Rosay, M., and Griffin, R. G. Atomic resolution structure of monomorphous A β 42 amyloid fibrils. *Journal of the American Chemical Society*, 138(30): 9663-9674, 2016.
- [219] Xiao, Y., Ma, B., McElheny, D., Parthasarathy, S., Long, F., Hoshi, M., and Ishii, Y. A [math]\beta(1-42) fibril structure illuminates self-recognition and replication of amyloid in Alzheimer's disease. *Nature structural & molecular biology*, 22(6): 499-505, 2015.
- [220] Wälti, M. A., Ravotti, F., Arai, H., Glabe, C. G., Wall, J. S., Böckmann, A., and Riek, R. Atomic-resolution structure of a disease-relevant A β (1-42) amyloid fibril. *Proceedings of the National Academy of Sciences*, 113(34): E4976-E4984, 2016.
- [221] Krieger, E. and Vriend, G. New ways to boost molecular dynamics simulations. *Journal of Computational Chemistry*, 36(13): 996-1007, 2015.
- [222] Bousset, L., Pieri, L., Ruiz-Arlandis, G., Gath, J., Jensen, P. H., Habenstein, B., and Melki, R. Structural and functional characterization of two alpha-synuclein strains. *Nature communications*, 4: 2575, 2013.
- [223] Petkova, A. T., Leapman, R. D., Guo, Z., Yau, W. M., Mattson, M. P., and Tycko, R. Self-propagating, molecular-level polymorphism in Alzheimer's β -amyloid fibrils. *Science*, 307(5707): 262-265, 2005.
- [224] Seubert, P., Vigo-Pelfrey, C., Esch, F., Lee, M., Dovey, H., Davis, D., Sinha, S., Schlossmacher, M., Whaley, J., and Swindlehurst, C. Isolation and

BIBLIOGRAPHY

- quantification of soluble Alzheimer's beta-peptide from biological fluids. *Nature*, 359 (6393): 325-327, 1992.
- [225] Calamai, M., Kumita, J. R., Mifsud, J., Parrini, C., Ramazzotti, M., Ramponi, G., and Dobson, C. M. Nature and significance of the interactions between amyloid fibrils and biological polyelectrolytes. *Biochemistry*, 45(42): 12806-12815, 2006.
- [226] Barrantes, A., Camero, S., Garcia-Lucas, A., J Navarro, P., J Benitez, M., and S Jimenez, J. Alzheimer's disease amyloid peptides interact with DNA, as proved by surface plasmon resonance. *Current Alzheimer Research*, 9(8): 924-934, 2012.
- [227] Barrantes, A., Rejas, M. T., Benitez, M. J., and Jiménez, J. S. Interaction between Alzheimer's A β 1-42 peptide and DNA detected by surface plasmon resonance. *Journal of Alzheimer's Disease*, 12(4): 345-355, 2007.
- [228] Udomprasert, A., Bongiovanni, M. N., Sha, R., Sherman, W. B., Wang, T., Arora, P. S., and Seeman, N. C. Amyloid fibrils nucleated and organized by DNA origami constructions. *Nature nanotechnology*, 9(7): 537-541, 2014.
- [229] Schnitzler, T. and Herrmann, A. DNA block copolymers: functional materials for nanoscience and biomedicine. *Accounts of chemical research*, 45(9): 1419-1430, 2012.
- [230] Gour, N., Kedracki, D., Safir, I., Ngo, K. X., and Vebert-Nardin, C. Self-assembling DNA-peptide hybrids: morphological consequences of oligonucleotide grafting to a pathogenic amyloid fibrils forming dipeptide. *Chemical Communications*, 48(44): 5440-5442, 2012.
- [231] Abraham, J. N., Kedracki, D., Prado, E., Gourmet, C., Maroni, P., and Nardin, C. Effect of the interaction of the amyloid β (1-42) peptide with short single-stranded synthetic nucleotide sequences: morphological characterization of the inhibition of fibrils formation and fibrils disassembly. *Biomacromolecules*, 15(9): 3253-3258, 2014.
- [232] Macke, T.J. and Case, D.A. Molecular Modeling of Nucleic Acids. *American Chemical Society, Washington DC*, 1998.
- [233] Jan, A., Gokce, O., Luthi-Carter, R., and Lashuel, H.A. The ratio of monomeric to aggregated forms of A β 40 and A β 42 is an important determinant

BIBLIOGRAPHY

- of amyloid- β aggregation, fibrillogenesis, and toxicity. *The Journal of Biological Chemistry*, 283(42):28176-28189, 2008.
- [234] Economou, N. J., Giammona, M. J., Do, T. D., Zheng, X., Teplow, D. B., Buratto, S. K., and Bowers, M. T. Amyloid β -protein assembly and Alzheimer's disease: Dodecamers of A β 42, but not of A β 40, seed fibril formation. *Journal of the American Chemical Society*, 138(6): 1772-1775, 2016.
- [235] Tran, J., Chang, D., Hsu, F., Wang, H., and Guo, Z. Cross-seeding between A β 40 and A β 42 in Alzheimer's disease. *FEBS letters*, 591(1): 177-185, 2017.
- [236] Vivekanandan, S., Brender, J. R., Lee, S. Y., and Ramamoorthy, A. A partially folded structure of amyloid-beta (1–40) in an aqueous environment. *Biochemical and biophysical research communications*, 411(2): 312-316, 2011.
- [237] Murphy, M.P., Beard, J., Das, P., and Jansen, K. A β 42 is essential for parenchymal and vascular amyloid deposition in mice. *Neuron*, 47(2): 191-199, 2005.
- [238] Sadigh-Eteghad, S., Sabermarouf, B., Majdi, A., Talebi, M., Farhoudi, M., and Mahmoudi, J. Amyloid-beta: a crucial factor in Alzheimer's disease. *Medical Principles and Practice*, 24(1): 1-10, 2014.
- [239] Cukalevski, R., Yang, X., Meisl, G., Weininger, U., Bernfur, K., Frohm, B., and Linse, S. The A β 40 and A β 42 peptides self-assemble into separate homomolecular fibrils in binary mixtures but cross-react during primary nucleation. *Chemical Science*, 6(7):4215-4233, 2015.
- [240] Yan, Y. and Wang, C. A β 40 protects non-toxic A β 42 monomer from aggregation. *Journal of molecular biology*, 369(4): 909-916, 2007.
- [241] Chebaro, Y., Mousseau, N., and Derreumaux, P. Structures and Thermodynamics of Alzheimer's Amyloid- β A β (16– 35) Monomer and Dimer by Replica Exchange Molecular Dynamics Simulations: Implication for Full-Length A β Fibrillation. *The Journal of Physical Chemistry B*, 113(21):7668-7675, 2009.
- [242] Lazo, N. D., Grant, M. A., Condron, M. C., Rigby, A. C., and Teplow, D. B. On the nucleation of amyloid β -protein monomer folding. *Protein Science*, 14(6): 1581-1596, 2005.

BIBLIOGRAPHY

- [243] Gnanakaran, S., Nussinov, R., and García, A. E. Atomic-level description of amyloid β -dimer formation. *Journal of the American Chemical Society*, 128(7): 2158-2159, 2006.
- [244] Barr, R. K., Verdile, G., Wijaya, L. K., Morici, M., Taddei, K., Gupta, V. B., and Fraser, P. E. Validation and characterization of a novel peptide that binds monomeric and aggregated β -amyloid and inhibits the formation of neurotoxic oligomers. *Journal of Biological Chemistry*, 291(2): 547-559, 2016.
- [245] Hiramatsu, H., Ochiai, H., and Komuro, T. Effects of N-Methylated Amyloid- β 30–40 Peptides on the Fibrillation of Amyloid- β 1–40. *Chemical biology & drug design*, 87(3): 425-433, 2016.
- [246] Grillo-Bosch, D., Carulla, N., Cruz, M., Sánchez, L., Pujol-Pina, R., Madurga, S., Rabanal, F., and Giralt, E., Retro-enantio N-methylated peptides as β -amyloid aggregation inhibitors. *ChemMedChem*, 4(9): 1488–1494, 2009.
- [247] Cummings, J., Morstorf, T., and Lee, G. Alzheimer's drug-development pipeline: 2016. *Alzheimer's & Dementia: Translational Research & Clinical Interventions*, 2(4): 222-232, 2016.
- [248] Kokkoni, N., Stott, K., Amijee, H., Mason, J. M., and Doig, A. J. N-Methylated analogues of β -amyloid aggregation and toxicity: Optimization of the inhibitor structure. *Biochemistry*, 45(32): 9906–9918, 2006.
- [249] Matharu, B., El-Agnaf, O., Razvi, A., and Austen, B. M. Development of retro-inverso peptides as anti-aggregation drugs for β -amyloid in Alzheimer's disease. *Peptides*, 31(10): 1866-1872, 2010.
- [250] Morales-Zavala, F., Arriagada, H., Hassan, N., Velasco, C., Riveros, A., Álvarez, A. R., and Rodriguez, K. Peptide multifunctionalized gold nanorods decrease toxicity of β -amyloid peptide in a *Caenorhabditis elegans* model of Alzheimer's disease. *Nanomedicine: Nanotechnology, Biology and Medicine*, 13(7): 2341-2350, 2017.
- [251] Fradinger, E. A., Monien, B. H., Urbanc, B., Lomakin, A., Tan, M., Li, H., Spring, S. M., Condron, M. M., Cruz, L., Xie, C. W., Benedek, G. B., and Bitan, G. C-Terminal peptides coassemble into A β 1–42 oligomers and protect neurons against A β 1–42-induced neurotoxicity. *Proceedings of the National Academy of Sciences*, 105(37): 14175-14180, 2008.

BIBLIOGRAPHY

- [252] Pratim Bose, P., Chatterjee, U., Nerelius, C., Govender, T., Norström, T., Gogoll, A., Sandegren, A., Göthelid, E., Johansson, J., and Arvidsson, P. I.. Poly-N-methylated amyloid β -peptide (A β) C-terminal fragments reduce A β toxicity in vitro and in *Drosophila melanogaster*. *Journal of medicinal chemistry*, 52(24): 8002-8009, 2009.
- [253] Hilbich, C., Kisters-Woike, B., Reed, J., Masters, C. L., and Beyreuther, K. Substitutions of hydrophobic amino acids reduce the amyloidogenicity of Alzheimer's disease A β peptides. *Journal of molecular biology*, 228(2): 460-473, 1992.
- [254] Jarrett, J. T., Berger, E. P., and Lansbury, P. T., Jr. The carboxy terminus of the amyloid protein is critical for the seeding of amyloid formation: Implications for the pathogenesis of Alzheimer's disease? *Biochemistry*, 32(18): 4693-4697, 1993.
- [255] Bansal, S., Maurya, I. K., Yadav, N., Thota, C. K., Kumar, V., Tikoo, K., and Jain, R. C-Terminal fragment, A β 32–37, analogues protect against A β aggregation-induced toxicity. *ACS chemical neuroscience*, 7(5): 615-623, 2016.
- [256] Guex, N. and Peitsch, M.C. SWISS-MODEL and the Swiss-PdbViewer: An environment for comparative protein modeling. *Electrophoresis*, 18(15): 2714-2723, 1997.