

REFERENCES

- [1] Stefani M, Rigacci S. Protein folding and aggregation into amyloid: the interference by natural phenolic compounds. *Int J Mol Sci* 2013;14(6):12411-57.
- [2] Herczenik E, Gebbink MFBG. Molecular and cellular aspects of protein misfolding and disease. *FASEB J* 2008;22(7):2115-33.
- [3] Brahma A, Mandal C, Bhattacharyya D. Characterization of a dimeric unfolding intermediate of bovine serum albumin under mildly acidic condition. *Biochim Biophys Acta,Proteins Proteomics* 2005;1751(2):159-69.
- [4] Militello V, Casarino C, Emanuele A, Giostra A, Pullara F, Leone M. Aggregation kinetics of bovine serum albumin studied by FTIR spectroscopy and light scattering. *Biophys Chem* 2004;107(2):175-87.
- [5] Thai CK, Dai H, Sastry MSR, Sarikaya M, Schwartz DT, Baneyx F. Identification and characterization of Cu₂O- and ZnO-binding polypeptides by Escherichia coli cell surface display: toward an understanding of metal oxide binding. *Biotechnol Bioeng* 2004;87(2):129-37.
- [6] Agorogiannis EI, Agorogiannis GI, Papadimitriou A, Hadjigeorgiou GM. Protein misfolding in neurodegenerative diseases. *Neuropathol Appl Neurobiol* 2004;30(3):215-24.
- [7] Radford SE, Dobson CM. From computer simulations to human disease. *Cell* 2017;97(3):291-8.
- [8] Stefani M, Dobson CM. Protein aggregation and aggregate toxicity: new insights into protein folding, misfolding diseases and biological evolution. *J Mol Med* 2003;81(11):678-99.

- [9] Fabrizio C, Christopher MD. Protein misfolding, functional amyloid, and human disease. *Annu Rev Biochem* 2006;75(1):333-66.
- [10] Dobson CM. Protein folding and misfolding. *Nature* 2003;426(6968):884-90.
- [11] Selkoe DJ. Folding proteins in fatal ways. *Nature* 2003;426(6968):900-4.
- [12] Serpell LC, Sunde M, Benson MD, Tennent GA, Pepys MB, Fraser PE. The protofilament substructure of amyloid fibrils. *J Mol Biol* 2000;300(5):1033-9.
- [13] Nelson R, Eisenberg D. Recent atomic models of amyloid fibril structure. *Curr Opin Struct Biol* 2006;16(2):260-5.
- [14] Eisenberg D, Jucker M. The amyloid state of proteins in human diseases. *Cell* 2012;148(6):1188-203.
- [15] Sunde M, Serpell LC, Bartlam M, Fraser PE, Pepys MB, Blake CCF. Common core structure of amyloid fibrils by synchrotron X-ray diffraction. *J Mol Biol* 1997;273(3):729-39.
- [16] Alam P, Siddiqi K, Chturvedi SK, Khan RH. Protein aggregation: from background to inhibition strategies. *Int J Biol Macromol* 2017;103:208-19.
- [17] Lindberg DJ, Wenger A, Sundin E, Wesén E, Westerlund F, Esbjörner EK. Binding of Thioflavin-T to amyloid fibrils leads to fluorescence self-quenching and fibril compaction. *Biochemistry* 2017;56(16):2170-4.
- [18] Wilson MR, Yerbury JJ, Poon S. Potential roles of abundant extracellular chaperones in the control of amyloid formation and toxicity. *Mol Biosyst* 2008;4(1):42-52.
- [19] Jarrett JT, Berger EP, Lansbury PT. The carboxy terminus of the beta amyloid protein is critical for the seeding of amyloid formation:

implications for the pathogenesis of Alzheimer's disease. *Biochemistry* 1993;32(18):4693-7.

- [20] Peterson SA, Klabunde T, Lashuel HA, Purkey H, Sacchettini JC, Kelly JW. Inhibiting transthyretin conformational changes that lead to amyloid fibril formation. *Proc Natl Acad Sci* 1998;95(22):12956-60.
- [21] van Gestel J, de Leeuw SW. The formation of fibrils by intertwining of filaments: model and application to amyloid A β protein. *Biophys J* 2007;92(4):1157-63.
- [22] Hippius H, Neundörfer G. The discovery of Alzheimer's disease. *Dialogues Clin Neurosci* 2003;5(1):101-8.
- [23] Alzheimer's A. 2017 Alzheimer's disease facts and figures 2017. 325-73 p.
- [24] Masters CL, Bateman R, Blennow K, Rowe CC, Sperling RA, Cummings JL. Alzheimer's disease. *Nat Rev* 2015;1:1-18.
- [25] Holtzman DM, Bales KR, Paul SM, DeMattos RB. A β immunization and anti-A β antibodies: potential therapies for the prevention and treatment of Alzheimer's disease. *Adv Drug Deliv Rev* 2002;54(12):1603-13.
- [26] Xia W, Zhang J, Kholodenko D, Citron M, Podlisny MB, Teplow DB, et al. Enhanced production and oligomerization of the 42-residue Amyloid β -protein by Chinese hamster ovary cells stably expressing mutant presenilins. *J Biol Chem* 1997;272(12):7977-82.
- [27] Deane R, Sagare A, Hamm K, Parisi M, Lane S, Finn MB, et al. apoE isoform-specific disruption of amyloid β peptide clearance from mouse brain. *J Clin Invest* 2008;118(12):4002-13.
- [28] Ries M, Sastre M. Mechanisms of A β clearance and degradation by glial cells. *Front Aging Neurosci* 2016;8:160.

- [29] Yoon S-S, Jo SA. Mechanisms of Amyloid- β peptide clearance: potential therapeutic targets for Alzheimer's disease. *Biomol Ther (Seoul)* 2012;20(3):245-55.
- [30] Götz J, Ittner A, Ittner LM. Tau-targeted treatment strategies in Alzheimer's disease. *Br J Pharmacol* 2012;165(5):1246-59.
- [31] Hroudová J, Singh N, Fišar Z. Mitochondrial dysfunctions in neurodegenerative diseases: relevance to Alzheimer's disease. *Biomed Res Int* 2014;2014:1-9.
- [32] Gu L, Guo Z. Alzheimer's A β_{42} and A β_{40} peptides form interlaced amyloid fibrils. *J Neurochem* 2013;126(3):305-11.
- [33] Qiu T, Liu Q, Chen Y-X, Zhao Y-F, Li Y-MCPSCR. A β_{42} and A β_{40} : similarities and differences. *J Pept Sci* 2015;21(7):522-9.
- [34] Gravina SA, Ho L, Eckman CB, Long KE, Otvos L, Younkin LH, et al. Amyloid β protein (A β) in Alzheimer's disease brain: biochemical and immunocytochemical analysis with antibodies specific for forms ending at A β_{40} or A $\beta_{42(43)}$. *J Biol Chem* 1995;270(13):7013-6.
- [35] Iwatsubo T, Odaka A, Suzuki N, Mizusawa H, Nukina N, Ihara Y. Visualization of A $\beta_{42(43)}$ and A β_{40} in senile plaques with end-specific A β monoclonals: evidence that an initially deposited species is A $\beta_{42(43)}$. *Neuron* 2017;13(1):45-53.
- [36] Teplow DB. Structural and kinetic features of amyloid β -protein fibrillogenesis. *Amyloid* 1998;5(2):121-42.
- [37] Lesné S, Koh MT, Kotilinek L, Kayed R, Glabe CG, Yang A, et al. A specific amyloid- β protein assembly in the brain impairs memory. *Nature* 2006;440(7082):352-7.

- [38] Murray PS, Kirkwood CM, Gray MC, Ikonomovic MD, Paljug WR, Abrahamson EE, et al. β -Amyloid 42/40 ratio and kalirin expression in Alzheimer disease with psychosis. *Neurobiol Aging* 2012;33(12):2807-16.
- [39] Nutu M, Zetterberg H, Londos E, Minthon L, Nägega K, Blennow K, et al. Evaluation of the cerebrospinal fluid Amyloid- β 1-42/Amyloid- β 1-40 ratio measured by Alpha-LISA to distinguish Alzheimer's disease from other dementia disorders. *Dement Geriatr Cogn Disord* 2013;36(1-2):99-110.
- [40] Peng X-L, Hou L, Xu S-H, Hua Y, Zhou S-J, Zhang Y, et al. Novel APP K724M mutation causes Chinese early-onset familial Alzheimer's disease and increases amyloid- β 42 to amyloid- β 40 ratio. *Neurobiol Aging* 2014;35(11):2657.e1-e6.
- [41] Fernandez MA, Klutkowski JA, Freret T, Wolfe MS. Alzheimer presenilin-1 mutations dramatically reduce trimming of long amyloid β -peptides (A β) by γ -secretase to increase 42-to-40-residue A β . *J Biol Chem* 2014;289(45):31043-52.
- [42] Wiltfang J, Esselmann H, Bibl M, Hüll M, Hampel H, Kessler H, et al. Amyloid β peptide ratio 42/40 but not A β ₄₂ correlates with phospho-Tau in patients with low- and high-CSF A β ₄₀ load. *J Neurochem* 2007;101(4):1053-9.
- [43] Goure WF, Krafft GA, Jerecic J, Hefti F. Targeting the proper amyloid-beta neuronal toxins: a path forward for Alzheimer's disease immunotherapeutics. *Alzheimers Res Ther* 2014;6(4):42-.
- [44] Ahmed M, Davis J, Aucoin D, Sato T, Ahuja S, Aimoto S, et al. Structural conversion of neurotoxic amyloid- β 1-42 oligomers to fibrils. *Nat Struct Mol Biol* 2010;17(5):561-7.
- [45] Hepler RW, Grimm KM, Nahas DD, Breese R, Dodson EC, Acton P, et al. Solution state characterization of Amyloid β -derived diffusible ligands. *Biochemistry* 2006;45(51):15157-67.

- [46] Lambert MP, Barlow AK, Chromy BA, Edwards C, Freed R, Liosatos M, et al. Diffusible, nonfibrillar ligands derived from A β 1–42 are potent central nervous system neurotoxins. *Proc Natl Acad Sci U S A* 1998;95(11):6448–53.
- [47] Walsh DM, Tseng BP, Rydel RE, Podlisny MB, Selkoe DJ. The oligomerization of Amyloid β -protein begins intracellularly in cells derived from human brain. *Biochemistry* 2000;39(35):10831–9.
- [48] Sengupta U, Nilson AN, Kayed R. The role of Amyloid- β oligomers in toxicity, propagation, and immunotherapy. *EBioMedicine* 2016;6:42–9.
- [49] Garzon-Rodriguez W, Vega A, Sepulveda-Becerra M, Milton S, Johnson DA, Yatsimirsky AK, et al. A conformation change in the carboxyl terminus of Alzheimer's A β (1–40) accompanies the transition from dimer to fibril as revealed by fluorescence quenching analysis. *J Biol Chem* 2000;275(30):22645–9.
- [50] O'Nuallain B, Freir DB, Nicoll AJ, Rissee E, Ferguson N, Herron CE, et al. A β dimers rapidly form stable synaptotoxic protofibrils. *J Neurosci* 2010;30(43):14411–9.
- [51] Kayed R, Lasagna-Reeves CA. Molecular mechanisms of amyloid oligomers toxicity. *J Alzheimers Dis* 2012;33(s1):S67–S78.
- [52] Morkuniene R, Cizas P, Jankeviciute S, Petrolis R, Arandarcikaite O, Krisciukaitis A, et al. Small A β 1–42 oligomer-induced membrane depolarization of neuronal and microglial cells: role of N-methyl-D-aspartate receptors. *J Neurosci Res* 2015;93(3):475–86.
- [53] Aisen PS, Gauthier S, Ferris SH, Saumier D, Haine D, Garceau D, et al. Tramiprosate in mild-to-moderate Alzheimer's disease – a randomized, double-blind, placebo-controlled, multi-centre study (the Alphase Study). *Arch Med Sci* 2011;7(1):102–11.

- [54] Gupta-Bansal R, Frederickson RCA, Brunden KR. Proteoglycan-mediated inhibition of A β Proteolysis: a potential cause of senile plaque accumulation. *J Biol Chem* 1995;270(31):18666-71.
- [55] Janusz M, Zablocka A. Colostral Proline-rich polypeptides - immunoregulatory properties and prospects of therapeutic use in Alzheimer's disease. *Curr Alzheimer Res* 2010;7(4):323-33.
- [56] Salloway S, Sperling R, Keren R, Porsteinsson AP, van Dyck CH, Tariot PN, et al. A phase 2 randomized trial of ELND005, scyllo-inositol, in mild to moderate Alzheimer disease. *Neurology* 2011;77(13):1253-62.
- [57] Matlack KES, Tardiff DF, Narayan P, Hamamichi S, Caldwell KA, Caldwell GA, et al. Clioquinol promotes the degradation of metal-dependent amyloid- β (A β) oligomers to restore endocytosis and ameliorate A β toxicity. *Proc Natl Acad Sci U S A* 2014;111(11):4013-8.
- [58] Robert A, Liu Y, Nguyen M, Meunier B. Regulation of copper and iron homeostasis by metal chelators: a possible chemotherapy for Alzheimer's disease. *Acc Chem Res* 2015;48(5):1332-9.
- [59] Ryan TM, Roberts BR, McColl G, Hare DJ, Doble PA, Li Q-X, et al. Stabilization of nontoxic A β -oligomers: insights into the mechanism of action of Hydroxyquinolines in Alzheimer's disease. *J Neurosci* 2015;35(7):2871-84.
- [60] Wang Z, Wang Y, Li W, Mao F, Sun Y, Huang L, et al. Design, synthesis, and evaluation of multitarget-directed Selenium-containing Clioquinol derivatives for the treatment of Alzheimer's disease. *ACS Chem Neurosci* 2014;5(10):952-62.
- [61] Pérez-Hernández J, Zaldívar-Machorro VJ, Villanueva-Porras D, Vega-Ávila E, Chavarría A. A potential alternative against neurodegenerative diseases: phytodrugs. *Oxid Med Cell Longev* 2016;2016:1-19.

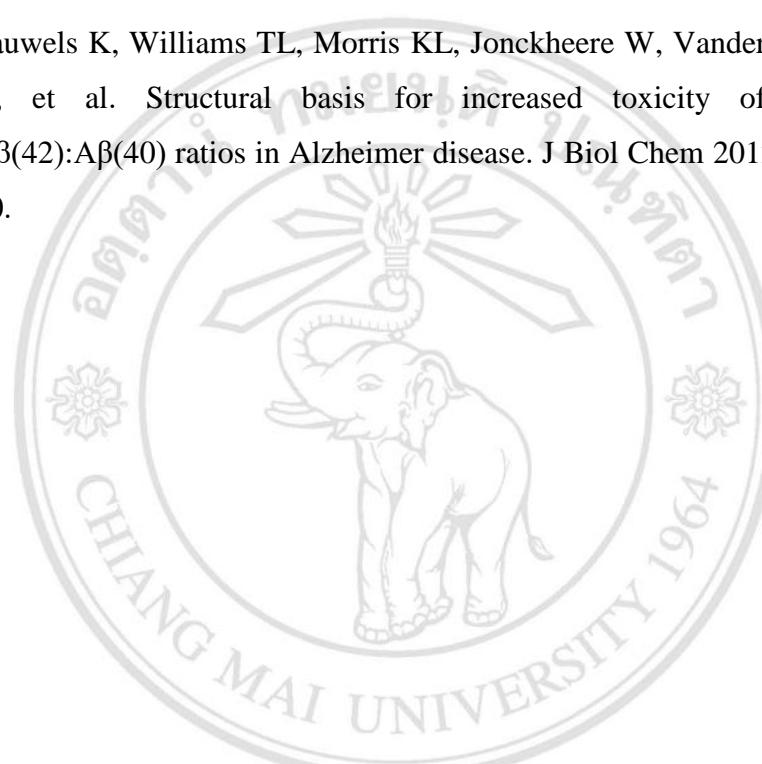
- [62] Ng YP, Or TCT, Ip NY. Plant alkaloids as drug leads for Alzheimer's disease. *Neurochem Int* 2015;89:260-70.
- [63] Konrath EL, Passos CdS, Klein-Júnior LC, Henriques AT. Alkaloids as a source of potential anticholinesterase inhibitors for the treatment of Alzheimer's disease. *J Pharm Pharmacol* 2013;65(12):1701-25.
- [64] Matharu B, Gibson G, Parsons R, Huckerby TN, Moore SA, Cooper LJ, et al. Galantamine inhibits beta-amyloid aggregation and cytotoxicity. *J Neurol Sci* 2009;280(1):49-58.
- [65] Melo JB, Sousa C, Garçao P, Oliveira CR, Agostinho P. Galantamine protects against oxidative stress induced by amyloid-beta peptide in cortical neurons. *Eur J Neurosci* 2009;29(3):455-64.
- [66] Asai M, Iwata N, Yoshikawa A, Aizaki Y, Ishiura S, Saido TC, et al. Berberine alters the processing of Alzheimer's amyloid precursor protein to decrease A β secretion. *Biochem Biophys Res Commun* 2007;352(2):498-502.
- [67] Durairajan SSK, Huang Y-Y, Yuen P-Y, Chen L-L, Kwok K-Y, Liu L-F, et al. Effects of Huanglian-Jie-Du-Tang and its modified formula on the modulation of Amyloid- β precursor protein processing in Alzheimer's disease models. *PLoS One* 2014;9(3):e92954.
- [68] Arendash GW, Mori T, Cao C, Mamcarz M, Runfeldt M, Dickson A, et al. Caffeine reverses cognitive impairment and decreases brain Amyloid- β levels in aged Alzheimer's disease mice. *J Alzheimers Dis* 2009;17(3):661-80.
- [69] Ngoungoue VLN, Schluesener J, Moundipa PF, Schluesener H. Natural polyphenols binding to amyloid: a broad class of compounds to treat different human amyloid diseases. *Mol Nutr Food Res* 2015;59(1):8-20.
- [70] Sgarbossa A. Natural biomolecules and protein aggregation: emerging strategies against amyloidogenesis. *Int J Mol Sci* 2012;13(12):17121-37.

- [71] Lee SJC, Nam E, Lee HJ, Savelieff MG, Lim MH. Towards an understanding of amyloid- β oligomers: characterization, toxicity mechanisms, and inhibitors *Chem Soc Rev* 2017;46(2):310-23.
- [72] Ringman JM, Frautschy SA, Cole GM, Masterman DL, Cummings JL. Cummings, a potential role of the curry spice curcumin in Alzheimer's disease,. *Curr Alzheimer Res* 2005;2(2):131-6.
- [73] Singh PK, Kotia V, Ghosh D, Mohite GM, Kumar A, Maji SK. Curcumin modulates alpha-synuclein aggregation and toxicity. *ACS Chem Neurosci* 2013;4(3):393-407.
- [74] Ehrnhoefer DE, Bieschke J, Boeddrich A, Herbst M, Masino L, Lurz R, et al. EGCG redirects amyloidogenic polypeptides into unstructured, off-pathway oligomers. *Nat Struct Mol Biol* 2008;15(6):558-66.
- [75] Feng Y, Wang X-p, Yang S-g, Wang Y-j, Zhang X, Du X-t, et al. Resveratrol inhibits beta-amyloid oligomeric cytotoxicity but does not prevent oligomer formation. *Neurotoxicology* 2009;30(6):986-95.
- [76] Jang J-H, Surh Y-J. Protective effect of resveratrol on beta-amyloid-induced oxidative PC12 cell death. *Free Radic Biol Med* 2003;34(8):1100-10.
- [77] Ono K, Hasegawa K, Naiki H, Yamada M. Anti-amyloidogenic activity of tannic acid and its activity to destabilize Alzheimer's β -amyloid fibrils in vitro. *Biochim Biophys Acta* 2004;1690(3):193-202.
- [78] Ono K, Condron MM, Ho L, Wang J, Zhao W, Pasinetti GM, et al. Effects of grape seed-derived polyphenols on Amyloid β -protein self-assembly and cytotoxicity. *J Biol Chem* 2008;283(47):32176-87.
- [79] Gazova Z, Siposova K, Kurin E, Mučaji P, Nagy M. Amyloid aggregation of lysozyme: The synergy study of red wine polyphenols. *Proteins* 2013;81(6):994-1004.

- [80] Li S, Leblanc RM. Aggregation of insulin at the interface. *J Phys Chem B* 2014;118(5):1181-8.
- [81] Orci L, Ravazzola M, Amherdt M, Madsen O, Vassalli J-D, Perrelet A. Direct identification of prohormone conversion site in insulin-secreting cells. *Cell* 1985;42(2):671-81.
- [82] Jørgensen AMM, Olsen HB, Balschmidt P, Led JJ. Solution structure of the superactive monomeric Des-[Phe(B25)] human insulin mutant: elucidation of the structural basis for the monomerization of Des-[Phe(B25)] insulin and the dimerization of native insulin. *J Mol Biol* 1996;257(3):684-99.
- [83] Groenning M, Frokjaer S, Vestergaard B. Formation mechanism of insulin fibrils and structural aspects of the insulin fibrillation process. *Curr Protein Pept Sci* 2009;10(5):509-28.
- [84] Kelly JW. The alternative conformations of amyloidogenic proteins and their multi-step assembly pathways. *Curr Opin Struct Biol* 1998;8(1):101-6.
- [85] Pasternack RF, Gibbs EJ, Sibley S, Woodard L, Hutchinson P, Genereux J, et al. Formation kinetics of insulin-based amyloid gels and the effect of added metalloporphyrins. *Biophys J* 2006;90(3):1033-42.
- [86] Hua Q-x, Weiss MA. Mechanism of Insulin Fibrillation: The structure of insulin under amyloidogenic conditions resembles a protein-folding intermediate. *J Biol Chem* 2004;279(20):21449-60.
- [87] Sluzky V, Klibanov AM, Langer R. Mechanism of insulin aggregation and stabilization in agitated aqueous solutions. *Biotechnol Bioeng* 1992;40(8):895-903.
- [88] Bekard IB, Dunstan DE. Tyrosine autofluorescence as a measure of bovine Insulin fibrillation. *Biophys J* 2009;97(9):2521-31.
- [89] Biancalana M, Koide S. Molecular mechanism of Thioflavin-T binding to amyloid fibrils. *Biochim Biophys Acta* 2010;1804(7):1405-12.

- [90] Voropai ES, Samtsov MP, Kaplevskii KN, Maskevich AA, Stepuro VI, Povarova OI, et al. Spectral properties of Thioflavin T and its complexes with amyloid fibrils. *J Appl Spectrosc* 2003;70(6):868-74.
- [91] Stsiapura VI, Maskevich AA, Kuzmitsky VA, Turoverov KK, Kuznetsova IM. Computational study of Thioflavin T Torsional relaxation in the excited state. *J Phys Chem A* 2007;111(22):4829-35.
- [92] Dzwolak W, Pecul M. Chiral bias of amyloid fibrils revealed by the twisted conformation of Thioflavin T: an induced circular dichroism/DFT study. *FEBS Lett* 2005;579(29):6601-3.
- [93] Sen P, Fatima S, Ahmad B, Khan RH. Interactions of Thioflavin T with serum albumins: spectroscopic analyses. *Spectrochim Acta A Mol Biomol Spectrosc* 2009;74(1):94-9.
- [94] Groenning M, Norrman M, Flink JM, van de Weert M, Bukrinsky JT, Schluckebier G, et al. Binding mode of Thioflavin T in insulin amyloid fibrils. *J Struct Biol* 2007;159(3):483-97.
- [95] Khurana R, Ionescu-Zanetti C, Pope M, Li J, Nielson L, Ramírez-Alvarado M, et al. A general model for amyloid fibril assembly based on morphological studies using atomic force microscopy. *Biophys J* 2003;85(2):1135-44.
- [96] Selkoe DJ. Cell biology of protein misfolding: The examples of Alzheimer's and Parkinson's diseases. *Nat Cell Biol* 2004;6:1054.
- [97] Kayed R, Head E, Thompson JL, McIntire TM, Milton SC, Cotman CW, et al. Common structure of soluble amyloid oligomers implies common mechanism of pathogenesis. *Science* 2003;300(5618):486-9.
- [98] Manno M, Craparo EF, Podestà A, Bulone D, Carrotta R, Martorana V, et al. Kinetics of Different Processes in Human Insulin Amyloid Formation. *J Mol Biol* 2007;366(1):258-74.

- [99] Kuperstein I, Broersen K, Benilova I, Rozenski J, Jonckheere W, Debulpae M, et al. Neurotoxicity of Alzheimer's disease A β peptides is induced by small changes in the A β_{42} to A β_{40} ratio. EMBO J 2010;29(19):3408-20.
- [100] Porat Y, Abramowitz A, Gazit E. Inhibition of amyloid fibril formation by polyphenols: structural similarity and aromatic interactions as a common inhibition mechanism. Chem Biol Drug Des 2006;67(1):27-37.
- [101] Pauwels K, Williams TL, Morris KL, Jonckheere W, Vandersteen A, Kelly G, et al. Structural basis for increased toxicity of pathological A β (42):A β (40) ratios in Alzheimer disease. J Biol Chem 2012;287(8):5650-60.



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