# Dennis J Selkoe

## List of Publications by Year in Descending Order



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| 112 papers | 33,424 citations | 63 h-index | 130 g-index |
| 130 ext. papers | 36,760 ext. citations | 16.4 avg, IF | 7.97 L-index |

| # | Paper | IF | Citations |
|---|---|---|---|
| 112 | Identification of the Aβ37/42 peptide ratio in CSF as an improved AD biomarker for Alzheimer's disease.. *Alzheimer's and Dementia*, **2022**, | 1.2 | 1 |
| 111 | A Brain-Penetrant Stearoyl-CoA Desaturase Inhibitor Reverses α-Synuclein Toxicity.. *Neurotherapeutics*, **2022**, 1 | 6.4 | 0 |
| 110 | Generation and application of semi-synthetic p-Tau181 calibrator for immunoassay calibration.. *Biochemical and Biophysical Research Communications*, **2022**, 611, 85-90 | 3.4 | 0 |
| 109 | An ultra-sensitive immunoassay detects and quantifies soluble Aβ oligomers in human plasma. *Alzheimer's and Dementia*, **2021**, | 1.2 | 3 |
| 108 | Alzheimer's drugs: Does reducing amyloid work?-Response. *Science*, **2021**, 374, 545-546 | 33.3 | |
| 107 | A Stearoyl-Coenzyme A Desaturase Inhibitor Prevents Multiple Parkinson Disease Phenotypes in α-Synuclein Mice. *Annals of Neurology*, **2021**, 89, 74-90 | 9.4 | 16 |
| 106 | Hydrophilic loop 1 of Presenilin-1 and the APP GxxxG transmembrane motif regulate γ-secretase function in generating Alzheimer-causing Aβ peptides. *Journal of Biological Chemistry*, **2021**, 296, 100393 | 5.4 | 8 |
| 105 | Altered conformation of α-synuclein drives dysfunction of synaptic vesicles in a synaptosomal model of Parkinson's disease. *Cell Reports*, **2021**, 36, 109333 | 10.6 | 5 |
| 104 | Wild-type GBA1 increases the α-synuclein tetramer-monomer ratio, reduces lipid-rich aggregates, and attenuates motor and cognitive deficits in mice. *Proceedings of the National Academy of Sciences of the United States of America*, **2021**, 118, | 11.5 | 5 |
| 103 | Non-denaturing affinity purification of soluble oligomeric Aβ from human brain using a novel calcium-sensitive monoclonal antibody.. *Alzheimer's and Dementia*, **2021**, 17 Suppl 3, e051725 | 1.2 | |
| 102 | Predicting development of AD clinical symptoms and their progression through a collection of novel plasma Aβ immunoassays. *Alzheimer's and Dementia*, **2020**, 16, e043670 | 1.2 | 1 |
| 101 | Plasma levels of an N-terminal tau fragment are highly associated with future cognitive decline and neurodegeneration in clinically normal elderly. *Alzheimer's and Dementia*, **2020**, 16, e045261 | 1.2 | |
| 100 | A mechanistic hypothesis for the impairment of synaptic plasticity by soluble Aβ oligomers from Alzheimer's brain. *Journal of Neurochemistry*, **2020**, 154, 583-597 | 6 | 68 |
| 99 | Amyloid β-protein and beyond: the path forward in Alzheimer's disease. *Current Opinion in Neurobiology*, **2020**, 61, 116-124 | 7.6 | 56 |
| 98 | Transcriptomic correlates of neurite degeneration due to human brain-derived Aβ and protection by clinical anti-Aβ antibodies. *Alzheimer's and Dementia*, **2020**, 16, e043057 | 1.2 | |
| 97 | Environmental enrichment prevents Aβ oligomer-induced synaptic dysfunction through mirna-132 and hdac3 signaling pathways. *Neurobiology of Disease*, **2020**, 134, 104617 | 7.5 | 19 |
| 96 | Plasma N-terminal tau fragment levels predict future cognitive decline and neurodegeneration in healthy elderly individuals. *Nature Communications*, **2020**, 11, 6024 | 17.4 | 18 |

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| 95 | Potential human transmission of amyloid □pathology: surveillance and risks. *Lancet Neurology, The*, **2020**, 19, 872-878 | 24.1 | 23 |
| 94 | Analysis of □synuclein species enriched from cerebral cortex of humans with sporadic dementia with Lewy bodies. *Brain Communications*, **2020**, 2, fcaa010 | 4.5 | 12 |
| 93 | Alzheimer disease and aducanumab: adjusting our approach. *Nature Reviews Neurology*, **2019**, 15, 365-366 | 16.5 | 107 |
| 92 | Target engagement in an alzheimer trial: Crenezumab lowers amyloid □oligomers in cerebrospinal fluid. *Annals of Neurology*, **2019**, 86, 215-224 | 9.4 | 41 |
| 91 | Identification of neurotoxic cross-linked amyloid-□dimers in the Alzheimer's brain. *Brain*, **2019**, 142, 1441-1457 | 11.5 | 40 |
| 90 | Multiple BACE1 inhibitors abnormally increase the BACE1 protein level in neurons by prolonging its half-life. *Alzheimer's and Dementia*, **2019**, 15, 1183-1194 | 1.2 | 12 |
| 89 | miR-212 and miR-132 Are Downregulated in Neurally Derived Plasma Exosomes of Alzheimer's Patients. *Frontiers in Neuroscience*, **2019**, 13, 1208 | 5.1 | 61 |
| 88 | Cell models of lipid-rich □synuclein aggregation validate known modifiers of □synuclein biology and identify stearoyl-CoA desaturase. *Proceedings of the National Academy of Sciences of the United States of America*, **2019**, 116, 20760-20769 | 11.5 | 34 |
| 87 | Learnings about the complexity of extracellular tau aid development of a blood-based screen for Alzheimer's disease. *Alzheimer's and Dementia*, **2019**, 15, 487-496 | 1.2 | 60 |
| 86 | A cellular complex of BACE1 and □secretase sequentially generates A□from its full-length precursor. *Journal of Cell Biology*, **2019**, 218, 644-663 | 7.3 | 39 |
| 85 | Detection of Aggregation-Competent Tau in Neuron-Derived Extracellular Vesicles. *International Journal of Molecular Sciences*, **2018**, 19, | 6.3 | 86 |
| 84 | An in vitro paradigm to assess potential anti-A□antibodies for Alzheimer's disease. *Nature Communications*, **2018**, 9, 2676 | 17.4 | 26 |
| 83 | Enriched environment enhances □adrenergic signaling to prevent microglia inflammation by□amyloid-□*EMBO Molecular Medicine*, **2018**, 10, | 12 | 30 |
| 82 | Decoding the synaptic dysfunction of bioactive human AD brain soluble A□to inspire novel therapeutic avenues for Alzheimer's disease. *Acta Neuropathologica Communications*, **2018**, 6, 121 | 7.3 | 28 |
| 81 | DT-02-03: TARGET ENGAGEMENT IN AN AD TRIAL: CRENEZUMAB LOWERS A□OLIGOMER LEVELS IN CSF **2018**, 14, P1669-P1670 | | 3 |
| 80 | Light at the End of the Amyloid Tunnel. *Biochemistry*, **2018**, 57, 5921-5922 | 3.2 | 11 |
| 79 | Abrogating Native □Synuclein Tetramers in Mice Causes a L-DOPA-Responsive Motor Syndrome Closely Resembling Parkinson's Disease. *Neuron*, **2018**, 100, 75-90.e5 | 13.9 | 71 |
| 78 | □2-Adrenoreceptor is a regulator of the □synuclein gene driving risk of Parkinson's disease. *Science* , **2017**, 357, 891-898 | 33.3 | 238 |

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| 77 | A user's guide for α-synuclein biomarker studies in biological fluids: Perianalytical considerations. *Movement Disorders*, **2017**, 32, 1117-1130 | 7 | 35 |
| 76 | Cell-type Dependent Alzheimer's Disease Phenotypes: Probing the Biology of Selective Neuronal Vulnerability. *Stem Cell Reports*, **2017**, 9, 1868-1884 | 8 | 43 |
| 75 | Large Soluble Oligomers of Amyloid β-Protein from Alzheimer Brain Are Far Less Neuroactive Than the Smaller Oligomers to Which They Dissociate. *Journal of Neuroscience*, **2017**, 37, 152-163 | 6.6 | 185 |
| 74 | Caspase-1 clipping causes complications for α-synuclein. *Proceedings of the National Academy of Sciences of the United States of America*, **2016**, 113, 9958-60 | 11.5 | 6 |
| 73 | The amyloid hypothesis of Alzheimer's disease at 25 years. *EMBO Molecular Medicine*, **2016**, 8, 595-608 | 12 | 2876 |
| 72 | Soluble Aβ oligomers impair hippocampal LTP by disrupting glutamatergic/GABAergic balance. *Neurobiology of Disease*, **2016**, 85, 111-121 | 7.5 | 92 |
| 71 | A critical appraisal of the pathogenic protein spread hypothesis of neurodegeneration. *Nature Reviews Neuroscience*, **2016**, 17, 251-60 | 13.5 | 198 |
| 70 | Nicastrin functions to sterically hinder γ-secretase-substrate interactions driven by substrate transmembrane domain. *Proceedings of the National Academy of Sciences of the United States of America*, **2016**, 113, E509-18 | 11.5 | 95 |
| 69 | The amyloid-beta forming tripeptide cleavage mechanism of γ-secretase. *ELife*, **2016**, 5, | 8.9 | 74 |
| 68 | A highly sensitive novel immunoassay specifically detects low levels of soluble Aβ oligomers in human cerebrospinal fluid. *Alzheimer's Research and Therapy*, **2015**, 7, 14 | 9 | 63 |
| 67 | C-Terminally Truncated Forms of Tau, But Not Full-Length Tau or Its C-Terminal Fragments, Are Released from Neurons Independently of Cell Death. *Journal of Neuroscience*, **2015**, 35, 10851-65 | 6.6 | 106 |
| 66 | Association between α-synuclein blood transcripts and early, neuroimaging-supported Parkinson's disease. *Brain*, **2015**, 138, 2659-71 | 11.2 | 47 |
| 65 | Purification of α-synuclein from human brain reveals an instability of endogenous multimers as the protein approaches purity. *Biochemistry*, **2015**, 54, 279-92 | 3.2 | 56 |
| 64 | P4-221: Nicastrin functions as a molecular gatekeeper to a high-affinity γ-secretase-substrate interaction driven by substrate transmembrane domain **2015**, 11, P864-P864 | | |
| 63 | Systematic analysis of time-dependent neural effects of soluble amyloid β oligomers in culture and in vivo: Prevention by scyllo-inositol. *Neurobiology of Disease*, **2015**, 82, 152-163 | 7.5 | 34 |
| 62 | Physical and functional interaction between the β- and γ-secretases: A new model of regulated intramembrane proteolysis. *Journal of Cell Biology*, **2015**, 211, 1157-76 | 7.3 | 45 |
| 61 | γ-Secretase: a horseshoe structure brings good luck. *Cell*, **2014**, 158, 247-249 | 56.2 | 6 |
| 60 | Secreted amyloid β-proteins in a cell culture model include N-terminally extended peptides that impair synaptic plasticity. *Biochemistry*, **2014**, 53, 3908-21 | 3.2 | 71 |

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| 59 | SnapShot: pathobiology of Alzheimer's disease. *Cell*, **2013**, 154, 468-468.e1 | 56.2 | 35 |
| 58 | The therapeutics of Alzheimer's disease: where we stand and where we are heading. *Annals of Neurology*, **2013**, 74, 328-36 | 9.4 | 91 |
| 57 | Environmental novelty activates β2-adrenergic signaling to prevent the impairment of hippocampal LTP by Aβ oligomers. *Neuron*, **2013**, 77, 929-41 | 13.9 | 122 |
| 56 | New ELISAs with high specificity for soluble oligomers of amyloid β-protein detect natural Aβ oligomers in human brain but not CSF. *Alzheimer's and Dementia*, **2013**, 9, 99-112 | 1.2 | 92 |
| 55 | Preventing Alzheimer's disease. *Science*, **2012**, 337, 1488-92 | 33.3 | 280 |
| 54 | Resolving controversies on the path to Alzheimer's therapeutics. *Nature Medicine*, **2011**, 17, 1060-5 | 50.5 | 383 |
| 53 | β-Synuclein occurs physiologically as a helically folded tetramer that resists aggregation. *Nature*, **2011**, 477, 107-10 | 50.4 | 849 |
| 52 | Soluble amyloid beta-protein dimers isolated from Alzheimer cortex directly induce Tau hyperphosphorylation and neuritic degeneration. *Proceedings of the National Academy of Sciences of the United States of America*, **2011**, 108, 5819-24 | 11.5 | 641 |
| 51 | Alzheimer's disease. *Cold Spring Harbor Perspectives in Biology*, **2011**, 3, | 10.2 | 310 |
| 50 | Aph-1 associates directly with full-length and C-terminal fragments of gamma-secretase substrates. *Journal of Biological Chemistry*, **2010**, 285, 11378-91 | 5.4 | 28 |
| 49 | Identification of beta-secretase (BACE1) substrates using quantitative proteomics. *PLoS ONE*, **2009**, 4, e8477 | 3.7 | 140 |
| 48 | Amyloid-beta protein dimers isolated directly from Alzheimer's brains impair synaptic plasticity and memory. *Nature Medicine*, **2008**, 14, 837-42 | 50.5 | 2779 |
| 47 | Soluble oligomers of the amyloid beta-protein impair synaptic plasticity and behavior. *Behavioural Brain Research*, **2008**, 192, 106-13 | 3.4 | 844 |
| 46 | Rapid purification of active gamma-secretase, an intramembrane protease implicated in Alzheimer's disease. *Journal of Neurochemistry*, **2008**, 104, 210-20 | 6 | 35 |
| 45 | Developing preventive therapies for chronic diseases: lessons learned from Alzheimer's disease. *Nutrition Reviews*, **2007**, 65, S239-43 | 6.4 | 29 |
| 44 | A beta oligomers - a decade of discovery. *Journal of Neurochemistry*, **2007**, 101, 1172-84 | 6 | 1609 |
| 43 | Presenilin: running with scissors in the membrane. *Cell*, **2007**, 131, 215-21 | 56.2 | 313 |
| 42 | Alzheimer disease: mechanistic understanding predicts novel therapies. *Annals of Internal Medicine*, **2004**, 140, 627-38 | 8 | 206 |

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| 41 | Detergent-dependent dissociation of active gamma-secretase reveals an interaction between Pen-2 and PS1-NTF and offers a model for subunit organization within the complex. *Biochemistry*, **2004**, 43, 323-33 | 3.2 | 122 |
| 40 | Purification and characterization of the human gamma-secretase complex. *Biochemistry*, **2004**, 43, 9774-89 | 3.2 | 206 |
| 39 | Aging, amyloid, and Alzheimer's disease: a perspective in honor of Carl Cotman. *Neurochemical Research*, **2003**, 28, 1705-13 | 4.6 | 65 |
| 38 | Folding proteins in fatal ways. *Nature*, **2003**, 426, 900-4 | 50.4 | 1191 |
| 37 | Gamma-secretase is a membrane protein complex comprised of presenilin, nicastrin, Aph-1, and Pen-2. *Proceedings of the National Academy of Sciences of the United States of America*, **2003**, 100, 6382-7 | 11.5 | 665 |
| 36 | Toward a remembrance of things past: deciphering Alzheimer disease. *Harvey Lectures*, **2003**, 99, 23-45 | | 11 |
| 35 | Intraneuronal A□42 accumulation in Down syndrome brain. *Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis* , **2002**, 9, 88-102 | 2.7 | 209 |
| 34 | Deciphering the genetic basis of Alzheimer's disease. *Annual Review of Genomics and Human Genetics*, **2002**, 3, 67-99 | 9.7 | 226 |
| 33 | Activity-dependent isolation of the presenilin- gamma -secretase complex reveals nicastrin and a gamma substrate. *Proceedings of the National Academy of Sciences of the United States of America*, **2002**, 99, 2720-5 | 11.5 | 346 |
| 32 | Alzheimer's disease is a synaptic failure. *Science*, **2002**, 298, 789-91 | 33.3 | 3246 |
| 31 | Deciphering the genesis and fate of amyloid beta-protein yields novel therapies for Alzheimer disease. *Journal of Clinical Investigation*, **2002**, 110, 1375-81 | 15.9 | 69 |
| 30 | Alzheimer's disease results from the cerebral accumulation and cytotoxicity of amyloid beta-protein. *Journal of Alzheimer's Disease*, **2001**, 3, 75-80 | 4.3 | 437 |
| 29 | Toward a comprehensive theory for Alzheimer's disease. Hypothesis: Alzheimer's disease is caused by the cerebral accumulation and cytotoxicity of amyloid beta-protein. *Annals of the New York Academy of Sciences*, **2000**, 924, 17-25 | 6.5 | 481 |
| 28 | Rapid Notch1 nuclear translocation after ligand binding depends on presenilin-associated gamma-secretase activity. *Annals of the New York Academy of Sciences*, **2000**, 920, 223-6 | 6.5 | 28 |
| 27 | Nasal administration of amyloid-□peptide decreases cerebral amyloid burden in a mouse model of Alzheimer's disease. *Annals of Neurology*, **2000**, 48, 567-579 | 9.4 | 295 |
| 26 | An improved method of preparing the amyloid beta-protein for fibrillogenesis and neurotoxicity experiments. *Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis*, **2000**, 7, 166-78 | 2.7 | 211 |
| 25 | Nasal administration of amyloid-□peptide decreases cerebral amyloid burden in a mouse model of Alzheimer's disease **2000**, 48, 567 | | 2 |
| 24 | Two transmembrane aspartates in presenilin-1 required for presenilin endoproteolysis and gamma-secretase activity. *Nature*, **1999**, 398, 513-7 | 50.4 | 1705 |

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|---|---|---|---|
| 23 | Translating cell biology into therapeutic advances in Alzheimer's disease. *Nature*, **1999**, 399, A23-31 | 50.4 | 1214 |
| 22 | Effects of the amyloid precursor protein Glu693-Gln Dutch mutation on the production and stability of amyloid protein. *Biochemical Journal*, **1999**, 340, 703-709 | 3.8 | 54 |
| 21 | A substrate-based difluoro ketone selectively inhibits Alzheimer's gamma-secretase activity. *Journal of Medicinal Chemistry*, **1998**, 41, 6-9 | 8.3 | 204 |
| 20 | Presenilin 1 regulates the processing of beta-amyloid precursor protein C-terminal fragments and the generation of amyloid beta-protein in endoplasmic reticulum and Golgi. *Biochemistry*, **1998**, 37, 16465-71 | 3.2 | 166 |
| 19 | Additive effects of PS1 and APP mutations on secretion of the 42-residue amyloid beta-protein. *Neurobiology of Disease*, **1998**, 5, 107-16 | 7.5 | 87 |
| 18 | Mutant presenilins of Alzheimer's disease increase production of 42-residue amyloid beta-protein in both transfected cells and transgenic mice. *Nature Medicine*, **1997**, 3, 67-72 | 50.5 | 1183 |
| 17 | The E280A presenilin 1 Alzheimer mutation produces increased A beta 42 deposition and severe cerebellar pathology. *Nature Medicine*, **1996**, 2, 1146-50 | 50.5 | 440 |
| 16 | Alzheimer's amyloid of another flavour. *Nature Medicine*, **1995**, 1, 998-9 | 50.5 | 11 |
| 15 | The Swedish mutation causes early-onset Alzheimer's disease by beta-secretase cleavage within the secretory pathway. *Nature Medicine*, **1995**, 1, 1291-6 | 50.5 | 466 |
| 14 | Aggregation of secreted amyloid beta-protein into sodium dodecyl sulfate-stable oligomers in cell culture. *Journal of Biological Chemistry*, **1995**, 270, 9564-70 | 5.4 | 306 |
| 13 | Normal cellular processing of the beta-amyloid precursor protein results in the secretion of the amyloid beta peptide and related molecules. *Annals of the New York Academy of Sciences*, **1993**, 695, 109-16 | 6.5 | 94 |
| 12 | Targeting of cell-surface beta-amyloid precursor protein to lysosomes: alternative processing into amyloid-bearing fragments. *Nature*, **1992**, 357, 500-3 | 50.4 | 817 |
| 11 | Amyloid beta-peptide is produced by cultured cells during normal metabolism. *Nature*, **1992**, 359, 322-5 | 50.4 | 1791 |
| 10 | Mutation of the beta-amyloid precursor protein in familial Alzheimer's disease increases beta-protein production. *Nature*, **1992**, 360, 672-4 | 50.4 | 1594 |
| 9 | Amyloid beta-protein deposition in tissues other than brain in Alzheimer's disease. *Nature*, **1989**, 341, 226-30 | 50.4 | 381 |
| 8 | The deposition of amyloid proteins in the aging mammalian brain: implications for Alzheimer's disease. *Annals of Medicine*, **1989**, 21, 73-6 | 1.5 | 53 |
| 7 | Studies on the Relationship between Plasma Proteins and Amyloid Fibrils Found in Alzheimer's Disease. *Annals of the New York Academy of Sciences*, **1988**, 529, 233-235 | 6.5 | |
| 6 | Neurofibrillary tangles and senile plaques in aged bears. *Journal of Neuropathology and Experimental Neurology*, **1988**, 47, 629-41 | 3.1 | 102 |

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| 5 | HPLC Analysis of Proteins from Alzheimer Paired Helical Filaments. *Annals of the New York Academy of Sciences*, **1987**, 494, 369-372 | 6.5 | |
| 4 | Tau antisera recognize neurofibrillary tangles in a range of neurodegenerative disorders. *Annals of Neurology*, **1987**, 22, 514-20 | 9.4 | 107 |
| 3 | Isolation of low-molecular-weight proteins from amyloid plaque fibers in Alzheimer's disease. *Journal of Neurochemistry*, **1986**, 46, 1820-34 | 6 | 310 |
| 2 | Neurofibrillary degeneration of cholinergic and noncholinergic neurons of the basal forebrain in Alzheimer's disease. *Annals of Neurology*, **1986**, 20, 482-8 | 9.4 | 89 |
| 1 | Antibodies to paired helical filaments in Alzheimer's disease do not recognize normal brain proteins. *Nature*, **1983**, 304, 727-30 | 50.4 | 259 |