Additional Table 1 Natural polyphenols effects in Alzheimer's disease (AD) and Parkinson's disease (PD): *in vitro* and *in vivo* studies cited in the paper.

Natural polyphenols	Research models	Doses and effects	References
Natural polyphenols effects in AD			
In vitro studies			
Myricetin, Morin, Quercetin, Kaempferol, (+)-catechin, (–)-epicatechin, Tannic acid	Polymerization & destabilization assays (fluorescent spectroscopic analysis)	EC_{50}: 0.1–1.0 μM Dose-dependently inhibition of fibrillary β-amyloid (fAβ) formation and extension from fresh β-amyloid $(Aβ)_{1-40}$ and $Aβ_{1-42}$ and dose-dependently destabilization of preformed fAbs.	Ono et al. (2003) Ono et al. (2004)
Resveratrol, Piceid, Resveratrol diglucoside, Piceatannol, Astringin, Viniferin, Curcumin	Polymerization assays (ultraviolet (UV)-visible measurements and electron microscopy)	Resveratrol EC_{50}: 5.6 μM; Piceid EC_{50}: 4.7 μM Inhibition in $Aβ_{25-35}$ fibrils formation stronger with these two polyphenols	Riviere et al. (2007)
Verbascoside & its esterified derivative	Metal-free and metal-induced aggregation *in vitro* (spectroscopy)	Concentration used: 50 μM Interaction of compounds simultaneously with both Aβ and metal ions. Modulation of the early steps in aggregation of both metal-free Aβ and metal-Aβ. Ability of compounds to disaggregate Cu(II)–$Aβ_{40}$ aggregates	Korshavn et al. (2015)
Resveratrol, trans ε-viniferin	Aggregation and disaggregation assays (scanning electron microscopy)	Concentration used: 1 μM Inhibition of $Aβ_{1-42}$ aggregation and induction of $Aβ_{1-42}$ disaggregation by these polyphenols with better efficiency for trans ε-viniferin	Vion et al. (2017)
Resveratrol, ε-viniferin glucoside	Aggregation assays (UV-visible measurements) ε-viniferin glucoside/Aβ complex formation assay (ESI mass spectrometric analysis)	Concentrations used: 1, 5 and 10 μM Inhibition of $Aβ_{25-35}$, $Aβ_{1-40}$ and $Aβ_{1-42}$ fibril formation with better efficiency for ε-viniferin glucoside. Interaction between ε-viniferin glucoside and $Aβ_{1-40}$	Richard et al. (2011)
Rosmarinic acid	Docking stimulation	EC_{50}: 20.3 μM Directly interaction of rosmarinic acid with $Aβ_{1-42}$	Taguchi et al. (2017)
EGCG	Synthesis of peptide and *in vitro* study	Binding to phosphorylation site of tau and inhibition of its aggregation	Gueroux et al. (2017)
Cyanine dye family member (C11)	Disaggregation assay	Concentration used: 0.001 μM Reduction of aggregated tau levels	Duff et al. (2010)
GSPE (grape seed-derives polyphenol extract)	Circular dichroism spectroscopy and electron microscopy	Potential interference with the assembly of tau peptides into neurotoxic aggregates	Wang et al. (2010)
GSPE	Study of the effect of GSPE exposure on the ultrastructure of paired helical filaments (PHFs) isolated from AD brain by transmission electron microscopy	Induction of dose- and time-dependent alterations in the morphology of PHFs with partial disintegration of filaments.	Ksiezak-Reding et al. (2012)
Rosmarinic acid	ThT binding fluorescence assay of tau protein	Concentration used: 10–100 μM Prevention of β-sheet assembly by RA, by direct interaction with tau	Cornejo et al. (2017)
In vivo studies			
Curcumin	Aged APPsweTg2576	Chronic curcumin (500 ppm) injection for 5 months Crossing the blood brain barrier by curcumin. Bounding to amyloid plaques. Reduction of amyloid levels and plaques formation	Yang et al. (2005)
Curcumin	APP(Swe)/PS1dE9	Daily tail vein injections [7.5 mg/kg/day in phosphate-buffered saline (PBS)] for 7 days Crossing the blood brain barrier by curcumin. Labelling senile plaques. Reduction of existing amyloid plaques. Partially restoration of distorted neurites	Garcia-Alloza et al. (2007)
Polyphenol-rich grape seed extract	APP(Swe)/PS1dE9	Feeding during 6 months between 3 and 9 months of age Decrease in Aβ deposition	Wang et al. (2009)
Diets including 0.5% phenolic compounds (Myricetin, nordihydroguaiaretic acid (NDGA) or rosmarinic acid)	Old female APPsweTg2576	Feeding during 10 months from the age of 5 months Decrease in amyloid plaques	Hamaguchi et al. (2009)
Resveratrol	AD Tg19959 mice	Administration by food during 45 days Decrease in plaque formation in a region specific manner	Karuppagounder et al. (2009)
GSPE	TMHT mouse model of AD	Oral administration (200 mg/kg/day) Decrease in the development of AD type tau neuropathology	Wang et al. (2010)
EGCG	Old APPsweTg2576	Daily intraperitoneal injections (20 mg/kg) between 12 and 14 months Decrease in insoluble hyperphosphorylated tau in brain	Rezai-Zadeh et al. (2005)
EGCG	Old APPsweTg2576	Oral treatment (50 mg/kg in drinking water) between 12 and 14 months Decrease in insoluble hyperphosphorylated tau in brain	Rezai-Zadeh et al. (2008)

Additional Table 1 Continued.

Natural polyphenols	Research models	Doses and effects	References
Natural polyphenols effects in PD			
In vitro studies			
13 antioxidants	Formation and destabilization of preformedα-synuclein (α-syn) fibrils assays (fluorescence spectroscopy with thoflavin S and electron microscopy)	EC_{50}: 0.012–18.820 μM Anti fibrillogenic and fibril-destabilizing activity	Ono and Yamada (2006)
39 polyphenols including baicalein, delphinidin, gallocatechin gallate and rosmarinic acid	α-syn filament formation assay	IC_{50}: 2.5–9.8 μM Strong inhibition of α-syn filament formation	Masuda et al. (2006)
48 flavonoids belonging to several classes	α-syn aggregation assay	Concentration used: 50 μM Inhibition of the aggregation of α-syn by stabilizing non-pathogenic protein conformation	Meng et al. (2010)
14 polyphenols	α -syn filament formation assay	Concentration used: 10–50 μM Determination of key molecular scaffolds most effective in inhibiting oligomer formation by α-syn and disaggregating pre-formed oligomers	Caruana et al. (2011)
NDGA, morin, baicalein and apigenin	Evaluation of protective role against membrane perturbation	Concentration used: 20 μM Strong protection against membrane perturbation induced by aggregated WT and mutant α-syn	Caruana et al. (2012)
Baicalein	Study of aggregation inhibition mechanism	Concentration used: 50 μM Formation of Schiffbase	Meng et al. (2009)
Baicalein	Study of aggregation inhibition mechanism (circular discroism and fourier transform infra red analysis)	Concentration used: 100 μM Stabilization of oligomers by β-sheet enrichment	Hong et al. (2008)
Curcumin and curcumin derivatives	α-syn aggregation and neurotoxicity assays (Biophysical, imaging techniques, dot blot and cell based assays)	Concentration used: 210 μM Inhibition of α-syn aggregation and reduction of α-syn associated neurotoxicity by curcumin pyrazole derivatives	Ahsan et al. (2015)
EGCG	Binding and cytotoxicity assays	Concentration used: 100 μM Binding of EGCG to the natively undfolded polypeptides, forming complexes and decreasing their cytotoxicity	Ehrnhoefer et al. (2008)
EGCG	Binding and cytotoxicity assays	Concentrations used: 0.2–6.0 μM Binding of EGCG to the oligomeric state of α-syn, destabilization of it and decreasing its cytotoxicity	Lorenzen et al. (2014)
EGCG	Cytotoxicity assays	Concentrations used: 10, 30, 50 and 70 μM Facilitation of the conversion of "active" oligomers into fibrils	Yang et al. (2017)
Gallic acid	Oligomers formation assay	Concentration used: 40 μM Inhibition of fibrils of α-syn formation and reduction of formation of oligomers rate by binding to soluble and non-toxic oligomers and stabilization of their structure	Liu et al. (2014)
Piceatannol	α-syn fibrils formation and destabilization of preformed filaments assays, toxicity assay in cellular model	Concentrations used: 100 and 200 μM Inhibition of α-syn fibrils formation and destabilization of preformed filaments. Protection of PC12 against α-syn-induced toxicity	Temsamani et al. (2016)
Resveratrol	α-syn assay, toxicity assay in cellular model	Concentration used: 7.5 μM Protection of SK-N-BE from the toxicity arising grom aggregation-prone protein α-syn(A30P)	Albani et al. (2009)
Resveratrol	α-syn-expressing PC12 cell lines Pharmacological induction of autophagy by resveratrol	Concentration used: 50 μM Increase degradation of α-syn	Wu et al. (2011)
Curcumin analog	Synphilin-1 aggregation assay in a cellular model (SHSY-5Y cells)	Concentration used: 1 μM Decrease in synphilin-1 aggregation by preventing covalent modifications and by maintaining the expression of the protein disulfide isomerase	Pal et al. (2011)

EC_{50}: Concentration for 50% of maximal effect; EGCG: epigallocatechin 3-gallate.