

**Figure S1.** Mechanisms of neuroinflammation in Alzheimer's disease and drug targets [Obtained and reproduced without any changes from [1] and *Frontiers in Pharmacology* in accordance with **Creative Commons Attribution License**] (CC BY)].

**Key:** IDE, insulin-degrading enzyme; Aβ, amyloid beta; IL-1α, interleukin 1α; TNF-α, tumour necrosis factor-α; C3, complement component 3; C1q, complement protein 1q; TREM2, triggering receptor expressed on myeloid cells 2; ITAM, immunoreceptor tyrosine-based activation motif; SYK, spleen tyrosine kinase; P, phosphate; PI3K, phosphatidylinositol 3-kinase; NFκB, nuclear factor kappa β; RIPK1, receptor-interacting serine/threonine-protein kinase 1; Cst7, cystatin F gene; RTK, receptor tyrosine kinase; PDK1, phosphoinositide-dependent kinase 1; mTOR, mammalian Target of Rapamycin; Akt, protein kinase B; GSK-3β, glycogen synthase kinase 3β; TSC, tuberous sclerosis complex; AMP, adenosine monophosphate; AMPK, AMP-activated protein kinase; SIRT1, silent information regulator type 1; BACE1, β-secretase 1; cAMP, cyclic adenosine monophosphate; PKA, protein kinase A; CREB, cAMP response element binding protein; NMDAR, NMDA receptor; Ca<sup>2+</sup>/Calmodulin-dependent protein kinase II (CAMKII); LIMK1, LIM kinase 1; BDNF, brain-derived neurotrophic factor. Asterisk (\*) in the diagram indicates uncertain changes of activity in AD.

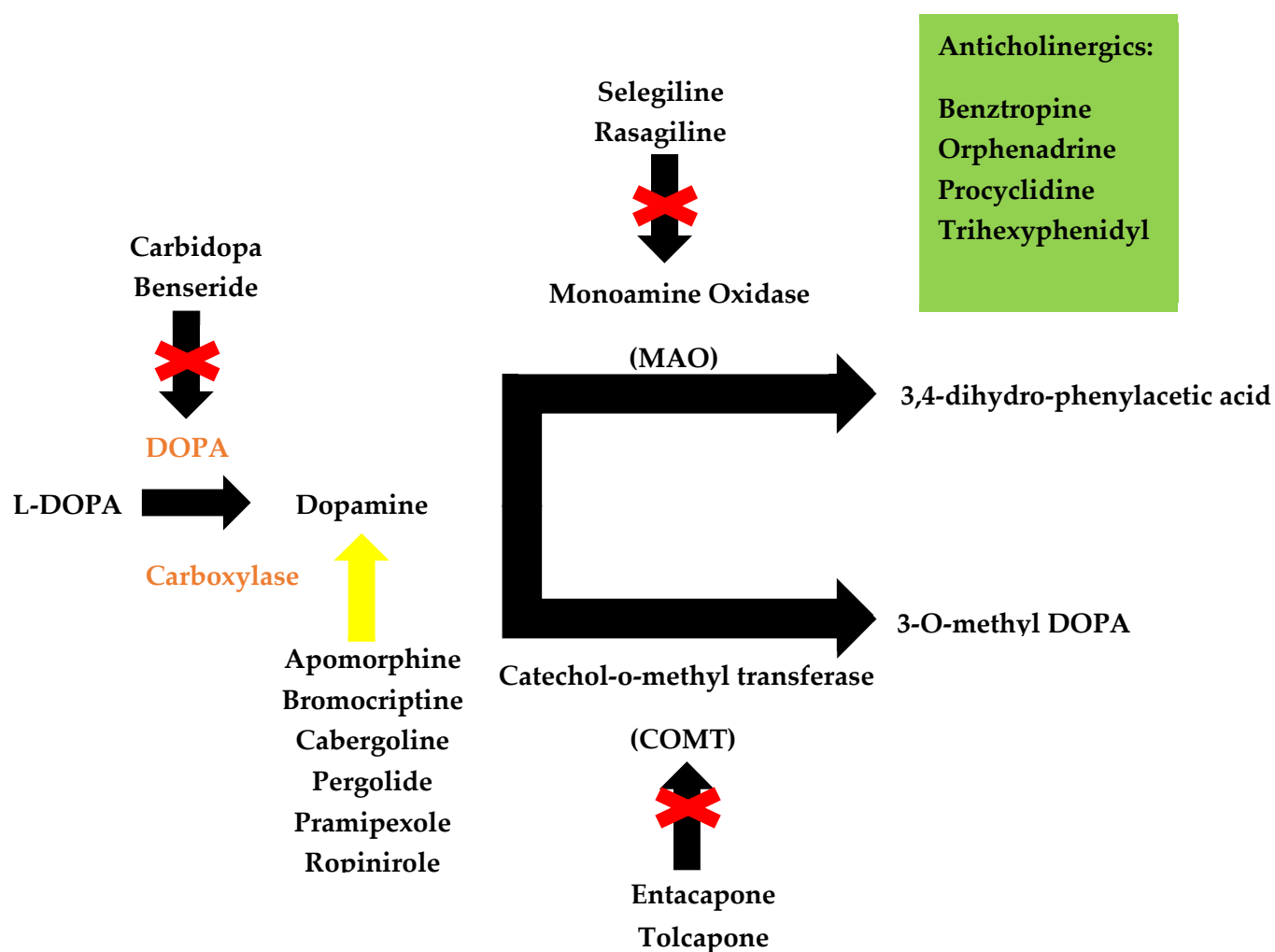
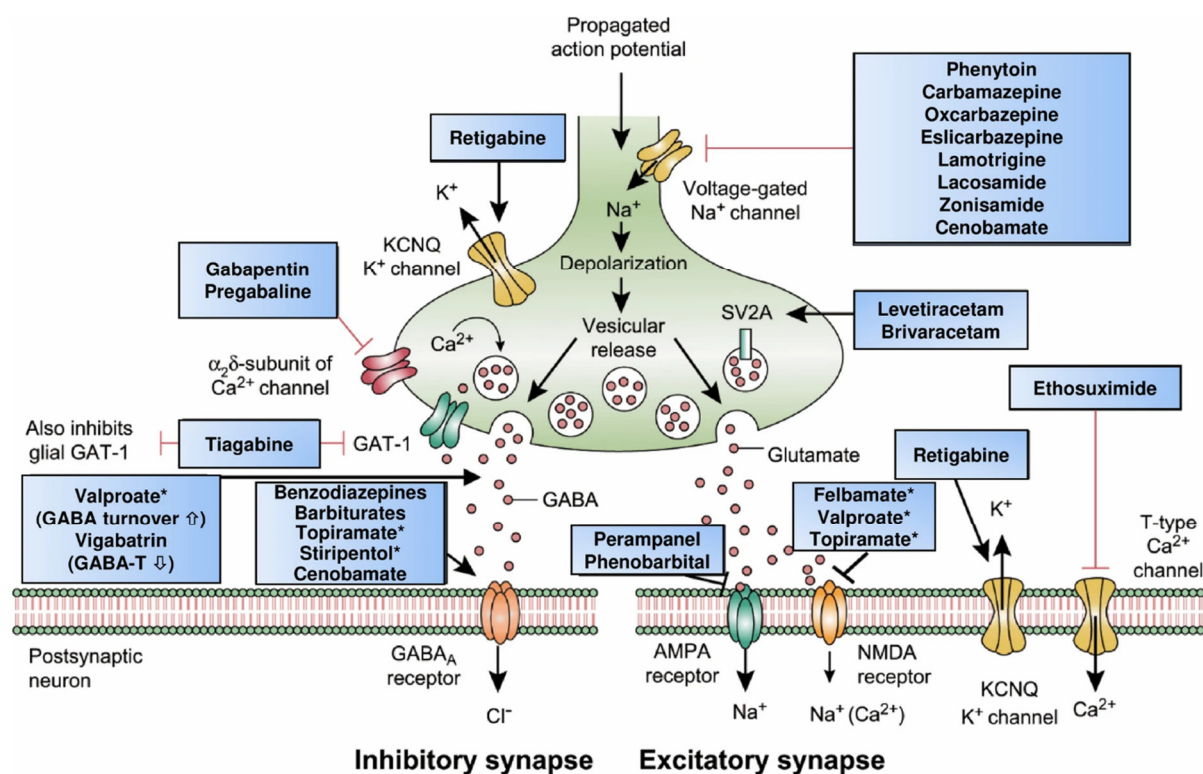
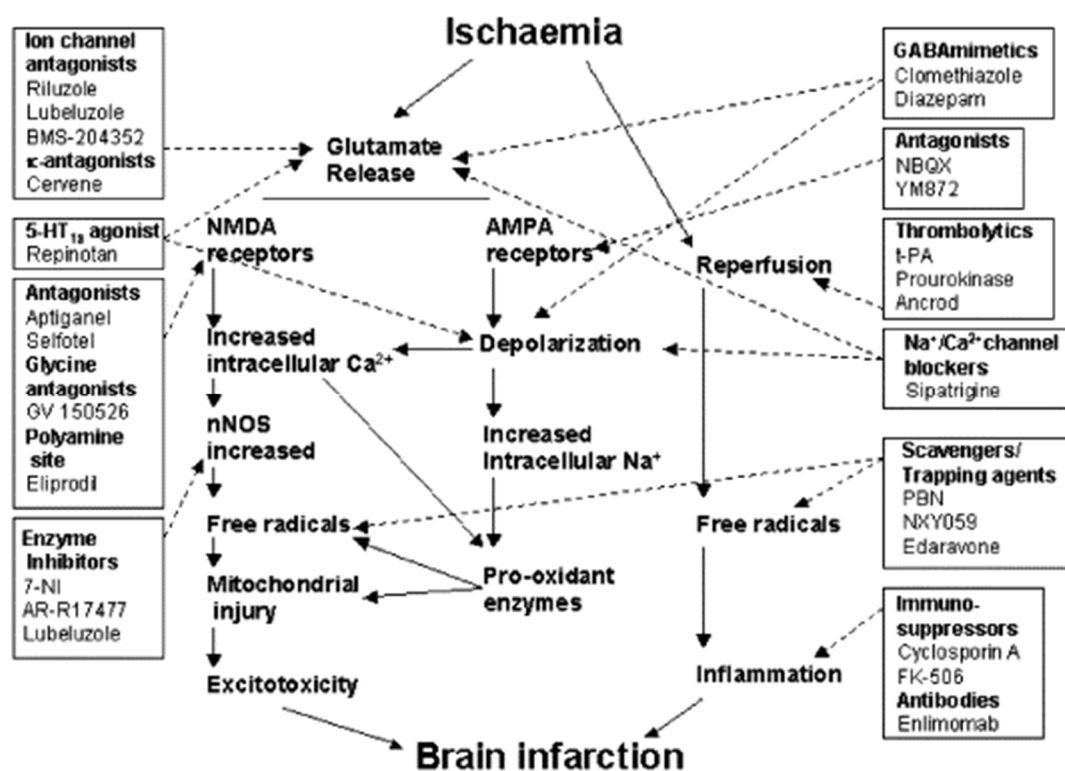


Figure S2. Summary of the mechanisms of action of the drugs used to treat Parkinson's Disease.



**Figure S3.** Mechanism of action of clinically approved antiepileptic drugs (AAED). [Obtained and reproduced without any changes from [2] and Springer in accordance with **Creative Commons Attribution License (CC BY)**]. The asterisks are used to indicate that the compounds elicit their action through multiple mechanisms of which not all are depicted.

**Key:** AMPA- $\alpha$ -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid, GABA- $\gamma$ -aminobutyric acid, GABA-T- GABA aminotransferase, GAT-1 GABA transporter 1, KCNQ Kv7 potassium channel family, NMDA N-methyl-D-aspartate, SV2A synaptic vesicle protein 2A



**Figure S4.** Schematic representation of the ischemic cascade showing compounds developed to interfere with mechanisms and provide neuroprotection. Reproduced from [3] with permission from Elsevier.