CORRECTION

Correction: Targeting NLRP3 inflammasome for neurodegenerative disorders

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In this article, an incorrect table was inadvertently displayed as Table 2. Correct Table 2 is shown below:

Table 2. Targeting synaptic function for Alzheimer's drug development*

Molecular target	Drugs	Clinical trials	References
Fyn kinase	Saracatinib	Phase 2	[197–199]; NCT02167256
Tyrosine kinase	Masitinib	Phase 3	[199, 200]; NCT01872598
Rho kinase	Fasudil	Preclinical	[201, 202]
Rho kinase	FSD-C10	Preclinical	[203, 204]
NMDA receptor	Memantine	Approved	[205, 206]
Glutamatergic pathway	NitroSynapsin	Preclinical	[207, 208]
Glutamate generation	Riluzole	Phase 2	[98, 209]; NCT01703117
Cholinesterase	Donepezil	Approved	[210–212]
Cholinesterase	Galantamine	Approved	[213–215]
Cholinesterase	Rivastigmine	Approved	[213, 216, 217]
Serotonin reuptake	Sertraline	Phase 4	[126, 218–220]; NCT00009191
Neurotrophic factors	BDNF	Phase 1	[221–224]; NCT05040217
Neurotrophin receptor	LM11A-31	Phase 2	[225]; NCT03069014

*Adapted from Table 1 of Peng L, Bestard-Lorigados I, Song W. Mol Psychiatry. 2022;27:2940–2949.

In this article, the captions for Figs. 3 and 4 were inadvertently swapped. The correct captions for these figures are shown below:

Figure 3. The effect of Aβ on synaptic transmission. APP can be cleaved by β-secretase at the Asp1 site, leading to the generation of membrane-bound C99, which is subsequently cleaved by γ-secretase to produce intracellular C-terminal fragments and Aβ. The increased presence of Aβ monomers promotes the formation of Aβ oligomers, which further induce tau phosphorylation, leading to the subsequent formation of neurofibrillary tangles and impairing LTP by disrupting the balance between glutamatergic and GABAergic activity. Aβ amyloid β protein, APP amyloid precursor protein, C99 C-terminal fragment of 99 amino acids, GABAergic gamma-aminobutyric acid (GABA)-ergic, LTP long-term potentiation.

Figure 4. NLRP3-mediated glia cells contribute to neuronal dysfunction. Aβ aggregation induces the activation and accumulation of NLRP3 inflammasome in microglia, which results in the secretion of IL-1β. The increased level of IL-1β further accelerates the senescence of astrocytes and reduces the phagocytic capacity of astrocytes, leading to the accumulation of synaptic debris and neuronal dysfunction. ApoE4 exacerbates this process by promoting the accumulation of C1q, resulting in hyperactive microglia and an intensified loss of synapses. Aβ amyloid β protein, ApoE4 apolipoprotein-E4, C1q complement 1q, IL-1β interleukin-1β, NLRP3 the NACHT, LRR and PYD domains-containing protein 3.

The original article has been corrected.

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