Erratum to: Animal Models of Alzheimer's Disease: Utilization of Transgenic Alzheimer's Disease Models in Studies of Amyloid Beta Clearance

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The reference list and tables were erroneously printed, and are provided thusly in remedy.

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Table 1 Microglial cytokine expression profiles in some Alzheimer's disease mouse models

Study	AD mouse model	Development of A β pathology	Detection method	Cytokine expression profile
Ruan et al. [82]	APPswe/PS1dE9	4 months	IHC	TNF- α $\uparrow$ starting at age 8 mo and IL-1 β , IL-6, and MCP-1 $\uparrow$ starting at age 10 mo
Hickman et al. [17]	APPswe/PS1dE9	4 months	Isolated microglia; qPCR	SRA, CD36, RAGE, insulysin, neprilysin, and MMP-9 $\downarrow$; IL-1 β and TNF- α $\uparrow$ starting at age 8 mo
Abbas et al. [83]	Tg2576	9–11 months	IHC; in situ hybridization	IFN- γ and IL-12 $\uparrow$ and IL-4 $\downarrow$ starting at age 9 mo
Patel et al. [18]	APPswe; APP/PS1	11 months in APPswe; 10 weeks in APP/PS1	Organotypic slice cultures; multiplex cytokine assay	IL-1 α , TNF- α , GM-CSF, and IL-6 $\uparrow$ at age 15 mo in both models. APP/PS1 brain slices produced significantly more IL-12p40, IL-1 β , IL-1 α , TNF- α , GM-CSF, and IL-6 compared to APPswe brain slices. Cytokine levels correlated with brain A β in both models
Jimenez et al. [19]	PS1(M146L) x APP751sl	3–4 months	Brain homogenates; qPCR	TNF α , iNOS, IL-1 β , FASL, TRAIL, nox1, and Cox2 $\uparrow$ at age 18 mo. IL-1 β $\uparrow$ at age 12 mo
Jimenez et al. [19]	PS1(M146L) x APP751sl	3–4 months	IHC	Microglia in the near vicinity of A β plaques were shown to adopt an incomplete alternative activation state characterized by elevation of YM-1 and absence of TNF- α and iNOS at age 18 mo

A β beta amyloid; AD Alzheimer's disease; FASL Fas ligand; GM-CSF granulocyte-macrophage colony-stimulating factor; IFN- γ interferon-gamma; IHC immunohistochemistry; IL interleukin; iNOS inducible nitric oxide synthase; MCP monocyte chemotactic protein-1; MMP-9 matrix metalloproteinase 9; qPCR quantitative polymerase chain reaction; RAGE receptor for advanced glycation endproducts; SRA scavenger receptor A; TNF- α tumor necrosis factor alpha; TRAIL TNF-related apoptosis-inducing ligand

Table 2 Role of specific myeloid cells or factors regulating myeloid cell migration or activity affiliated to A β clearance in the animal models of AD

Study	Cells/Factors	Model	Effect on A β burden
Mildner et al. [2••]	BM CCR2 ⁺ cells	CCR2 ^{-/-} BM cell chimera in APPswe/PS1 and Tg2576 mice	Irradiation preconditioning and CCR2 expression in BM cells are required for their brain engraftment. Peripheral macrophages rather than parenchymal microglia modulate A β deposition in AD mice
El Khoury et al. [5]	CCR2	CCR2 ^{-/-} mice crossed with Tg2576 mice	Absence of CCR2 impairs microglia accumulation and increases A β levels and mortality in AD mice
Lebson et al. [30•]	BM CD11b ⁺ cells	Adoptive transfer of neprilysin-transfected BM CD11b ⁺ cells into APP/PS1 mice	BM CD11b ⁺ cells home to AD mouse brain after adoptive transfer. Injection of CD11b ⁺ cells expressing secreted form of neprilysin is associated with reduced A β burden
Grathwohl et al. [58]	Microglia/CD11b ⁺ cells	CD11b ⁺ cell ablation in APPPS1 and APP23 mice	Nearly complete ablation of CD11b ⁺ cells did not alter A β levels in AD mice within the observation period of 4 weeks
Town et al. [84]	CD11c ⁺ TGF- β ⁺ cells	Dominant negative TGF- β in CD11c ⁺ cells in Tg2576 and APPswe/PS1dE9 mice	TGF- β deficiency in CD11c ⁺ reduced parenchymal and vascular A β burden involving infiltration of peripheral macrophages
Zhu et al. [85]	CD45	CD45 ^{-/-} crossed with APPswe/PS1dE9 mice	CD45 deficiency promotes proinflammatory microglial activation, reduces their phagocytic activity, and increases soluble and insoluble A β levels
Hao et al. [86]	Myeloid differentiation factor 88	MyD88 ^{-/-} BM cell chimera in TgCRND8 and APPswe/PS1dE9 mice	MyD88 deletion in BM cells attenuates neuroinflammation, enhances A β phagocytosis, and reduces A β burden
Lee et al. [87] and Liu et al. [88]	CX3CR1	CX3CR1 ^{-/-} crossed with APPPS1 mice	Absence of CX3CR1 leads to altered inflammation, enhancement of microglia A β phagocytosis, and reduction of A β burden
Reed-Geaghan et al. [89]	CD14	CD14 ^{-/-} crossed with APPswe/PS1dE9 mice	TLR co-receptor CD14 deficiency alters microglia activation and reduces microgliosis and A β burden
Song et al. [90]	TLR4	TLR4 mutation mice crossed with APPswe/PS1dE9 mice	TLR mutation decreases microglia activation and increases A β deposition in early AD possibly involving reduced A β clearance
Doi et al. [91]	TLR9 ligand	TLR9 ligand CpG injection (i.c.v.) into Tg2576 mice	TLR9 ligand CpG reduces A β burden and increases microglial production of degrading enzymes and reduction of A β
Keene et al. [92]	EP2	EP2 ^{-/-} BM cell chimera in APPswe/PS1dE9 mice	EP2 deletion in BM cells reduces A β burden
Terwel et al. [93] and Fitz et al. [94]	Liver X receptor	LXR agonist p.o. into APP23 mice, LXR ^{-/-} crossed with APPswe/PS1dE9 mice	LXR agonist reduces A β burden in AD mice on high-fat diet. Astrocytic LXR activation and the release of ApoE are involved in microglial A β phagocytosis
Maier et al. [95]	Complement factor C3	C3 ^{-/-} crossed with APP mice	Absence of C3 drives microglia into M2 alternative activation phenotype and increases A β burden
Chakrabarty et al. [96]	IL-6	AAV1-induced IL-6 overexpression in brain of TgCRND8 and Tg2576 mice	Overexpression of IL-6 leads to massive gliosis, and attenuates A β deposition by enhanced microglia activation and possibly by increased A β phagocytosis
ShafteI et al. [97]	IL-1 β	Overexpression of IL-1 β in brain of APPswe/PS1dE9 mice	Sustained overexpression of IL-1 β enhances microglia activation and reduces A β burden
Boissonneault et al. [98•]	MCSF	Weekly i.p. injections of MCSF into APPswe/PS1	MCSF increases microgliosis and BM cell infiltration and reduces A β burden by phagocytosis

A β beta amyloid; AAV adeno-associated virus; AD Alzheimer's disease; apoE apolipoprotein E; BM bone marrow; EP2 prostaglandin E2 receptor subtype 2; i.c.v. intracerebroventricular; IL interleukin; i.p. intraperitoneal; LXR liver x receptor; MCSF macrocyte colony-stimulating factor; p.o. per oral; TGF- β ⁺ transforming growth factor beta; TLR9 toll-like receptor 9

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- Of major importance

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