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Erratum

Tau Immunotherapies for Alzheimer's Disease and Related Tauopathies: Progress and Potential Pitfalls

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On page S560 and 561 only four of the six passive trials consisting of antibodies were printed. The points 5 and 6 were excluded from the published article. The full listing, with point 5 and 6 in bold, is given below:

The six passive trials consist of antibodies targeting:
1) tau8–19 in healthy subjects and PSP patients, that was developed by iPerian and subsequently by Bristol-Meyers Squibb and has now been licensed to Biogen [38, 85, 86]. Currently named BIIB092 (previously BMS-986168 and IPN007), it is in Phase I-II for PSP;
2) tau25–30 in AD (Phase II, [87]) and PSP (Phase II; [88]). It was developed by C2N Diagnostics, LLC (C2N 8E12; [32, 89]) and has been licensed to AbbVie (ABBV-8E12);
3) an unidentified epitope that may be phosphoserine 409 (RO7105705) in healthy subjects and AD patients [72, 90];
4) an unidentified epitope (LY3303560) in subjects that are healthy, or with mild cognitive impairments or AD (Phase I, [91, 92]) that is likely a humanized form of the conformational antibody MC1 [73, 93], which as mentioned above has been effective in different mouse studies [20, 31];
5) The middle region of tau in healthy subjects and AD patients (JNJ-63733657) [94, 95], and;
6) Tau235-246 in healthy subjects (UCB0107) [95, 96].

On Page S565, in the Reference section, second column, there were three missing references. The correct listing of references is (with the previous three missing references in bold):

[94] ClinicalTrials.gov (2018) A Study to Investigate Safety and Tolerability, Pharmacokinetics and Pharmacodynamics of JNJ-63733657 in Healthy Subjects and Subjects with Alzheimers' Disease. https://clinicaltrials.gov/ct2/show/NCT03375697. US National Library of Medicine.
[95] Rogers MB (2018) To block tau's proteopathic spread, antibody must attack its mid region. Alzforum https://www.alzforum.org/news/conference-coverage/block-taus-proteopathic-spread-antibody-must-attack-its-mid-region.
[96] ClinicalTrials.gov (2018) A Study to Test the Safety, Pharmacokinetics, and Pharmacodynamics of Single Ascending Intravenous Doses of UCB0107 in Healthy Male Subjects. https://clinicaltrials.gov/ct2/show/NCT03464227.

[97] Saint-Aubert L, Lemoine L, Chiotis K, Leuzy A, Rodriguez-Vieitez E, Nordberg A (2017) Tau PET imaging: Present and future directions. *Mol Neurodegener* **12**, 19.
[98] Hall B, Mak E, Cervenka S, Aigbirhio FI, Rowe JB, O'Brien JT (2017) *In vivo* tau PET imaging in dementia: Pathophysiology, radiotracer quantification, and a systematic review of clinical findings. *Ageing Res Rev* **36**, 50-63.

On Page S561, right column, lines 7 and 10, references 94 and 95 should then be changed to references 97 and 98. The correct sentences are:

Advances in tau brain imaging have now resulted in several promising β-sheet dye compounds that appear to be selective for tau aggregates, although non-specific binding has now been reported for some of them and their use discontinued **[97, 98]**. Also, these probes are not good at detecting non-AD tauopathies, suggesting some structural differences in the tau lesions **[97, 98]**.

Finally, one ongoing passive tau antibody trial was missed. BIIB076, against an unidentified epitope, originally developed by Neurimmune (NI-105, 6C5), was acquired by Biogen and is currently in Phase I trial in healthy subjects and AD patients (NCT03056729). It has shown target engagement without apparent toxicity in cynomolgus monkeys (Czerkowicz J et al., Alzheimer's & Dementia, July 2017, Vol. 13, Issue 7, Supplement, page P1271).