Correspondence

Amyloid- β , the Retina, and Mouse Models of Alzheimer Disease

To the Editor-in-Chief:

In our recent publication, "Vaccinations Reduce β -Amyloid Plaques but Exacerbate Vascular Deposition and Inflammation in the Retina of Alzheimer's Transgenic Mice,"¹ we cited work by Ning et al² describing retinal degeneration in a mouse model of Alzheimer disease. It has been brought to our attention that some of our statements regarding the work by Ning et al are incorrect.

On page 2107 of the Discussion, we stated, "Interestingly, although intraneuronal accumulation of $A\beta$ was reported in association with ganglion cell degeneration in Tg2576 mice, no $A\beta$ -associated plaques were found in their retinas in the previous studies (Ning et al²)." This is not correct, as the publication by Ning et al demonstrates immunoreactivity for $A\beta$ as an extracellular deposit in the retinas of the transgenic mice. Importantly, the two transgenic mouse strains examined by Ning et al were APP/PS1-bitransgenic, not of Tg2576. Further, our statement about intraneuronal accumulation of $A\beta$ is also incorrect as the authors demonstrated cytoplasmic staining of APP (amyloid precursor protein), not $A\beta$.

We apologize to Ning et al² for these misstatements, as well as to the readers of either article for any confusion we may have caused.

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References

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2. Ning A, Cui J, To E, Ashe KH, Matsubara J: Amyloid- β deposits lead to retinal degeneration in a mouse model of Alzheimer disease. *IOVS* 2008, 49:5136–5143