

Brain and Eye Coordination in Alzheimer’s Disease

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Abstract

The eye is the anatomical extension of brain where multiple parallels can be drawn between the neurons, vasculature and immune response. Further both organs modify similar with diseases. As such stands to reason that multidisciplinary research that investigates neurodegenerative diseases

Keywords: Neurodegenerative Diseases, Alzheimer’s Disease, Vascular Diseases, Parkinson’s Disease, Dementia, Optic Nerve, Neuroimaging, Subcortical Infraction, Optical Coherence Tomography, Macula

Alzheimer’s Disease

Most common form of dementia. Alzheimer’s disease is associated with hall mark, amyloid beta in the retina [1].

Immunohistochemical cortical 6E10 immunostaining. (A) Tg mice (blue) had significantly higher percentage of cortical area immunostained relative to their WT controls (red) (B) There was a trend towards higher cortical immunostaining in Tg mice (blue) that begins at nine months of age [2]. There was a moderately strong correlation and increase of immunostaining over time for Tg mice [3]. (C) Representative cross-section of an 18 month Tg mouse brain immunoreacted for Aβ. Note the numerous Aβ plaques shown by the red chromogenic reaction product in cortex and hippocampal areas, compared to (D), the age-matched WT brain immunoreacted for Aβ. Scale bar for (C,D) is shown in D = 400 microns. (E) Retinal cross-section of an 18 month Tg mouse eye demonstrating Aβ immunoreactivity (arrows) in the inner retina in the ganglion cell layer (GCL) and the inner plexiform layer (IPL) compared to (F) the age-matched WT retina, which demonstrates a pale pink baseline level of immunoreactivity.

As shown in the diagnosis in the following Table: 1

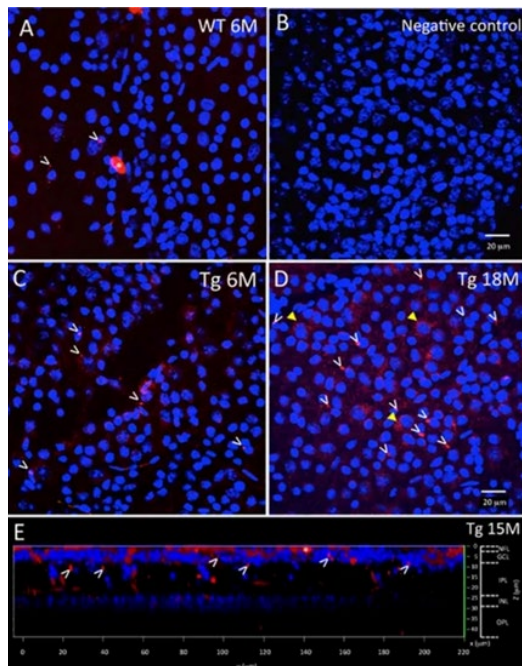
Case no.	Age	Sex	Primary Path Dx	Additional Path Dx	A-beta (Thal, Biel) (1–5)	Braak stage (1–6)	Neuritic Plaque (CERAD, Biel)	Diffuse Plaque (CERAD, Biel)
1	75	M	N/A	–	–	–	–	–
2	70	M	N/A	–	–	–	–	–
3	75	M	N/A	–	–	–	–	–
4	73	M	N/A	–	–	–	–	–
5	75	M	N/A	–	–	–	–	–
6	66	M	N/A	–	–	–	–	–
7	69	M	N/A	–	–	–	–	–
8	69	M	N/A	–	–	–	–	–
9	55	F	N/A	–	–	–	–	–
10	63	F	N/A	–	–	–	–	–
11	78	F	AD	–	5	6	Frequent	Frequent
12	88	F	AD	FTLD-TDP, CAA	5	6	Frequent	Frequent
13	59	F	AD	DLB	5	6	Frequent	Frequent
14	70	M	AD	CAA	5	6	Moderate	Frequent
15	80	M	DLB	mod. AD	3	4	Moderate	Frequent
16	94	F	AD	CAA	5	6	Frequent	Frequent
17	75	M	AD	DLB, CAA	5	6	Frequent	Sparse
18	82	F	AD	DLB	5	6	Frequent	Frequent
19	82	M	DLB	mod. AD	5	5	Moderate	Frequent
20	79	M	AD	–	5	6	Frequent	Frequent
21	87	F	CVD	mod. AD	5	4	Moderate	Frequent
22	82	F	AD	CAA	5	6	Frequent	Frequent
23	81	F	AD	mild DLB	5	6	Frequent	Frequent
24	89	F	CVD	mod. AD, FTLD-TDP	5	5	Moderate	Frequent
25	82	F	AD	HS, CAA	5	6	Frequent	Frequent

AD, Alzheimer’s disease; DLB, dementia with Lewy bodies; CVD, cerebrovascular dementia; FTLD-TDP, frontotemporal lobar degeneration with TDP-43 inclusions; CAA, cerebral amyloid angiopathy; HS, Huntington’s disease; N/A, not applicable (Control eyes).

Scale bar in D = 400 microns. Scale bar for (E,F) is shown in F = 25 microns [4].

Also shown in the retinal fluorescence

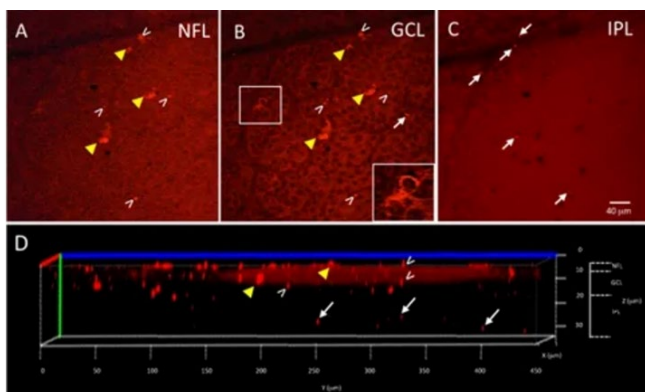
Figure 1: Ex vivo WT and Tg Mouse Wholemount and 6E10 Immunofluorescence



Also shows (A) Representative confocal image after A β immunofluorescence of a younger WT retina. Note the small specks of A β deposits in red immunofluorescence, some shown by white arrowheads. A larger opaque red profile is an artifact (B) Representative image of a negative control section, in which the primary antibody was replaced with a non-specific IgG. Image demonstrates very low background levels of red immunofluorescence. (C) Representative image of A β immunoreactivity in the 6 month Tg retina with representative red specks of immunofluorescence shown by white arrowheads. (D) In an older Tg retina, the retinal immunofluorescence increased with more red immunoreactive specks and an occasional immunoreactive retinal ganglion cell [5]. (A–D) Scale bar: 20 μ m. (E) An orthogonal view of the confocal z-stack reconstruction from a 15 month Tg retina demonstrates that the majority of the A β immunoreactivity is present in the inner retina, specifically in the NFL and GCL. Some A β immunoreactive specks can be seen extending deeper into the IPL. Cell nuclear labeling with DAPI reveals lamination in the retina. Lamination and scale bars are shown on the right axes [6].

Also Immunofluorescences in Figure 2

Representation of (A) Representative high power confocal image of the NFL

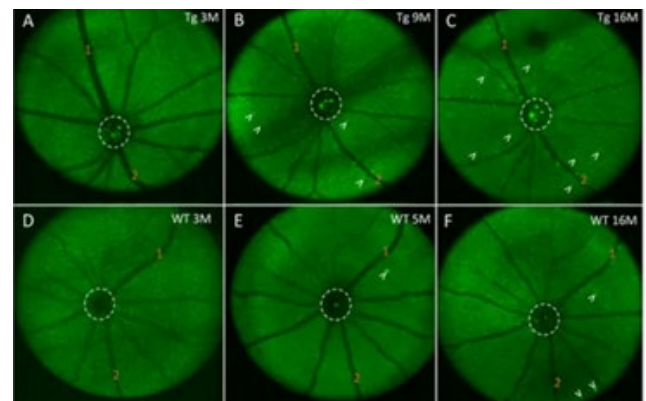


demonstrating red immunofluorescence. Note that A β specks can be seen in the NFL, while the fluorescence associated with the RGC cell bodies in the GCL is also seen [7]. (B) As the confocal imaging descends into the wholemout, the GCL is identified in an optical slice by the presence of nuclei, here shown as black

circular profiles as the DAPI channel is not shown. Note the fluorescence of the RGCs is evident in the cytoplasm and align with the RGCs seen in A. The extracellular A β specks can be seen. (C) An optical slice through the IPL reveals fewer nuclei, as evidence by the lack of black circular nuclear profiles as seen in B. A few A β specks are seen, but generally less A β immunoreactivity is observed in IPL. Scale bar for (A–C) is shown in. (D) The orthogonal view of the confocal z-stack reconstruction from the Tg retina shown in (A–C). Note red immunoreactivity in the NFL, GCL and IPL. The immunoreactivity present in the IPL indicates that the antibody penetrated deep into the wholemout tissue. Additional optical slices from this wholemout [8].

Also Fluorescence of Mouse Retina also shown in Figure 4

In vivo fluorescence of the mouse retina after curcumin tail vein injections. (A–C) In vivo longitudinal fluorescence images of an individual Tg mouse at 3, 9, and 16 months of age. (D–F) In vivo longitudinal fluorescence images of an individual WT mouse at 3, 5, and 16 months of age [9]. The pattern of A β in vivo fluorescence is seen as bright green specks. Note that the A β in vivo fluorescent deposits increased as the animal aged. Representative landmark



blood vessels are labelled with orange numbers and can be identified in each of the images in the Tg (A–C) and in the WT (D–F). Dashed white circle indicates area of the optic nerve head. Representative examples of the in vivo longitudinal fluorescence images of an individual WT mouse at 3, 5, and 16 months of age reveals that A β fluorescent deposits only marginally increased as the animal aged (D–F). Note a significantly higher load of A β fluorescent deposits in the 16 month Tg (C) compared to the 16 month WT (F) mice [10].

Results

Cortical Abeta Immunoreactivity is greater in Tg than WT
Retinal Abeta Immunoreactivity is greater in Tg than WT

Treatment

Stem cell therapy or monoclonal genetic antibodies with required gene can help with condition also alzheimers treatment through gene therapy or stem cell therapy can lead to its treatment.

Discussion

- Brain eye coordination
- Alzheimer's disease
- Effect of eye during Alzheimer's disease

Conclusion

Brain eye coordination and effect of eye during Alzheimer's disease.

Conflict of Interest: Author declares there is no conflict of interest

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