

Questions about Wu et al., 2010:

- What is the Tg2576 mouse line, and why was it used in this paper?
- Tg2576 mice don't begin to show AD-related phenotypes until adulthood. In this study, the neuronal cultures used were from embryonic mice. Do you find it surprising that embryonic neurons from Tg2576 mice show such dramatic phenotypes in culture, while young Tg2576 mice appear healthy? Can you come up with a potential explanation for why this might be?
- Did the authors mention blinding to genotype or treatment condition, either in the imaging or analysis phase of the project? Do you think blinding is necessary for experiments like this?
- In experiments where the authors applied conditioned media to the cultures, how did the authors confirm that the observed effects were due to Abeta itself, and not something else in the media (e.g. in Fig. 3D)?
- If you had funding to develop a therapy for people with AD, and you could only use this paper as a guide, what would you do with your funding?

Questions about Lesne et al., 2006:

- Why do you think A β oligomerization has no significant effect on memory impairment early in the disease?
- The authors claim that "the variability in levels of A β assemblies between animals of the same age provided an opportunity to examine correlations between the different A β oligomers and memory impairment" What are these correlations? What was the conclusion? Do you agree with their interpretations?
- The authors claim the A β *56 is responsible for memory retention deficits? What is their evidence? Is it sufficient to make such a strong claim?

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7.342 Synapse Remodeling in Health and Disease
Fall 2022

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