



# Is Tauism Dead?



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## Opinion

After the latest failure of drug trials in Alzheimer’s disease [1], we have to conclude that we don’t understand the disease [2]. In an editorial in the New England Journal of Medicine, Murphy [3] wrote “the field is clearly in need of innovative ideas and we are very well be nearing the end of the amyloid hypothesis rope”. Antidiabetic drugs will not be the new cure for Alzheimer’s as this research also relies heavily on the amyloid-beta-tau hypothesis [4]. So, is this the end of “Tauism” after 15 years of failing drug trials? Frankly, the answer is yes. Research in neurodegenerative diseases will move more to genetics, epigenetics, immunology and inflammation, as is illustrated by two very recent publications.

Scientists are a step closer now to understanding which genes are responsible for early onset Alzheimer’s in Down syndrome. By the time they reach their 60’s two third of people with Down syndrome will have early onset dementia. The high rates of Alzheimer’s in Down syndrome thought to be caused by a particular gene on chromosome 21, called APP. This chromosome contains 231 genes, but APP was the prime suspect, because it produces amyloid precursor proteins. Now, researchers of the Francis Crick Institute and UCL found that extra copies of other genes on chromosome 21 increase Alzheimer’s like brain pathology and cognitive impairments in a mouse model of Down syndrome. This is the first time genes other than the APP gene are implicated in the development of Alzheimer’s in people with Down syndrome. Although the pathways opened by these new genes are not known

yet, they offer new research possibilities in preventing Alzheimer’s in Down syndrome [5,6].

Another example of shifting research is a recent finding of University of Virginia brain researchers. They found that immune cells commonly blamed in Alzheimer’s and other neurodegenerative diseases are actually precision cleaning machines. This sheds a new light on these microglial cells. If these microglial cells are too aggressive in removing debris or perhaps something could go wrong and contribute to disease is also not known yet. Thus, while “tauism” has ended, the new clear innovative ideas that Murphy [3] asked for, are coming up already.

## References

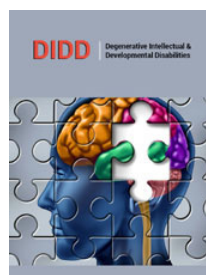
1. Crow D (2018) Big pharma efforts on Alzheimer’s tested by pfizer exit. Financial Times, San Francisco, USA.
2. Shurkin J (2018) Alzheimer’s drug trials keep failing:it may be because we don’t understand the disease. Inside Science, USA.
3. Murphy MP (2018) amyloid beta solubility in the treatment of alzheimer’s disease. N Engl J Med 378(4): 391-392.
4. Naafs MA (2018) No cure for Alzheimer’s with antidiabetic drugs. Clin J Diab Care and Control, In press.
5. Wiseman FK, Pulford LJ, Barkus C, Liao F, Portelius E, et al. (2018) Trisomy of human chromosome 21 enhances amyloid-beta deposition independently of an extra copy of APP. Brain DOI: 10.1093/brain/awy159.
6. Norris GT, Smirnov I, Filiano AJ, Shadowen HM, Cody KR, et al. (2018) Neuronal integrity and complement control synaptic material clearance by microglia after CNS injury. J Exp Med 215(7): 1789-1801.



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