

## Is Alzheimer's disease a medical notion of dementia worth keeping in Neuroscience

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The eponym of Alzheimer's disease has been around for more than one century from 1910. Its diagnosis varies: from senile dementia to senium praecox (for both pre-senile and senile dementia) to Mild Cognitive Impairment (MCI) leading to AD as the most feared form of dementia. Such uncertainties create serious problems which most neuroscientists have refused to admit. They prefer to perpetuate the eponym even to 2006 in *Science* to keep it alive for contrast with other forms of dementia; debate on its cause, by regarding AD as the effect to seek a one-to-one cause-effect relationship, Amyloid Beta Hypothesis, for instance, which has dominated the field for more than 20 years, albeit to no avail; advocate the intervention or prevention, unaware of AD as a fiction, resulting from its confusion with dementia, to the extent of using animal models to mimick the existence of AD; take the liberty on the basis of DSM-IV to assign lesion site unaware that Auguste's brain at autopsy had widespread atrophy, and that pre-mortem she had four vascular disorders, one of which, decubitus angina, was the direct cause of her death. I take exception to such predilections. Dementia can be tested and diagnosed as the effects of brain atrophy; it manifests in a cluster of behavioral alterations; the causes can be vascular or non-vascular in origin. Thus, dementia is neither a disease nor equivalent to AD. Plaques and tangles should be called Fischer's disease (FD) which gradually causes brain atrophy to result progressively from simple dementia to presbyophrenia in a dichotomy as suggested by Fischer. For this reason, MCI is a cheap reinvention of Fischer's dichotomy. Once AD is replaced by FD, neuroscientists and clinicians must be made to understand that: (1) As a medical notion of dementia, it is not worth keeping; (2) dementia can start from any brain location and will spread, because its cause is wear and tear as an on-going process of aging; (3) the cause is either vascular or non-vascular in origin, but the effect can vary, resulting in a cluster of behavioral alterations, language disorder included, from onset of simple dementia to presbyophrenia before the patient succumbs; and (4) there is no one-to-one cause-effect relation, as the brain atrophy spreads from cortical to cortical, cortical to subcortical, and/or subcortical to cortical regions.

### Biography

Fred C. C. Peng is a Behavioral Neuroscientist in the Department of Neurosurgery and the Neurological Institute at Taipei, VGH, Taiwan. He has a wide range of interests in behavioral alterations caused by differing brain lesions, such as epilepsy, FD, stroke, PD, SCA (spino-cerebellar ataxia), Pick's Disease, aphasia, PSP (progressive supranuclear palsy), and many others.

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