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1: [J Alzheimers Dis.](#) 2008 May; 13(4): 381-91.



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Chronic inflammation and amyloidogenesis in Alzheimer's disease -- role of Spirochetes.

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Alzheimer's disease (AD) is associated with dementia, brain atrophy and the aggregation and accumulation of a cortical amyloid-beta peptide (A β). Chronic bacterial infections are frequently associated with amyloid deposition. It had been known from a century that the spirochete *Treponema pallidum* can cause dementia in the atrophic form of general paresis. It is

noteworthy that the pathological hallmarks of this atrophic form are similar to those of AD. Recent observations showed that bacteria, including spirochetes contain amyloidogenic proteins and also that Abeta deposition and tau phosphorylation can be induced in or in vivo following exposure to bacteria or LPS. Bacteria or their poorly degradable debris are powerful inflammatory cytokine inducers, activate complement, affect vascular permeability, generate nitric oxide and free radicals, induce apoptosis and are amyloidogenic. All these processes are involved in the pathogenesis of AD. Old and new observations, reviewed here, indicate that to consider the possibility that bacteria, including several types of spirochetes highly prevalent in the population at large or their persisting debris may initiate cascade of events leading to chronic inflammation and amyloid deposition in AD is important, as appropriate antibacterial and antiinflammatory therapy would be available to prevent dementia.
PMID: 18487847 [PubMed - indexed for MEDLINE]

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