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Letter to the Editor

Microbes and Alzheimer' disease: lessons from *H. pylori* and GUT microbiota

Dear Editor,

In their review Franceschi et al[1] summarized substantial studies linking Alzheimer's disease (AD) with *Helicobacter pylori* (*Hp*) and gut microbiota.

Nevertheless, they do exist substantial additional data, which provide significant evidence regarding *Hp* and worth to be reported. The North Manhattan Study (NOMAS) performed by Katan et al[2] included 1,625 participants; cognition was assessed with mini-mental state examination (MMSE) and modified telephone interview for cognitive status. Infectious burden (IB), among others including *Hp*, was evaluated serologically (ELISA). The authors concluded that that IB was associated with cognitive performance. Likewise, Wright et al[3] demonstrated later by using 588 stroke-free NOMAS patients, who underwent a battery of neuropsychological tests, that their IB including *Hp* was associated with worse cognitive performance. Moreover, Xu et al[4] investigated the impact of *Hp* on vascular dementia in a sample of 173 patients. They observed that *Hp* positive patients, identified by using urea breath test, were characterized by higher pro-inflammatory cytokine levels (tumor necrosis factor (TNF)-α, interleukin (IL)-1β and IL-12) and worse scores of Montreal and MMSE. Some proposed mechanisms for *Hp*-related AD, mild cognitive impairment, and/or glaucoma (defined as ocular AD) pathophysiology include: influences on neuronal apoptotic damage via molecular mimicry, in which homologous *Hp* epitopes induce irregular humoral and cellular immune responses that cross-react with components of nerves[5-8]; molecular mimicry between *Hp* and endothelial antigens[7,8]; mononuclear cell production of a tissue factor-like pro-coagulant that converts fibrinogen into fibrin[5-8]; induction of reactive oxygen species and circulating lipid peroxidases[5,8]; activation and aggregation of platelets[5-8]; augmented homocysteine serum levels with subsequent endothelial damage and neurodegeneration[6]; upregutated toll-like receptors and the aforementioned pro-iflammatroy cytokines that induce cell signaling cascades implicated in AD pathogenesis[9]; and/or inappropriate human defensins' involvement in AD neurodegeneration[10]. Therefore, *Hp* eradication might display a positive impact on AD and thus further large-scale studies are warranted.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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