Alzheimer's disease pathology programmed by gut-derived disparity: A comprehensive understanding of neurodegeneration

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Abstract

The human gut is colonized with microbial species that not only resides but also facilitate in many functions. The alterations in this gastrointestinal microbiota directly influence many body systems including, central nervous system (CNS) disorders such as Alzheimer's disease (AD). The term microbiota is thus a determinant factor in the association between illness and health. AD, the most common form of dementia, is a neurodegenerative disorder associated with impaired cognition and cerebral accumulation of amyloid-β peptides (Aβ). Germ-free animals have provided enormous data on the existence of dysbiosis and its conversion by fecal microbiota transplantation. The main cause of AD is unknown and it is estimated that by 2050 the number of patients will increase up to three times. Bacteria populating the gut microbiota (GM) can secrete large amounts of amyloids and lipopolysaccharides, which might contribute to the modulation of signaling pathways and the production of proinflammatory cytokines associated with the pathogenesis of AD. The Gut-brain axis links the emotional and cognitive center of the brain with intestinal activities. Thus, it can be said that the dysbiosis of human microbiome could be a risk factor for AD. In this review, we provide an overview of GM and how their dysregulation accounts for the pathogenesis of AD. Illustration of the mechanisms underlying the modification of GM composition may pave the way for developing novel preventive and therapeutic approach for AD.

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