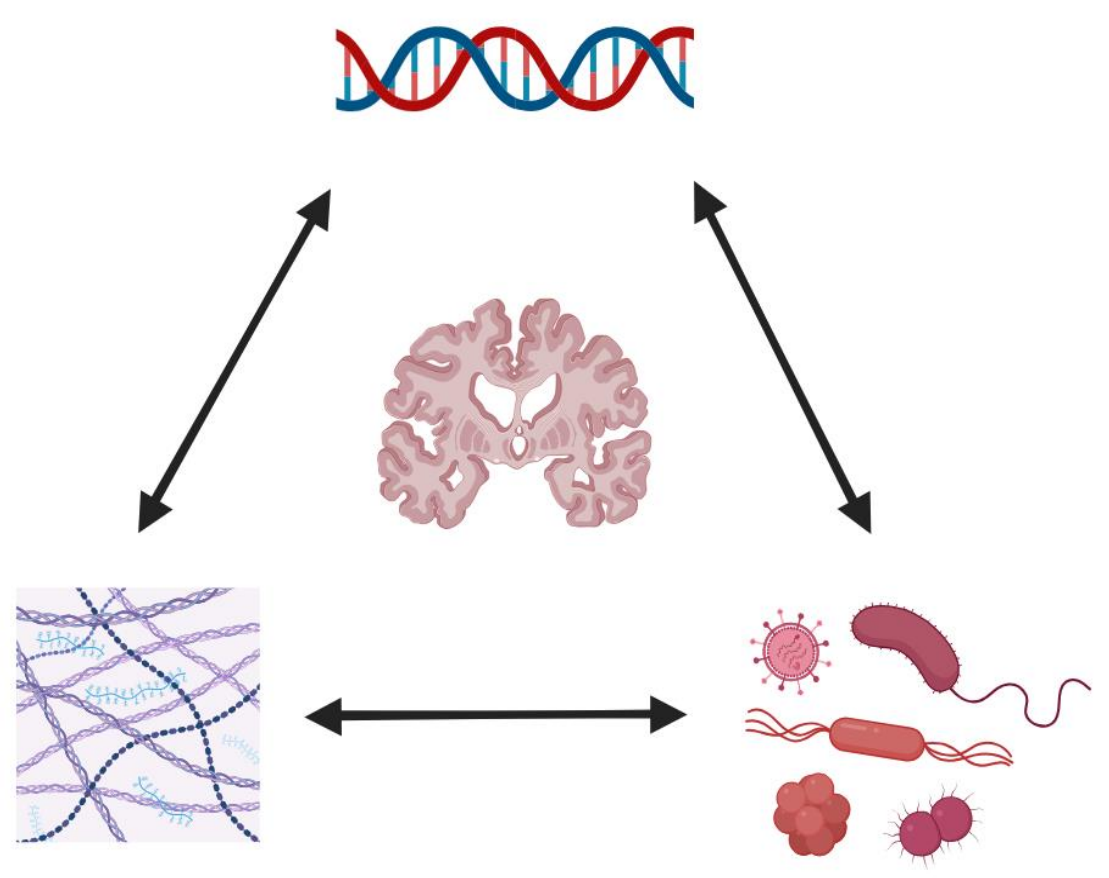


Analysis of CagA and LTBP2 protein concentration and *APOE* gene variants in patients with Alzheimer's disease

Joanna Poszwa¹, Wojciech Owecki^{1,2}, Julia Brząkalska¹, Oliwia Szymanowicz¹, Bartosz Słowikowski³, Paweł P. Jagodziński³, Wojciech Kozubski⁴, Jolanta Dorszewska¹
¹Poznan University of Medical Sciences, Laboratory of Neurobiology, Department of Neurology, Poznan, Poland, ²Student Scientific Society of Poznan University of Medical Sciences, Poznan, Poland, ³Poznan University of Medical Sciences, Department of Biochemistry and Molecular Biology, Poznan, Poland, ⁴Poznan University of Medical Sciences, Department of Neurology, Poznan, Poland

Levels of the extracellular matrix protein LTBP2 in Alzheimer's disease may be influenced by the presence of the *APOE* ε4 allele and *Helicobacter pylori* virulence factors.

INTRODUCTION	METHODS	RESULTS	CONCLUSIONS	REFERENCES
<i>Helicobacter pylori</i> may participate in the pathogenesis of Alzheimer's disease (AD) with the participation of its key virulent factor, cytotoxin-associated gene A (CagA), which may lead to the development of dementia through the development of neuroinflammation. Increased inflammation results in increased expression of latent transforming growth factor beta binding protein 2 (LTBP2), which further amplifies neuroinflammation, promoting tau pathology and neurodegeneration. The regulation of the inflammatory response in AD is largely genetically controlled. Currently, the biggest genetic risk factor of AD is <i>APOE</i>, present in 3 alleles: protective (ε2), neutral (ε3), and pathogenic (ε4).	The aim of the study was to analyze CagA and LTBP2 protein concentration and the <i>APOE</i> genetic variants in the peripheral blood of AD patients and related (CR) and unrelated (CU) controls to AD patients. The material included peripheral blood samples from 32 AD patients, 34 related controls (CR) and 33 unrelated controls (CU). CagA and LTBP2 protein concentrations were analyzed using the enzyme-linked immunosorbent assay (ELISA). The <i>APOE</i> genetic variants were determined using real-time polymerase chain reaction (PCR).	The study showed a statistically significant increase in LTBP2 levels observed without the presence of the <i>APOE</i> ε4 allele in AD patients compared with CU (p=0.0224). In addition, a trend toward elevated LTBP2 levels was noted in CagA-positive AD patients compared with the CU group (p=0.0762). Furthermore, a trend toward increased CagA concentrations was observed in AD patients compared with CU (p=0.0740).	It appears that changes in LTBP2 protein levels in AD are influenced by the presence of the <i>APOE</i> ε4 allele as well as by the presence of virulence factors. These findings support a potential interaction between host genetics, bacterial infection, and extracellular matrix dysregulation in AD. 	Dorszewska J, Hurła M, Banaszek N, Kobylarek D, Piekut T, Kozubski W. From Infection to Inoculation: Expanding the Microbial Hypothesis of Alzheimer's Disease. <i>Curr Alzheimer Res</i>. 2023;19(13):849-853. doi: 10.2174/1567205020666230202155404. Piekut T, Hurła M, Banaszek N, Szejn P, Dorszewska J, Kozubski W, Predecki M. Infectious agents and Alzheimer's disease. <i>J Integr Neurosci</i>. 2022 Mar 28;21(2):73. doi: 10.31083/j.jin2102073
CONTACT				
Joanna Poszwa jposzwa@ump.edu.pl 				