CYREX® Laboratories

CLINICAL SPECIFICATIONS

AMYLOID-BETA PEPTIDE

Function:

Amyloid-Beta (Aβ) is a protein fragment from an amyloid precursor protein. As part of the innate immune system, amyloid-beta, in the presence of a microbial infection, becomes oligomerized in order to protect the brain against pathogens such as bacteria, enveloped viruses, fungi and parasites. Its function has not been fully elucidated; however, some potential roles for Aβ have been discussed, such as activation of kinase enzymes, protection against oxidative stress, regulation of cholesterol transport, and anti-microbial activity.

Associated With:

Alzheimer's disease mild cognitive impairment[1,2]
Alzheimer's disease[3]

Known Cross-Reactions: Tau Protein, Presenilin, Rabaptin-5, Aquaporin-4, α-Synuclein, S100-B;[4] *Campylobacter jejuni* CDTs, *Streptococcus sanguinis*, *Enterococcus*, Influenza A+B, Rabies and Human papillomavirus;[5,6] Egg Yolk, Lentil lectin, Pea lectin, canned Tuna, Hazelnut vicilin, Cashew vicilin, Scallops, Squid, Caseins, Alpha-Gliadin, Gliadin Toxic Peptide, Non-Gluten Wheat Proteins (globulins and amylase)[7]

Clinical Significance:

In a healthy brain, Amyloid-Beta (Aβ) protein fragments are broken down and eliminated. Aβ peptide can be called into action as an immune protection against pathogens and thus, in patients with chronic infections, Aβ peptide can accumulate in the brain.[8] If the patient's body cannot break down and eliminate the Aβ, and the Aβ has entered the brain, plaques may form. In Alzheimer's disease (AD), the fragments accumulate to form hard, insoluble plaques. Elevated Aβ peptide antibody production would be expected in response to Aβ accumulation, which can be due to deficient clearance mechanisms and/or increased formation of Aβ peptides.[3] Compared to controls, the AD group showed a correlation between increase in age and higher levels of Aβ IgG in serum. The elevated serum autoantibodies correlated with the Aβ presence in cerebral spinal fluid.[3] High levels of IgG can also be due to the exposure of toxic chemicals that form new antigens which resemble mis-folded Amyloid-Beta. Admittedly, as reviewed in Dorothée,[9] in typical patients with AD, Aβ serum antibody levels may be decreased, increased or unchanged compared to healthy controls. Variations in study results can be due to a variety of reasons. It is important to note that Aβ produced by the gut microbiome has been shown to cross the blood-brain barrier, possibly via specialized receptor-mediated transcytosis, and enter the central nervous system.[10]

References:

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