

AD/PD Research

Alpha 1 Antichymotrypsin

Prepared from human plasma

AD patients seem to express increased alpha 1-Antichymotrypsin (AACT) index, suggesting an intrathecal production of AACT. Indeed, AACT is a major component of the amyloid plaques found in the brains of AD patients. In Parkinson's disease (PD), AACT gene has been suggested as a susceptibility factor that might be related to the onset of PD^{1,2}.

Alpha 1 Antitrypsin

An acute phase plasma protein

Like alpha 1-antichymotrypsin alpha 1-antichymotrypsin might be functionally involved in the pathogenesis of the lesions of Alzheimer's disease. Both serine protease inhibitors are found in neurofibrillary tangles and senile plaques. The major cell producing alpha 1-antitrypsin localization in seems to be the astrocytes, which are involved in both lesions⁵.

Myeloperoxidase

Isolated from whole blood

Myeloperoxidase (MPO) was found to be a key oxidant-producing enzyme during inflammation and it appeared to be upregulated in the ventral midbrain of human PD. It has been suggested that inhibitors of MPO "may provide a protective benefit in PD"³. Although unexpected, myeloperoxidase expression was found in neurons and it increased in brain tissue showing AD neuropathology. Thus it is possible that MPO contributes to the oxidative stress implicated in the pathogenesis of the neurodegenerative disorder.

Alpha 2 Macroglobulin

Prepared from plasma

Alpha 2 Macroglobulin (A2M) seems to play a role in the development of sporadic AD. A2M-D allele was observed as a weak AD protective factor, and the possible interaction of APOE-epsilon 4 and A2M-G alleles may have cause an "increase AD risk in Mainland Han Chinese"⁶.

Plasmin

Isolated from human plasma

The plasmin system is involved in the degradation of Abeta peptides⁷. Brain plasmin enhances APP alpha-cleavage and Abeta degradation and is reduced in Alzheimer's disease brains^{7,8}.

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Now Available

Plasminogen

Isolated from human plasma

Plasminogen is the precursor of the serine protease plasmin which is involved in fibrinolysis. The Tissue Plasminogen Activator-Plasminogen Proteolytic Cascade Accelerates Amyloid- β (A β) Degradation and Inhibits A β induced Neurodegeneration⁹.

Transferrin

Isolated from human plasma

Transferrin is a glycosylated metal-carrying serum protein. Transferrin, the transport protein of iron, has been implicated as playing a role in AD and PD by regulating the iron concentrations in the brain^{12,13}.

Haptoglobin

Isolated from plasma

Particular fragments of Haptoglobin are found in the circulation of patients suffering from Alzheimer's dementia. Haptoglobin fragments have been used in the efforts to better diagnose Alzheimer's disease and determine risk-assessment, disease progression, disease state, therapeutic avenues, among other pharmaceutical agents deemed to be efficacious in treating the disease^{10,11}.

Chymotrypsin

Isolated from human plasma

Chymotrypsin-like proteasome activities were found to increase in cells treated with 10–100 μ M 6-OHDA, while “higher doses caused a marked decline”¹⁴. Such harsh oxidative attacks might cause failure of the ubiquitin-proteasome system (UPS) which leads to protein aggregation and cell death. “Mutations in familial PD have been associated with the failure of protein degradation through UPS”¹⁴.

References

- ¹Pirttilä T., et al., (1994) *Neurobiology of Aging*. May-Jun; **15**(3):313-7.
- ²Haines J.L., et al., *Genomics*, 1996 April; **33**(1):53-56(4)
- ³Choi, D-K, et al., (2005), *The J. Neuroscience*, **25**(28):6594-6600
- ⁴Green, PS, (2004), *J. Neurochemistry*, **90**(3): 724 -733
- ⁵Gollin PA., et al., (1992) *Neuroreport*. Feb;**3**(2):201-3.
- ⁶Chen D., et al., (2004) *J Neurol Sci*. Jan 15;**217**(1):13-5.
- ⁷Shibata, N., et. al., (2006), *Neurobiol Aging*. Jul 5, PMID: 16828203

- ⁸Ledesma, MD, et. al., (2000), *EMBO Rep*. Dec;**1**(6):530-5
- ⁹Melchor, JP., et. al., *The Journal of Neuroscience* (2003) **23**(26):8867-8871.
- ¹⁰[Matsuyama SS](#), et al., (1986) *Hum Hered.*; **36**(2):93-6
- ¹¹European Patent EP1495331
- ¹²Rensburg, SJV., et. al., (2004) *Metabolic Brain Disease*. **19**:89-96.
- ¹³Loeffler, DA, et. al., (1995) *Journal of Neurochemistry* **65**: 710.
- ¹⁴Elkon, H. et al., (2004), *J. Molecular Neuroscience* **24**(3):387-400

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