



Prevention of Alzheimer's Disease



Alzheimer's disease (AD) is the most common cause of dementia, accounting for up to 70% of all dementia cases, and is now estimated to be the third leading cause of death, after heart disease and cancer. AD currently affects 5.2 million people in the United States (US), with projected estimates reaching 13.8 million (115 million worldwide) by the year 2050. Amyloid dysfunction is known to be the main pathological reason in around 20 similar diseases: Parkinson, M. Huntington, ALS and Diabetes type 2 among others.

AD is pathologically characterized by amyloid plaques, neurofibrillary tangles and loss of synapses in the brain. Neurofibrillary tangles consist of hyperphosphorylated tau protein while fibrils of the amyloid beta-peptide (Ab) aggregate into amyloid plaques.

Biomarkers to assess Risk of developing Alzheimer's Disease

Measuring the activity of the enzyme BACE1 in your saliva gives you a very good indication regarding the possible risks of developing AD years before the first clinical onset of symptoms.

At the same time with advise testing a panel of biomarkers to assess your intracellular activity (contrary to classical biomarkers used in assessing organs, such as liver, heart and so on). This panel consists of GPNMB, alpha synuclein, UCH-L1 and 08-OHdG.

Treatment of Alzheimer's Disease

Please bear in mind that prevention is easier than treatment. The loss of brain cells is difficult to replace and involves a lengthy healing process. Treatment for AD include all the preventative measures but includes additional measures such as reducing GPNMB levels with a blood cell based vaccine, intranasal application of mesenchymal stem cells and brain protective cytokines such as BDNF.

Prevention of Alzheimer Disease

is focused mainly on

- reducing the enzyme BACE1 with the exosomes present in the stem cell derived **secretome**, preventing further accumulation of amyloid beta
- degradation of existing amyloid beta with Neprilysin
- Neuroprotective activity of brain derived neurotrophic factor BDNF in the **secretome**
- Neuroprotective activity of glia derived neurotrophic factor GDNF, insulin like growth factor 1 (IGF1) and fractalkine (CX3CL1) in the **secretome**

Amyloid beta (Ab) is a proteolytic product of the amyloid precursor protein (APP) which is sequentially cleaved in an amyloidogenic pathway by beta- and gamma-secretases. Beta-secretase cleaves APP close to the membrane, releasing a soluble APP beta-fragment.

The main enzyme responsible for the production of toxic amyloid beta in the body is BACE1 (Beta site amyloid precursor protein cleaving enzyme number 1).

BACE1 levels in saliva are considered the best biomarker to assess risk for developing AD. Levels of BACE1 in risk patients are high years before the onset of clinical signs.

Neprilysin (NEP) is a plasma membrane glycoprotein. NEP is primarily expressed in the kidney, however, it occurs at much lower levels in many other tissues, including brain. Neprilysin occurs naturally in the secretome of the ad-MSC, which is produced in vitro and in vivo as a means of communication from cell to cells and as a reaction to external influences. NEP degrades amyloid beta.

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