Relation between the onset of AD and the polymorphism of PRNP, gene for protein in PrPc.

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For many years, it has been said that the etiology of AD is similar to etiology and pathogenesis of prion diseases [1,2]. There is still no effective drug, and probably the disease begins up to several decades earlier and in the consequence when the first symptoms are registered there is too late for any possible treatment [3]. There is still actual a speculation that it might be possible to identify a population at AD risk, so that the potential treatment can be implemented at the right time.

We propose to test three patient populations. The first population with diagnosed Alzheimer's disease [4] with clinical symptoms confirmed by neurological and neuropsychological examination with current biomarkers in CSF (amyloid beta protein, tau protein and phospho tau protein) [5.6]). Second population will be without clinical symptoms and with correct CSF results, without the presence of biomarkers. The third population of subjects should be without clinical symptoms and with positive biomarkers in CSF.
The study is to be carried out in the direction of the presence of homozygosity in amino acid methionine encoded at position 129 in the PrPc protein gene and then all people are planed to be examined with IT tools [7,8,9]

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