A CADI metabolic Alzheimer Disease (AD) model was produced by Bio-Modeling System in 2017-2018. The model in its current state is able to produce biomarkers that could probably predict or increase alertness for identifying AD at its very early stages of progression.

Based on the current model we are able to understand and explain:

1. Why AD is specific to humans and not to other vertebrates (with the exception of some other primates that present neuropathological changes common to AD).
2. Why AD progression may take over 30 years before symptoms become apparent.
3. The origination of AD (entorhinal cortex) and its course of progression/ spreading through the different neuronal networks.
4. Why Aβ and Tau phosphorylation are direct consequences of the pathology and not its cause.
5. Why, commonly, tangle formation precedes plaque deposition.
6. The contribution of all brain cells populations (neurons, astrocytes, dendritic cells, etc.) to the progression of the pathology.
7. How, in direct consequence of the above, the animal models for AD are caricatural and existing bibliography should be interpreted with increased alertness and caution.
8. How genetics and lifestyle influence the pathology's incidence.
9. The cause and course of the considerable brain shrinkage during AD progression.
10. How stress, corticosteroids, sugars and cholesterol affect the pathology, clarifying a great deal the inconsistent bibliography.
11. The contribution and interplay (regulation) of the different metabolic networks to the progression of the pathology.
12. How sex hormones come into play.
13. Why all the current programs targeting the A Beta and Tau fail.
14. And, finally, which biomarkers would probably allow detection of AD in its very early stages of progression and how these could be non-invasively obtained.

We invite all interested ***people to contact us for*** more information or any request.