NEURO-DEGENERATIVE DISEASES

Alzheimer's Disease

Definition

Originally described by Dr. Alois Alzheimer in 1907, Alzheimer disease (AD) is the most common cause of dementia AD is defined pathologically by plaques and neurofibrillary tangles (NFT) in the cerebral cortex. Plaques and tangles are associated with synaptic dysfunction, neuronal degeneration, and progressive cognitive decline (AD dementia).

Epidemiology and Genetics

Epidemiology and Risk Factors

An estimated 5.5 million people in the United States have AD.[2] At age 60 years, the prevalence of AD is about 1%. For each 5 years of age thereafter, AD prevalence approximately doubles, reaching 30% to 50% by age 85.[2] Women are more affected than men at a ratio of almost 2:1, partly because of the larger population of women who are older than 70 years; however, the prevalence is higher in women even after statistical correction for longevity. Other reported risk factors include lower levels of intelligence and primary education, small head size, and a family history of the disease. Potentially preventable risk factors include diabetes, hypertension, sedentary lifestyle, smoking, and obesity.[3] Head injury is also implicated as a risk factor for AD in men. The cost of caring for AD patients in the United States is estimated at more than $183 billion annually and rising.

Genetics

About 70% of AD risk at any given age is attributable to genetics. The most common genetic risk factor for AD is the e4 allele of the gene for apo-lipoprotein E (ApoE), which is present in approximately 50% of individuals with AD.[4] e4 hetero-zygosity triples the risk of AD compared with non-carriers; homozygotes have a sevenfold risk. Other less prevalent risk genes and familial tendencies have also been identified

Mutations in the genes for amyloid precursor protein (APP, on chromosome 21), presenilin 1 (PS1, chromosome 14), and presenilin 2 (PS2, chromosome 1) cause autosomal dominant early-onset AD. These mutations account for the majority of familial midlife-onset AD, but represent less than 5% of all AD cases. Sortilin 1 (SorL1) mutations cause late-onset AD.

Pathophysiology

AD's core neuropathologic findings include extracellular amyloid plaques, intracellular NFTs, synaptic deterioration, and neuronal death.[1] Granulovacuolar degeneration in the hippocampus and amyloid deposition in

blood vessels (congophilic angiopathy) may also be seen on tissue examination, but are not required for the diagnosis. The "amyloid cascade" hypothesis posits that amyloid plaques interfere with synaptic activity and initiate a series of downstream effects that cause increasing inter- and intraneuronal dysfunction and, ultimately, cell death.

Amyloid Plaques

Although amyloid plaques may be subclassified according to their composition, all contain forms of β-amyloid protein (Aβ). Aβ is an amino acid peptide formed by proteolytic cleavage of APP by β- and γ-secretase. The main products of this cleavage are $A\beta_{1–40}$ and $A\beta_{1–42}$. A relative surplus of $A\beta_{1–42}$ predisposes toward amyloid aggregation into oligomers and fibrils, which assemble into amyloid plaques An important role for amyloid in AD pathophysiology is implied by the fact that the proteins encoded by APP, PS1, PS2, SorL1, and ApoE are all associated with amyloid generation, processing, and/or trafficking. However, several lines of evidence indicate that amyloid plaques are not the primary cause of AD. Amyloid plaque burden (a) can be found in cognitively normal adults, (b) does not correlate with degree of cognitive impairment in individuals with AD dementia and, (c) is associated with cognitive improvement in some AD mouse models.

Neurofibrillary Tangles

Tau, a protein involved in microtubule assembly, is essential for normal axonal growth and neuronal development. However, hyperphosphorylated tau protein aggregates into helical filamentous NFT that are deposited preferentially within neurons of the mesial temporal lobe (especially hippocampus), lateral parietotemporal region, and the frontal association cortices. The critical role of NFT in AD pathophysiology is suggested by the correlation between location and density of tau NFT and the symptoms and severity of AD dementiaMoreover, some studies have demonstrated that Aβ oligomers are not toxic unless tau is also present.

Neuron and Synapse Loss

The distribution of neuronal cell death and synapse loss is similar to that of NFT. In typical AD, the death of neurons in the nucleus basalis of Meynert leads to a deficit in acetylcholine (Ach), a neurotransmitter involved in memory. This cholinergic deficit is the target of most current treatments. In the brainstem, loss of median raphe and locus ceruleus neurons leads to deficits in serotonin and norepinephrine, respectively. Abnormal cerebral serotonergic and adrenergic activity likely contribute to dysphoria and insomnia in AD

Originally described by Dr. Alois Alzheimer in 1907, Alzheimer disease (AD) is the most common cause of dementiaAD is defined pathologically by plaques and neurofibrillary tangles (NFT) in the cerebral cortex. Plaques and tangles are associated with synaptic dysfunction, neuronal degeneration, and progressive cognitive decline (AD dementia).

There is no medicine or drug to date that could destroy amyloid plaques and allow recovery of neuronal tissue and its cognitive and memory faculties

The progression of the disease is directly proportional to plate development and the amount of β-amyloid protein (Aβ) plaques.

Despite considerable efforts by the Laboratories, there are mainly medications that can temporarily stop the symptomatology of Alzheimer's disease but most medications have side effects from low to severe grade.

Preventive pharmacological medicine does not yet exist or has not proved sufficient

Faced with this serious medical planetary problem, Dr. Christian Daniel Assoun tried to find another pharmacological pathway can bring concrete solutions to the patients, in preventive medicine, or even curative in the long term.

On the basis of the BIO PHYSICS concept of SYNAPTIC TRANSMISSION AND BLOCKING OF APOPTOSIS MECHANISMS.

Namely, the concept of bio-physics(quantum model) applied to neuronal connections that are inactive and no longer exchanging chemical mediators and quantified information because the signals are non-existent.

The intronic quantum foundation of experimental theory of bio-physicsl mechanisms associated with immature DNA-RNA sequences in living genetic material.

Prof. Dr. Daniel Assoun has been working for many years on the biophysical aspects of biological material and therefore DNA-RNA (intronic quantum biology)

First publication 1968- Silicon involved in quantum model of neurons.

It has been shown that our DNA encodes only 5% of the classical biological reactions known as EXONICS

95% of the germplasm appears to be missing visible participation in the biological material protein

This NON-CODING part of our DNA has been defined as INTRONIC RNA

An intron is a non-coding DNA sequence interrupting the coding sequence of a poly partite gene (opposed to exon). Reformulation: fragment of a gene located between two exons. Introns are present in immature mRNA and absent in mature mRNA. "Non-coding" fragment of the gene.

For the past 15 years, biologists have been confident that INTRONIC non-coding parts play an important role in genetic and protein synthesis reactions, contrary to accepted initial ideas.

These immature RNA introns could induce real genetic reactions and intervene in immune defense reactions and disappear once their mission is complete

One might consider immature Intron RNAs that can be assimilated to particular biological MESSENGERS-

Immature RNA Introns, could transmit their information according to mechanisms as well as biophysical (quantum signals) and non-classical chemical and therefore molecular-

Immature DNA-RNA sequences may be considered to encode real but STEALTH proteins, which once the signals transmitted by the transmitters (immature RNA sequence) and received by the genetic material, these sequences would be dematerialized in the medium biological.

Several researchers have devised a NON-MOLECULAR communication system, but BIO PHYSICS.

Indeed, the immature RNA genetic material would transmit quantum-type signals at specific frequencies mainly in the UV (Ultra Violet). Hydrogene serial atomic transition .

Several hundred works exist in this field-

Christian Daniel Assoun laid the mathematical and biophysical bases of intronic quantum medicine 1991 publications and books and conferences, lectures-

On the other hand, one of the strong ideas of the experimental theory provides for the presence of an N-H PLASMA OF HYDROGEN within the DNA double helix, which has been developed in a third catenary or strand of DNA-

This plasma alone could explain the emitted or associated UV radiation of the mRNA material which would bio-quantitatively encode stealth proteins -

More than 570,000 (95%) stealth proteins for 30,000 (5%) stable proteins.

Describing these stealth proteins may be 10^{-2} seconds does not prove that they do not exist, but have the kinetic time to transmit quantified information flows.

The logical consequence of the amplification of these quanta of energy lies in the collateral reasoning for the maintenance of the emitted signal (in order to avoid entropy)

There are very few elements in our biological material that could sustain such signals-

Christian Daniel Assoun has privileged natural elements such as SILICON and Germanium, both of which possess INVERSE OPTO QUANTUM properties - **accept radiation from a source or emit it -**

The final choice on elemental silicon made it possible to construct experimental pharmacological protocols applied to degenerative diseases such as Azheimer or Parkinson.

Several International (3) publications(2015-2017) following academic research and research have demonstrated the benefactor and reconstructive and protective role of Silicon G57 from Glycan Industries Ltd UK and US (patented complex molecule in association with a form of electrochemical (patent) zinc).

Regulatory authorizations and declarations were made in Europe and USA and FDA pending-

The goal of Glycan Industries is to continue research to obtain a drug or functional food that can be used by patients in hopes of returning to a healthy situation and thus rebuilding the neural tissue without adversal effects-

The construction of several laboratories will be necessary.

Several European or International Universities are selected mainly in the USA, which are very close to the experimental theories of Prof. Christian Daniel Assoun.

UCM University Compultense of Madrid(Pharmacology) continues its collaboration with Glycan Industries since 2015

The product will be available for patients next three months .