Alzheimer's disease is perhaps the most important of all the degenerative diseases today because of its frequent occurrence and devastating nature. It is the most common cause of dementia in the elderly, with all that this implies in the way of distress for patients and families and economic loss in the form of the costs entailed in the long-term care of patients totally disabled by the disease. Historically, the term Alzheimer's disease was applied to progressive dementia coming on in late middle life but preceding the senile period, following the original description by Alois Alzheimer in 1907, in which the illness of a woman dying at the age of 55 was depicted clinically and pathologically.

There are many different etiologies contributing to the pathogenesis, though this is a summary of the most important ones. The outstanding pathologic feature is death and disappearance of nerve cells in the cerebral cortex. This lends ultimately to extensive convolutional atrophy especially in the frontal, parietal, and medial temporal regions. There is a corresponding enlargement of the ventricular system, but this is usually not extreme. Two kinds of microscopic lesions are distinctive for the disease. The first, originally described by Alzheimer, consists of intraneuronal accumulations of filamentous material in the form of loops, coils, or tangled masses—referred to as Alzheimer neurofibrillary tangles. The neuropathologic evidence strongly suggests that these fibrillar masses are of major importance in bringing about the death of neurons. Neurofibrillary tangles and loss of synapses are the neuropathologic features most closely linked to dementia. The other histopathologic change that characterizes Alzheimer's disease is the presence of intraneuronal clusters of thickened neuronal processes, both axons and dendrites (collectively referred to as neurites), generally in the form of an irregular ring surrounding a spherical deposit of amyloid fibrils. These lesions, which had been recognized before Alzheimer's description of the neurofibrillary change, were termed senile plaques. Recent elucidation of their structure has led to their current designation as neuritic plaques. They have been shown to contain paired helical filaments identical to those found in the periphery of cytoplasm of the damaged neurons. One form of plaque, the diffuse plaque, consists of amorphous amyloid without neurites.

The amyloid peptide (β or A4 peptide) gene is on chromosome 21, on which the familial Alzheimer's disease gene also has been localized in some families. A recent advance has been the finding that in a handful of families with familial Alzheimer's disease there are point mutations in the amyloid precursor protein. This observation suggests that Alzheimer's disease can be linked to a primary defect in amyloid production or processing, but most cases of familial and sporadic Alzheimer's disease do not have a clear cause.

Biochemical studies show that choline acetyltransferase, the key enzyme required for the synthesis of acetylcholine, is decreased in the cerebral cortex in Alzheimer's disease. The major source of neocortical cholinergic innervation is a group of neurons situated in the basal part of the forebrain just beneath the corpus striatum - the nucleus basalis of Meynert. Careful neuropathologic investigations have shown that in Alzheimer's disease this nucleus is a site of major neuronal loss and of frequent Alzheimer neurofibrillary tangles. These studies suggest that impairment of cholinergic transmission may play a part in the clinical expression of the disease.

The rule of neurotoxic chemicals such as Aluminum, Mercury fillings, and neurotoxic food additives such as MSG have been discussed in many articles to be the etiology of Alzheimer's disease.

Certain areas of the brain show greater vulnerability to glucose deprivation. They are the same temporal and parietal regions wherein the characteristic deficits in Alzheimer's disease (AD) are shown by PET and SPECT imaging studies. Senile plaques and associated neurofibrillary tangles are probably the direct cause of Alzheimer's disease. The principal component of senile plaques is the beta amyloid of protein. There appears to be a reduction in essential fatty acids, with a corresponding increase in saturated fatty acids. This results in increased permeability of cellular membranes and a reduction in membrane fluidity. Consequently, the cellular membranes of Alzheimer's diseased brains might be penetrated by the enzyme that could cut the beta-amyloid precursor protein (B-APP). This would release a large fragment of this protein into the extracellular space with the beta-amyloid section intact. Essential fatty acid deficiency will also reduce prostaglandins. Another neuropathogenesis of Alzheimer's disease is said to be deficiency of essential fatty acids which results in prostaglandin reduction.

Clinical Appearance

The onset of Alzheimer's disease is insidious and subtle, with changes most noticeable first in memory for recent happenings and in other aspects of mental activity. Psychiatric disturbances such as depression, anxiety, or odd, unpredictable quirks of behavior, delusions, and hallucinations may be silent features in the early stages. Progression is usually slow and gradual, and unless other medical conditions supervene, it may smolder on for 10 or more years. In the milder cases, including those of the senile period, the noteworthy features are those of simple dementia. More unusual disorders of thought and intellect, including aphasia, apraxic disturbances, and abnormalities of space perception, may be seen, especially in the presenile group. Exceptionally, and only in the advanced stages of the disease, extrapyramidal signs appear; the patient walks in a shuffling manner with short steps, and there is a generalized stiffness of the musculature with slowness and awkwardness of all movements. In some patients, sudden jerky contractions of various muscles (myoclonus) may occur in the presence of otherwise typical Alzheimer's disease, but this is unusual and should immediately raise the suspicion of Creutzfeldt-Jakob disease. Terminally, the patient may become nearly decorticate, losing all ability to perceive, think, speak or move.
Alzheimer’s
degeneration of the nervous system, exacerbating the dementia. It would be interesting to see if lipid-soluble vitamin supplements alter the rate of the progression of the dementia.\(^7\)

3. **Zinc** (15-60 mg a day): The hippocampus is the area of the brain with the highest zinc concentration. Studies show that chronic neurotoxic medication increases amyloid induced production of NFT (neurofibrillary tangles), while zinc compounds can inhibit amyloid-induced production of neuronal paired helical filaments. Preliminary trials with zinc aspartate have shown promise.\(^11\)

4. **Improve mitochondrial function**: Mutated mitochondrial DNA is associated with cellular dysfunction in close association with the brains of Alzheimer’s patients. By improving mitochondrial function with vitamins B2, menadione (vitamin K) and coenzyme Q10, the function of SDAT (senile dementia of the Alzheimer type) patients may be temporarily improved by enhancing some impaired neurons and slowing mitochondrial decline. Coenzyme Q10, sodium ferrous citrate, and vitamin B6 have shown a benefit in improving mental function.\(^12\)

5. **Vitamin B12**: Studies evaluated the relationship between Vitamin B12 and dementia in Alzheimer’s disease. The deficiency of Vitamin B12 is prevalent in Alzheimer’s patients and must be screened.\(^13\)

6. **Estrogen**: Epidemiological study suggests that estrogen protects women against Alzheimer’s disease. Women using estrogen replacement therapy were 40% less likely to have Alzheimer’s disease and related dementias compared to women not on estrogen.\(^14\)

7. **Thiamine**: Activities of thiamine-dependent enzymes (pyruvate dehydrogenase (PDHC), alpha-ketoglutarate dehydrogenase (a-KGDH), and transketolase (TK)) were measured in autopsy samples of temporal cortex from six patients with Alzheimer’s disease and from eight age-matched control subjects who were free from neurological or psychiatric diseases. Significant decreases in PDHC, decreases were by 70%; a-KGDH, decreased by 70%; and TK, decreased by 52%; were observed in brain tissue from patients with Alzheimer’s.\(^16\)

8. **NADH**: In one study all patients showed improvement in cognitive and functional capacity. The mechanism of action of NADH is believed to be an increased synthesis of neurotransmitters, particularly dopamine and norepinephrine, in the brain; NADH is also believed to increase intracellular energy production.\(^16\)
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9. Botanicals:
   a. Hypericum serata, a Chinese tea contains Hyperzine A and this increases acetylcholine levels in the brain. Thereby improves cognitive function.11,22
   b. Lycoris radiata (adhuan), a Chinese herb, contains the alkaloids lycorine, lycoamine, lycorine, and galanthamine. Both lycorine and galanthamine have been shown to be reversible cholinesterase inhibitors.17
   c. Macleaya cordata (bolohul), a Chinese herb, contains several alkaloids including sanguinarine, chelerythrine, protopectin, and allocryptopine. Sanguinarine inhibits cholinesterase.17
   d. Codonopsis sinensis (huanglian), a Chinese herb, contains berberine alkaloids, mainly berberine, but also coptisine, wogonine, palmatine, and columbamine. Berberine is a cholinesterase inhibitor.17
   e. Berberis species (sankaenden), a Chinese herb, also contains berberine and palmatine that have a strong anticholinesterase activity.17
   f. Securinega suffruticosa (yiyiqiu), a Chinese herb, contains many alkaloids, mainly securinine and its derivatives. Securinine is a CNS stimulant that antagonizes the inhibitory action of meprobamate. Securinine inhibits cholinesterase activity.17
   g. Solanum nigrum (longcut), a Chinese herb, contains solanine, solasodine. Solanine has a strong anticholinesterase action.17
   h. Physostigma venenatum from the calabar bean blocks the action of acetylcholinesterase.18
   i. Pilocarpus jaborandi contains pilocarpine that has M1 mimetic activity, which can affect the M1 receptor site and take the place of acetylcholine in neurotransmission in the Nucleus basalis of Meynert.23
   j. Hawthorne berry, Blueberry, Elderberry, Red and Black grapes improve the integrity of blood vessel walls via antioxyins.25
   k. Ginkgo / Goto kola / Ziziphus jujube improve memory.27
   l. Ginkgo biloba / Salvia officinalis / Melissa officinalis, ginkgo contains the ginkgolides which have antioxidant, neuroprotective and cholinergic activities relevant to Alzheimer’s disease mechanisms. The therapeutic efficacy of Ginkgo extracts in Alzheimer’s disease in placebo controlled clinical trials is reported similarly to currently prescribed drugs such as tacrine or donepezil and, importantly, undesirable side effects of Ginkgo are minimal. Old European reference books, such as those on medical herbs, document a variety of other plants such as Salvia officinalis (sage) and Melissa officinalis (balm) with memory-improving properties, and cholinergic activities have been recently identified in extracts of these plants.28

m. Periwinkle / Hydrocotyle asiatica increases cerebral circulation and function.29

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References
8. Dinihan, M. Griffiths Five Minute Clinical Consult 1996. Williams & Wilkins, Baltimore, Maryland. Pgs. 84.