Plants and their bioactive principles have displayed their ability to restore the cholinergic systems.

9.2.7 Cholinesterase

Cholinergic deficit is the main hallmark of AD. Acetylcholinesterase (AChE) hydrolyzes ACh into choline and acetate in the brain. Hence, cholinesterase inhibition serves as the most effective treatment approach to alleviate symptoms of AD. The extracts from different plant sources are proven to inhibit cholinesterase, thereby enhancing memory and cognition. Increased ACh levels trigger non-amyloidogenic processing, thus lowering Aβ formation and toxicity.

9.3 PLANTS AS A SOURCE FOR AD THERAPEUTICS

Nature is a rich source of biological and chemical diversity. The unique and complex structures of natural products cannot be obtained easily by chemical synthesis. A number of plants in the world have been used in traditional medicinal remedies. Plant species and their derivatives have been used by humans for over 5000 years as medicines (Goldman 2001; Liu and Wang 2008). The bioactive phytochemicals from the extracts of plant species are classified into a number of different chemical groups: alkaloids, phenolics and terpenoids (Samuelsson 2004). A single entity or mixture of components in these groups offer the most fecund potential for the treatment of AD. Scientific research has opened new directions for the use of plants and their derivatives for AD therapeutics from ethnobotanical evidence through cultural, empirical and complementary medical uses of plants. Research in the past decades has revealed that plants are a welcome source of therapeutics for AD, due to their low toxic potential when compared to pharmaceuticals. In humans, plant derivatives as food supplements have been proved to possess inherent abilities to enhance cognitive function and prevent neurodegeneration (Spencer 2007).

One of the primary occurrences of AD is the deficits in the cholinergic nervous system which can be evidenced from the behavioural and cognitive symptoms (Bartus et al. 1982). The use of AChE inhibitors served as an effective therapeutic strategy for many years. AChE breaks down ACh in the synaptic cleft and AChE inhibitors prevent this breakdown, thus providing symptomatic treatment of AD (Cummings 2000; Nordberg and Svensson 1998). Although the pathogenesis of AD is not completely explored, the existence of Aβ plaques, loss of nerve cells and neurofibrillary tangles still occur within the brain. Aβ is the principal component in amyloid plaques generated with β-secretase and γ-secretase by proteolytic cleavage of APP into 39-42 amino acid Aβ peptide (Hardy and Allsop 1991). Aβ peptide, once formed, aggregates and is deposited in the cell membranes and the dendrites of the neurons, and thus triggers dementia leading to AD. The suppression of Aβ production could serve as a therapeutic target in AD.

All the drugs currently available for AD are symptomatic and have their own limitations; they hardly have influence over the advancement of AD and cause side effects in patients. Hence, the hunt for more cogent and competent drugs is always needed. Plants with a potential for AD therapeutics have drawn the heed of the scientific