signal pathways and therefore can act as a potential therapeutic agent in AD treatment. Shilajit is adaptogenic, immunomodulatory and reduces stress (Winston et al. 2007). These unique properties make it an ideal candidate for the treatment of AD. Shilajit in combination with *Withania somnifera* influences cholinergic signal transduction cascade in the cortical and basal forebrain (Schliebs et al. 1997). Tau protein buildup is a hallmark of AD. Fulvic acid, the primary constituent of shilajit, strongly inhibited tau protein aggregation and is one of the few polyphenolic compounds that exhibits this property (Guzmán-Martinez et al. 2013). Fulvic acid is not only capable of inhibiting tau filament formation, but it also breaks apart and untangles the tau filaments (Cornejo et al. 2011). In addition, exposure to fulvic acid resulted in the increased neurite outgrowth in the neural cell cultures. This special feature revealed from *E. royleana* Boiss may open up new possibilities towards AD treatment.

9.3.6 *Galanthus caucasicus*

G. caucasicus produces an alkaloid named galantamine in its bulbs and flowers, which acts as a competitive AChE inhibitor and mitigates the symptoms of AD (Theodorou 2006). Patients in the early stages of AD benefitted from galantamine treatment and exhibited improved cognition, as revealed from the randomized trials (Raskind et al. 2000). Galantamine possesses long-acting, selective, competitive and reversible AChE inhibitory activity for mild to moderate AD (Heinrich and Teoh 2004). The inhibition of AChE increases ACh levels in the synaptic cleft. In addition, it has been shown to slow down the process of neurological degeneration in AD. Galantamine offers neuroprotection (Egea et al. 2012), inhibits Aβ aggregation and therefore cytotoxicity (Matharu et al. 2009), scavenges ROS in neurons protecting them from oxidative damage (Tsvetkova et al. 2013) and promotes neurogenesis (Kita et al. 2014). Cholinergic and glutamatergic dysfunction are the symptoms of AD where galantamine, in combination with memantine, alleviates the impairments of both systems. Thus *G. caucasicus* may serve as a therapeutic agent in AD through neurogenesis and neuroprotection.

9.3.7 *Ginkgo biloba*

G. biloba has the potential to diminish memory loss, improve brain activity and retard the degeneration of neurons in AD (DeKosky et al. 2008). The extract of this plant contains ginkgolides, which possess antioxidants, cholinergic properties and neuroprotective properties and are used to alleviate the detrimental effects of AD (Perry et al. 1999). EGB761 is an extract from *G. biloba* that prevented Aβ toxicity in neurons and proved to be efficacious in AD treatment (Kanowski and Hoerr 2003). In both AD and control patients, the extracts from *G. biloba* enhanced cognitive functions (Rigney et al. 1999). In addition, the extract prevented Aβ and nitric oxide–induced toxicity and reduced apoptosis in neuronal cell culture (Bastianetto et al. 2000; Schindowski et al. 2001). In addition to its antioxidant activities, the extract facilitated a blood supply to the brain, thus enriching cognitive performance (Loffler et al. 2001). In aging and dementia, the efficient recuperation of cognitive deficits with *G. biloba* extracts was evidenced through many studies. Neurotoxicity