Executive Summary of the Project entitled

Neuropromotive effect of the tannoid principles of *Emblica officinalis* on Aluminium chloride induced rat model of Alzheimer's disease

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SUMMARY OF THE FINDINGS

Alzheimer's disease (AD) is an age related progressive neurodegenerative ailment characterized by the presence of extracellular amyloid aggregates and intracellular neurofibrillary tangles. Aluminium is reported to play an important role in the etiology, pathogenesis and development of AD. Recently, many experiments and clinical trials have shown that traditional herbal medicine, which has mulitiple targets, could provide effective treatment of neurodegenerative diseases including AD. Traditionally herbal drugs have been used to enhance cognitive functions and to alleviate other pathologies associated with AD. *Emblica officinalis* Gaertn (amla or Indian gooseberry), a member of the small genus of Emblica (Euphorbiaceae), grows in the tropical areas of India, China, Indonesia, and the Malay peninsula. It is the important dietary source of vitamin C, minerals and aminoacids and also contains tannins, rutin, phyllembelic acid, phyllemblin, emblicol, curcuminoides and various other phenolic compounds. The main tannoids present in *E. officinalis* are emblicanin A, emblicanin B, punigluconin and pedunculagin. The memory enhancing activity of the ripe fruit extract attributed to the presence of polyphenolic compounds and ascorbic acid (Ashwlayan and Singh, 2011). The fruits of *E. officinalis* are rich in tannins than comparing to the whole plant. But till now the effect of tannoid principles of *E. officinalis* (EoT) on AlCl3-induced AD in experimental animals was not reported.

Male wistar rats were divided into control, $AlCl_3$ treated, $AlCl_3$ and EoT (50,100 and 200 mg/kg bw) co-treated, and EoT (200 mg/kg bw) alone treated groups. Intraperitoneal injections concentration of aluminium (Al), activity of AchE and proteins expressions of amyloid precursor protein, $A\beta_{1\text{-}42}$ and γ-secretases, levels of TBARS and diminished the levels of GSH and activities of enzymatic antioxidant as compared to control group. Moreover toxicity of $AlCl_3$ is accompanied by the enhanced expressions of Bax, caspases-3,8,9, cytosolic cytochrome c (cyto c), IL-4,6,10,TNF-α, COX-2 and pTau along with diminished expressions of Bcl 2, mitochondrial cyto c, pGSK-3β and pAkt. Coadministration of EoT nullified the cognitive deficits, Al concentration, AChE activity, oxidative stress, inflammation, apoptosis induced by $AlCl_3$ treatment. Moreover EoT prevents tau hyperphosphorylation by treating the pGSK-3β/ pAkt signaling pathway. This study confirms that EoT would be used as a potential drug candidate for AD and other Aβ and tau pathology-related neuronal degenerative diseases.