

4th National Congress on Medicinal Plants 12, 13 May 2015



Tehran-Iran

1004

COMPARISON STUDIES OF ACETYLCHOLIONESTERASE INHIBITORS PHYSOSTIGMINE AND EXTRACT CELERY FOR ALZHEIMER'S DISEASE

<u>Naderi, Gholamali</u>^{1,*}, Yousefi, Ali¹, Hassanzadeh, Pooya¹, Omidi, Morteza¹ Ejvazzadeh, Shaian¹

¹Biochemistry Department School of Medicine, Shahed University. Tehran, Iran. E-mail: naderi@shahed.ac.ir

Alzheimer's disease (AD) is the most common cause of senile dementia in later life. Whereas several neurotransmitter systems are known to be involved and depleted in AD, the cholinergic system still receives the greatest attention by far. This is particularly true with regards to pharmacotherapy research and development [1] due to the involvement of the cholinergic system in learning and memory processing [2]. One promising therapeutic strategy for re-activating central cholinergic function has been the use of inhibitors of acetylcholinesterase (AChE), the enzyme responsible for the metabolic hydrolysis of ACh. Hypothetically, AChE inhibitors should increase the efficiency of cholinergic transmission by preventing the hydrolysis of released ACh, thus making more Ach available at the cholinergic synapse [3-5]. Such AChE inhibitors as physostigmine or tacrine are known to have limitations for clinical use due to their short half-lives and/or untoward side effects [6]. During screening for AChE inhibitors from natural resources, we found that a total extract of celery showed significant inhibition towards AChE. Celery is a plant. The fruit and seeds are dried or pressed into oil for use as medicine. Some people also take celery juice as medicine. Both physostigmine and extract celery inhibited AChE activity in a dose- dependent manner and Ki values of physostmine and celery were 40 and 5620 µg/ml respectively. This inhibitory effect of physostigmin is 140 fold more than celery. However, celery has not bad effect of chemical drugs.

References

- [1] Giacobini E.ed. by Becker R., Giacobini E., Birkhauser, Boston, 1997, 187-204.
- [2] Deutsch J., Science, 313, 7-11 (1985).
- [3] Collerton D., Neuroscience, 1986, 19, 1-28.
- [4] Bartus R. T., Dean R. L., III, Beer B., Lippa A. S., Science, 1982, 217, 408-414.
- [5] Benzi G., Morreti A., Eur. J. Pharmacol., 1998, 34,6, 1-13
- [6] 6-Parihar, M.S; Hemneni, T.J Clin Neurosci. 2004, 11(5); 456-67.