



Department of Biomedical Engineering
Faculty of Engineering

Mahidol University

Wisdom of the Land



EGBE 693 Research Seminar for Biomedical Engineering II **EGBE 691 Biomedical Engineering Seminar II**

Date: April 29, 2014

Time: 10AM-11AM

Department of Biomedical Engineering

Mahidol University (Room 6373)



Narawut Pakaprot MD.PhD.

Department of Physiology

Faculty of Medicine Siriraj Hospital, Mahidol University

**“Effect of Thai herbal medicine on learning and memory in dementias:
Behavioral, electrophysiological, and histological studies.”**

Learning and memory is a brain function that allows animals to acquire new experiences and knowledge in order to be able to change their behaviors accordingly. The neural mechanisms responsible for learning and memory processes rely on very intricate interactions between neurons. One of the important mechanisms underlying learning and memory formation is long-term potentiation (LTP) which is a long lasting enhancement of synaptic responses. Alzheimer disease is the most common cause of dementia, and the 1 in 3 American seniors will die from Alzheimer and other dementia. The first symptom of Alzheimer patients is memory impairment. Studies in animal models have shown the correlation between learning and memory deficit, and the impairment in LTP formation. Currently, many medications have been used to treated Alzheimer disease and other dementia; however, all the medications only slow down the progression of the disease, but will not cure it. Many herbal medicines such as Ginkgo and Tiger’s herb have been demonstrated that they possess a memory enhancing effect. Our laboratory is interested in a plant called Brahmi. The memory enhancing effect of Brahmi extract has been investigated by employing behavioral, electrophysiological, and histological studies. The data have shown that Brahmi extract could enhance the learning and memory performances in correlation with the increase of LTP. Moreover, it has a therapeutic effect against some types of dementia such as vascular dementia by preventing neuronal death after the cerebral hypoperfusion event.