



Positive effects of Teucrium polium on rat brain cholinergic network deficits caused by amyloid β

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INTRODUCTION

Alzheimer's disease (AD) is a progressive age-related neurodegenerative disorder characterized by progressive cognitive deficits. Neurodegenerative diseases are multifactorial disorders in which many biological processes (including cell signaling, apoptosis, and accumulation of aggregation-prone misfolded proteins) become unregulated. Several complex pathogenic pathways have been found to be involved in AD development and progression, including plaque formation, inflammatory cascade, cholinergic deficit, oxidative stress, etc.

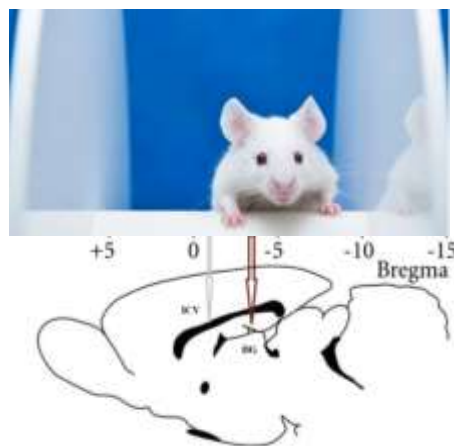


Teucrium polium

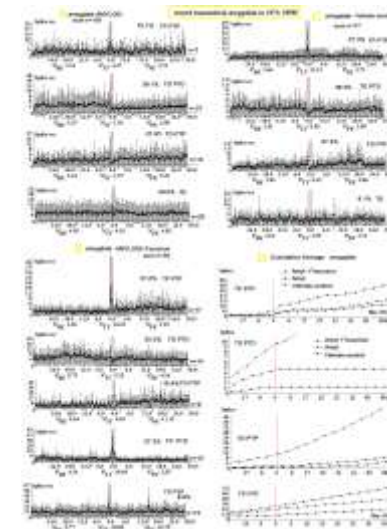
MATERIALS AND METHODS

In Vivo extracellular electrophysiology

15 weeks after $A\beta$ (25-35) infusion, the animals were anesthetized (Urethan 1.1 g/kg), immobilized with 1 % ditiline (25 mg/kg i/p), fixed in a stereotaxic head frame, and placed on artificial ventilation. The stimulatory electrode was inserted according to stereotaxic coordinates [Paxinos, Watson, 2005]. in the ipsilateral nucleus basalis magnocellularis (nbM) and a glass recording electrode (1–2 μ m tip diameter) filled with 3M KCl was repeatedly submerged into the hippocampal fields (at coordinates AP -3.3; L \pm 1.5-3.5; DV 3.0-4.0) and basolateral amygdala neurons to record the spike activity flow of single neurons. Rats were injected intracerebroventricularly (i.c.v.) with beta 25–35-amyloid peptide (Sigma-Aldrich, St. Louis, MO, USA) according to the method described by Maurice et al.



RESULTS



Peristimulus mean frequency diagrams, built on the basis of pre- and post-stimulus manifestations of spike activity of single amygdala neurons to high-frequency stimulation of nucleus basalis magnocellularis (NBM) in real time 30 sec before stimulation (Mbe), 30 sec after stimulation (Mpe) and during high-frequency stimulation (Mtt) exhibiting the specified type of responses (TP PTP, TD PTD, TD PTP) and nonreactivity in Amyloid (A), Amyloid + Teucrium (B) and Vehicle-control (C) groups. D – Cumulative Average curves of the responses of amygdala neurons in the above-mentioned experimental groups.

One of the earliest pathological events in most patients with clinical AD is a cholinergic deficit (dysfunction and loss of basal forebrain cholinergic neurons and their projections) [Wevers et al. 2000]. Plants are potential sources for drug development against AD. Our results contribute to the understanding of the role of amyloid β in synaptic disruption in the pivotal cholinergic network and Teucrium polium to enhance synaptic plasticity.

This work was supported by the Enterprise Incubator Foundation, Faculty Research Funding Program, 2022