II. Aim of this work

VILIP-1, a neuronal calcium sensor protein, is mainly expressed in retinal and brain neurons. However, the expression profile of VILIP-1 in peripheral organs was not yet studied, although VILIP-1 can be detected in squamous carcinoma cells of the skin. Interestingly, the abundance and special distribution pattern of VILIP-1 found in rat hippocampus compared to other neuronal calcium sensor proteins, together with modulation of VILIP-1 expression (up-regulation in certain interneurons and down regulation in the principal neurons in the hippocampus) in the brains of schizophrenic patients and ketamine treated rat suggests that VILIP-1 might be involved in synaptic plasticity and some pathological processes. The hippocampus attracts interest due to its unique structure and beautiful model of synaptic network. As one of the most studied models of synaptic plasticity, LTP plays a part in memory, spatial navigation and many other processes, as well as in diseases such as epilepsy, Alzheimer's disease (AD) and schizophrenia.

Therefore, my objectives were:

1. To study the distribution of VILIP-1 in peripheral organs, and rat hippocampus with the help of immunohistochemical studies, in order to get information about the precise distribution of VILIP-1.
2. To study the regulation of VILIP-1-expression in a model of synaptic plasticity with the help of Western blot analysis, in order to obtain insight into a possible involvement of VILIP-1 in hippocampal synaptic plasticity.
3. To study the functional effect of interaction between VILIP-1 and α4β2 nAChR with the help of electrophysiological measurements in hippocampal cultures, in order to analyze the molecular mechanisms of VILIP-1 in synaptic plasticity.