

## 1.6 Objectives

The overall hypothesis of this dissertation was that immune cells from inflamed LN express and upregulate signal sequence-encoding POMC mRNA, as well as its end-product END. To this end POMC mRNA and END production in lymphocytes was analyzed by biochemical (radioimmunoassay), cell biological (flow cytometry and immunohistochemistry) and molecular biological (PCR, cloning) methods in a model of inflammatory pain. In particular it was examined

- whether lymphocytes express POMC mRNA including the signal sequence or express truncated POMC mRNA only;
- the kinetics of lymphocytic POMC gene expression;
- which lymphocyte sub-populations express PC1/3 and PC2 mRNA;
- the kinetics of lymphocytic END production;
- which lymphocyte sub-populations contain END;
- whether POMC gene expression and END synthesis are stimulated by the inflammatory mediator IL-1 $\beta$ .