3 AIM OF THE STUDY

This work was intended to study the effects of selenium on the activation of microglial cells under oxidative stress conditions. Selenium was shown to protect neurons from primary neuronal damage, but so far its effects on the microglial cells have not been investigated. The microglia are the macrophages of the brain. The activation of microglial cells occurs during the pathogenesis of various neurologic diseases. After activation, the microglia cells migrate towards the sites of the neuronal injury where they phagocyte injured cells and produce large amounts of free oxygen radicals, which cause severe secondary neuronal damage. For this reason, to understand how selenium affects these mechanisms is of interest with regard to the potential therapeutic use of this element in the protection of neurons from secondary damage.

In particular, the objectives of this thesis were:

1. To analyse the effects of the selenium status on the activation of microglial cells in vitro.
2. To study in which way selenium prevents the activation of microglial cells induced by oxidative stress conditions.
3. To compare the selenoprotein patterns of microglial cells in relation to their selenium content.
4. To construct novel plasmids for the regulation of the Gpx1 protein expression and to establish the conditions for their transfection.
5. To evaluate the role of Gpx1 by its over-expression and down-regulation in microglial cells.
6. To examine the importance of the selenium status in microglial cells in vivo.