Cellular prion protein (PrP^C) is a membrane bound glycoprotein. The protein is expressed in all vertebrates, mainly in the nervous system, but it is present also in the cells of gastrointestinal tract, bone marrow, germ cells and heart. PrP^C is necessary for pathogenesis of prion diseases, which are deadly and without the possibility of therapy. The pathogenic isoform of prion protein is formed by changing of secondary structure of PrP^C and it's the main constituent of infectious prion particles. Pathological form of prion protein accumulates in brain of infected patients and this process is associated with neurodegradation. Physiological function of PrP^C is poorly understood. Knock-out of the PrP^C gene (*PRNP*) is not connected with any noticeable phenotype. Potential functions of PrP^C are dispersed, protein may have antiapoptotic effect, it can be involved in ions metabolism or in protection against oxidative stress. Latest results show, that PrP^C can play important role in cell differentiation. During the differentiation PrP^C can influence the development of cells and their typing. It could affect cell cycle and have an influence on formation of nervous system. Aim of the present study was to elucidate, whether the down-regulation of PrP^C or infection with prions has an impact on differentiation of neuronal cell line CAD 5. This thesis compares cell lines of CAD 5 with physiological expression of PrP^C and transduced lines of CAD 5, with lower expression of PrP^C due to RNA interferention. This work also looks into the influence of RML strain infection (scrapie adapted to mouse) in differentiation of CAD 5 cells. The lines were compared in their morphology, in ability of growing and differentiation, in expression of neuron-specific marker GAP-43 and in the expression of PrP^C. During seven days of differentiation the amount of PrP^C increased five times. The morphology study didn't found significant differences between normal PrP^C expressing cells (WT) and cells with down-regulated PrP^C (LP1 and LP2). There has been a difference in the morphology of RML infected cells during first three days of differentiation, when infected cells changed their morphology later then healthy cells. Expression of marker of differentiation GAP-43 in WT – healthy CAD 5 cells was similar to lines with down-regulation of PrP^C and infected cells. The results of this study do not provide any evidence that there is a difference in morphology or expression of GAP-43 in CAD 5 with normal expression of PrP^C and CAD 5 with down-regulation of PrP^C. Different morphology between healthy and RML infected cells was observed during early differentiation. The difference in the dynamic of differentiation of prion infected cells could be one of factors contributing to the brain damage during prion diseases.