Around 290 000 women are diagnosed with endometrial cancer (EC) worldwide each year. EC is associated with chronic exposure to estrogens and concurrent insufficient protection by progesterone. In endometrium progesterone may act through the classical nuclear and nonclassical membrane bound progesterone receptors. Progesterone exerts genomic actions via nuclear progesterone receptors, PRA and PRB, where PRA prevents estrogen-stimulated proliferation of endometrium. Rapid nongenomic actions of progesterone may be mediated via recently discovered membrane progesterone receptors (mPRs) that are coupled to G proteins. The expression and role of PRA and PRB in EC have been studied extensively showing that development of cancer is associated with changes in the expression of PR isoforms. However, the mPRs have not been examined in EC so far. Studies in breast cancer cell lines revealed that progesterone inhibits apoptosis through mPRs. Additionally, expression of mPR is up-regulated in breast cancer tissue [1], thus supporting the role of mPRs in carcinogenesis. Although pathogenesis of breast and endometrial cancer differ we hypothesised that expression of mPR is altered in EC. Our qPCR analysis in EC and adjacent control tissue showed decreased expression of both nuclear (PRA and PRB) and membrane bound progesterone receptors (mPR? and mPR?). On one hand, decreased levels of PRA may lead to lower protection of progesterone against growth-promoting effects of estrogens while reduced levels of mPR? and mPR? may increase cell apoptosis. We also showed that the mRNA levels of PRAB, PRB and mPR? are diminished in G2 and G3 EC cases only, while there is no difference between pre- and post-menopausal cases. Our data indicates that expression of these receptors is tumor-grade dependent. Further analyses at the protein levels are currently in progress.