Endometrial cancer (EC) is an estrogen-related cancer. Estrogens exert their biological effects through the estrogen receptors (ER). There are three recognized ERs: the classical ERα and ERβ that function traditionally as ligand-activated nuclear transcription factors, and the G-protein-coupled ER (GPER) that is involved in rapid signaling events. The cellular effects of estrogens depend on the specific receptors expressed in the tissues. It is thought that the ER status constitutes an independent prognostic factor, but the precise role of each ER type is not yet clear. Since many cells and tissues co-express classical estrogen receptors and GPR30, we aimed to examine their status in cancer and control endometrium. We investigated ERα, ERβ, and GPER at the mRNA, protein, and cellular levels. Our qPCR analysis revealed decreased ERα and ERβ mRNA levels in cancer endometrium, compared to adjacent control. Also, we observed unchanged GPER variant 2 expression and decreased levels of variants 3 and 4 in the cancer. Lower mRNA levels of ERα and GPER were confirmed at the protein level by western blotting, while the ERβ protein levels were higher in cancer compared to controls. Moreover, using immunohistochemistry analysis, we evaluated the ERα and ERβ cellular status. Both ERα and ERβ were detected in the nuclei and cytoplasm of the glandular cells of the tissue specimens. For ERα, the staining was stronger in nuclei, but the scores were significantly lower in cancerous glands compared to control tissues. ERβ staining in nuclei and cytoplasm was similar in cancer and control specimens. The immunohistochemistry analysis of GPER is currently in progress. Our data imply that in cancerous endometrium estrogens may exert their effects through increased levels of ERβ but also through lower levels of ERα and GPER.