

Licensing Opportunity (BioT-1059-UMG)

Oligopeptide Cancer Therapy for steroidal cancers and melanoma

Steroid-related cancers have the highest incidence among tumors and its market potential for Europe and U.S.A. is estimated to be at least **US\$ 2.4 bn p.a.** **Malignant melanoma** is one of the most aggressive cancer and there is no therapy known. Its market corresponds to **US\$ 1.2 bn p.a.**

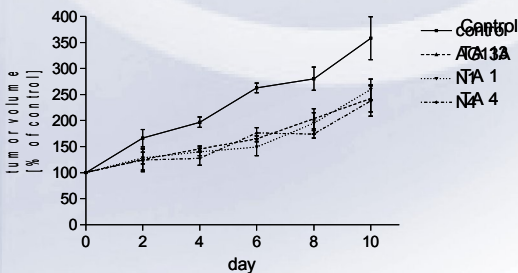
Scientists at the University of Göttingen developed new antagonists to the target receptor GnRH-II for the therapy of steroidal cancers, which potentially could also be used to treat malignant melanoma.

Hallmarks

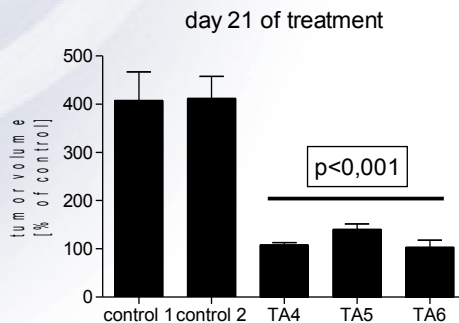
- Oligopeptide antagonists to target receptor GnRH-II.
- Target receptor is highly expressed in steroid related cancers (ovarian, endometrial, prostate and breast cancers).
- Target receptor is highly expressed in Malignant Melanoma.
- Target receptor is also expressed in reproductive organs, but less in normal tissue.
- High water solubility of oligopeptide antagonists.
- Target Antagonists inhibit tumor proliferation.
- Target Antagonists increase tumor apoptosis.
- *In vivo* proof of concept achieved for human Ovarian cancer, Endometrial cancer and Breast cancer.

Proof of concept

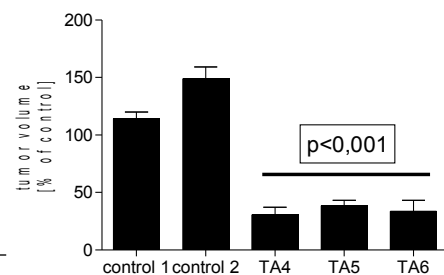
i.p. injection of Target Antagonists (TA) in mice with implanted carcinoma cells.



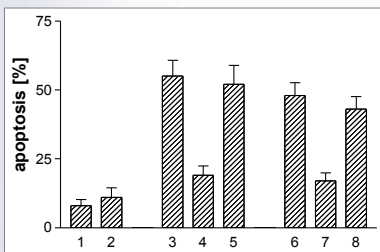
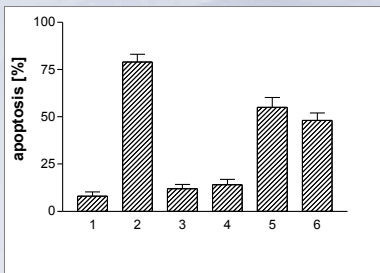
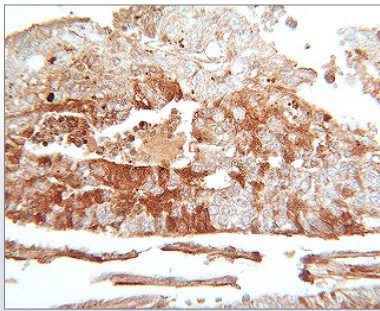
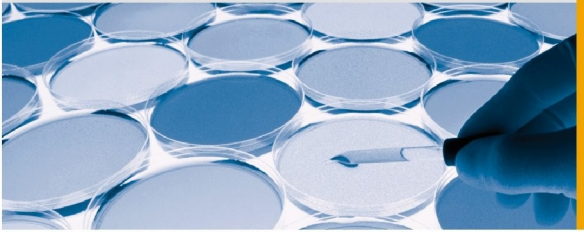
Ovarial cancer



Endometrial cancer



Breast Cancer



The target receptor is highly expressed in steroid related cancers, like Endometrial carcinoma (fig. left), Mamma carcinoma, Ovarian carcinoma, Prostate carcinoma, and also in Malignant melanoma.

Current agonist and antagonists to receptors related to the target receptor mainly inhibit the growth/proliferation of carcinomas, whereas the newly developed antagonists from Göttingen also induce apoptosis besides inhibiting tumor proliferation, as shown in the next experiments.

Target receptor antagonists increase tumor apoptosis.

In vitro results using Mamma carcinoma cells after 96 h of culture with 1) Control (no treatment), 2) Target receptor knock out, 3) Control (transfection), 4) Target agonist, 5) Target antagonist TA1, 6) Target antagonist TA2. Same results have been achieved using several of our newly developed target antagonists against different cancer cell lines even at low concentrations.

Specific mechanism. The activity of the target receptor antagonist could be reverted by the target receptor agonist. The target receptor does not show cross reaction with other agonists to related family receptors. In vitro results using Mamma carcinoma cells with: 1) Control, 2) Target agonist, 3) Target antagonist TA1, 4) Target antagonist TA1 after pre-treatment with target agonist, 5) Target antagonist TA1 after pre-treatment with family receptor agonist, 6) Target antagonist TA2, 7) Target antagonist TA2 after pre-treatment with target agonist, 8) Target antagonist TA2 after pre-treatment with family receptor agonist.

We filed a patent application covering this family of antagonists including its application particularly for steroidal cancer therapies and melanoma, and are now **looking for companies, which are interested in licensing, developing and commercializing our approach.**

Reference:

Eur J Endocrin 2005 153 613-625
Cancer Res 2007;67:(4) 1750-1756