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NEUTRALIZING ANTIBODY FOR IMMUNE REACTIVATION IN CANCER

Introduction

Most human and experimental tumors educate the immune system to promote rather than suppress tumor development. Therapeutic feasibility of re-activating the tumor-associated immune is demonstrated by immune checkpoint inhibitors, which are neutralizing antibodies directed against immune checkpoint ligands and receptors. However, only a subset of cancer patients benefit from immune checkpoint blockade. Therefore, other strategies to re-activate the tumor-associated immune system alone or in combination with current standard therapy or immunotherapy are required.

Invention

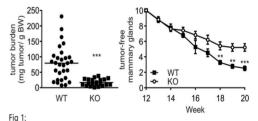
The discovery of an interleukin that is produced in tumors and suppresses the anti-tumor activity of a number of immune cells opens up a new approach to the therapy of cancer. The invention comprises a neutralizing against said interleukin, which is suitable for therapeutic administration. Because of its activity in an experimental tumor model that is resistant to current immune checkpoint therapy (anti-PD-1), the target may be suitable for patients/entities that do not respond to immune checkpoint blockade and for use in combinatorial approaches.

Market Potential

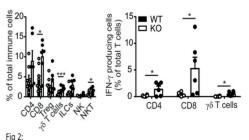
Commercial applications: monotherapy or combination therapy of solid tumors

Development Status

The concept has been demonstrated in gene knockout studies in a mouse model of mammary carcinoma and is currently under investigation in further animal experiments.



Wildtype (WT) and Knockout (KO) mice were bred into the polyoma middle T oncogene (PyMT) mammary carcinoma background and tumor development was monitored. Tumor burden and the number of tumor-free mammary glands (of 10 in total) at an age of 18 wweks are shown. Data are means ± SEM, n=10, **p<0.01, ***p<0.001.



Wildtype (WT) and Knockout (KO) mice were bred into the PyMT mammary carcinoma background and tumor development was monitored. Major lymphocyte populations infiltrating PyMT tumors and IFN- γ + T cell subsets were determined by flow cytometry at week 18. Data are means ± SEM, n.56, *p<0.05, ***p<0.001.