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Opinion Article

An Epcam/CD3-Bispecific Antibody in Stimulation of Tumour Growth

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DESCRIPTION

Cancer stem or Tumor-Initiating Cells (TICs) are a serious barrier to the use of traditional therapies for the disease because of their resilience to genotoxic and anti-proliferative medications as well as their capacity to form tumours and metastases from very few cells. Here, we investigated whether human T cells can lyse colorectal TICs and inhibit tumour formation from TICs when they are guided by an EpCAM/CD3-bispecific antibody known as MT110. EpCAM, a cell adhesion molecule found on TICs from several human carcinomas, is recognised by MT110 and has recently been demonstrated to stimulate tumour growth by activating components of the wnt pathway. In a soft agar assay, it was shown that MT110 was highly effective in mediating complete redirected lysis of colorectal TICs with KRAS, PI3 kinase, and mutations.

MT110 inhibited the development of tumours in immunodeficient mice following a 5,000-fold increase of a marginally tumorigenic TIC dosage. A powerful therapeutic method to eliminate TICs and bulk tumour cells produced from them may be provided by T cells activated by MT110. Over the previous 55 years, deaths from heart disease, stroke, and infectious diseases (such as the flu and pneumonia) have decreased and 58%, respectively. However, overall cancer mortality only decreased by 5%. (National Center for Health Statistics). One explanation could be that so-called cancer stem or Tumor-Initiating Cells (TICs) are resistant to conventional cancer treatments.

Due to the overexpression of detoxifying enzymes and multidrug resistance pumps, predilection for hypoxic environments, and low growth rate, this highly tumorigenic subgroup of cancer cells is difficult to detect and resistant to many chemotherapeutic treatments. Therefore, there is a lot of study being done on the importance of TICs for biology and cancer therapy. If the theory behind TICs is correct, then innovative medicines that target their eradication may treat cancer more effectively, if not completely. Today, TICs have been found and defined in a wide variety of human cancers. TICs have been identified by many laboratories employing antibodies that are specific for the epithelial cell adhesion molecule, such as from colorectal and pancreatic cancers (EpCAM; also called ESA).Additionally, it was demonstrated that the expression of EpCAM and CD44 correlated with the tumorigenic phenotype of such cells. The majority of human adenocarcinomas' primary tumours and metastases frequently express EpCAM at high levels. EpCAM overexpression is associated with a poor prognosis for survival in a number of human malignancies, including breast, ovarian, ampullary pancreas, gall bladder, and liver cancers.

Recent research has identified EpCAM as a cancer stem cell marker that is expressed along with CD44, CD133, and CD166. Because EpCAM can be activated by controlled intra-membrane proteolysis and behave as a signalling protein and proto-oncogene, TICs and their offspring may exhibit EpCAM.

It has been demonstrated that the EpCAM's released intracellular domain, or EpICD, joins with the transcription factors TCF/LEF, -catenin, and FHL-2 to form a nuclear complex that regulates the expression of the c-myc and cyclin genes. Tumor development is triggered by full-length EpCAM or EpICD overexpression in dormant cells. Many antibody- and vaccine-based treatment methods, some of which are now in clinical research, have chosen EpCAM as their target. Recently, a trifunctional anti-EpCAM antibody received market authorization in Europe.. Whereas there is evidence to suggest that EpCAM on normal epithelial tissues is mostly sequestered while it is accessible on the surface of cancer cells, certain normal epithelial tissues and embryonic stem cells produce EpCAM.

The T cell-engaging antibody MT110 belongs to the BiTE class, which stands for a T cell engager with dual specificity for CD3 and EpCAM. A thorough evaluation of the BiTE antibody concept has been done. Any cytotoxic T cell and a target cell that is bound by the BiTE antibody can create a cytolytic synapse thanks to BiTE antibodies. This will completely activate T cells for granzyme B synthesis, serial lysis mode adoption, and redirected lysis. In recurrent non-lymphoma Hodgkin's patients, the CD19/CD3-bispecific BiTE antibody blinatumomab demonstrated strong anti-tumor efficacy, offering clinical proof

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of concept for the therapeutic premise of BiTE antibodies. MT110 is now being investigated for safety and early signs of activity in patients with lung or gastrointestinal malignancies in a dose-escalating phase 1 clinical research. Studies in mice using a BiTE binding to murine CD3 and EpCAM showed anti-tumor efficacy without EpCAM destruction, indicating a therapeutic window.