

## Role of Epithelial Cell Adhesion Molecule in Human Pancreatic Cancer Stem Cells

## Patrick Dorado\*

Department of Pathology and Genetic, University of Southern California, California, United States

## DESCRIPTION

Early metastases and significant resistance to chemotherapy and radiation are characteristics of pancreatic cancer, the fourth most common cause of cancer-related death. Despite intensive research efforts, there hasn't really been much change in clinical objectives over the past few decades. The Whipple surgery, a highly invasive and involved surgical procedure, is the only viable treatment option for pancreatic cancer currently available. Only a small percentage of patients with local disease are candidates for surgical surgery, despite the fact that these patients have a long median life of 20 months. The antimetabolite gemcitabine was introduced more than ten years ago, and it has since improved clinical response for patients with advanced disease by lowering pain and weight loss. However, with a 5-year survival rate of only 3% to 4% and a median survival time of just 4 to 6 months, the prognosis for patients with metastatic pancreatic cancer has remained dreadfully dismal. Therefore, it is imperative to discover novel strategies for attacking the intricate biology of pancreatic cancer in order to pave the way for the creation of more potent therapies for these patients.

Stem cells may be crucial in the formation of complex multicellular organisms as well as the growth and development of malignancies, according to mounting data. Different types of human malignancies may arise and persist as a result of cells with stem cell qualities. A Cancer Stem Cell (CSC) is a cell within a tumour that has the capacity to self-renew and to produce the heterogeneous lineages of cancer cells that make up the majority of the tumour mass, according to the current accepted definition. Recently, evidence suggesting a hierarchical organisation of human pancreatic cancer was presented by us and others. The fact that pancreatic CSCs are heterogenic and that a subpopulation of CD133+ cells may be recognised by additional expression of the chemokine receptor CXCR4 bears

unique metastatic activity is significant. These extremely resistant tumorigenic CSCs could be the cause of the almost unavoidable relapse of pancreatic cancer due to their resistance to standard chemotherapy.

In fact, it is conceivable that the CSCs have not been totally killed when chemotherapy completely eradicates the bulk of the cancer only to be followed by a relapse. Malignant cells, especially CSCs, commonly overexpress and have altered functions of the Epithelial Cell Adhesion Protein Molecule (EpCAM; CD326). EpCAM is expressed by some types of normal epithelial tissues as well as embryonic stem cells, but new research suggests that EpCAM in normal epithelial tissues is primarily confined to intercellular spaces while becoming accessible on the surface of cancer cells that have disintegrated. EpCAM may therefore be a suitable therapeutic target for cancer cells that express EpCAM, including tumorigenic CSCs.

They proposed that MT110, a Bispecific T-cell-Engaging (BiTE) antibody construct, may also target and eliminate EpCAMexpressing pancreatic cancer cells, including CSCs, by diverting cytotoxic effector T cells. MT110 targets both EpCAM on tumour cells and the T-cell receptor-CD3 complex on T cells. In numerous xenograft models, MT110 and comparable EpCAMspecific BiTE antibodies have already demonstrated strong anticancer efficacy. It's significant to note that research in syngeneic animal models using a BiTE binding to murine EpCAM and murine CD3 demonstrated anticancer efficacy without damaging normal epithelia, which expressed EpCAM at a quantity and distribution identical to what was observed in human tissues. MT110 is now being evaluated for safety and early indications of anticancer activity in a dose-escalating phase I clinical trial involving patients with various epithelial malignancies.

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Correspondence to: Patrick Dorado, Department of Pathology and Genetic, University of Southern California, California, United States, Email: Patrick.dorado@email.com

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