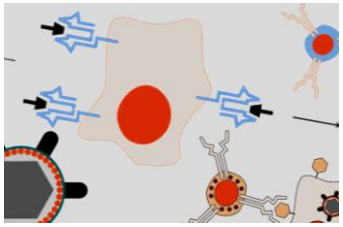
Engineered T-cells have the potential to revolutionize Cancer therapy

T-cells are a type of white-blood cell which form part of our immune system. The role of T-cells in our body is to detect and kill any cells which have become infected with a virus. T-cells are incredibly powerful machines, able to precisely hunt down and kill infected cells. They also can divide and survive in our bodies indefinitely, continuously patrolling for recurrence of the infection. Since cancer cells develop from normal cells in our body, T-cells rarely recognize them as something they need to attack.. Using genetic engineering tools developed originally for gene- therapy, these T-cells can be “re-programmed” in the laboratory so that a patient’s own T-cells recognize and kill cancer cells. The ATECT consortium represents the joint forces of three leading universities and two biotech companies to work towards easier ways of manufacturing these T-cells and also ways of making these engineered T-cells attack cancers more efficiently..



ATECT CONSORTIUM

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http://atect-fp7.org/

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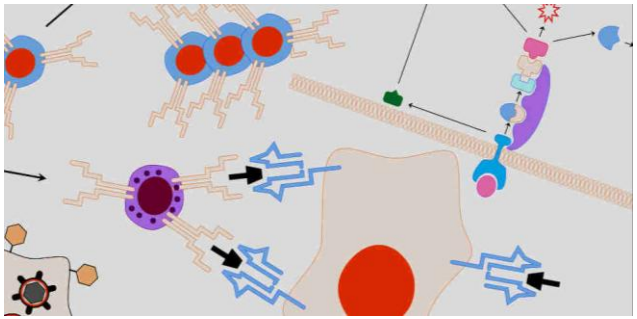
ATECT Consortium

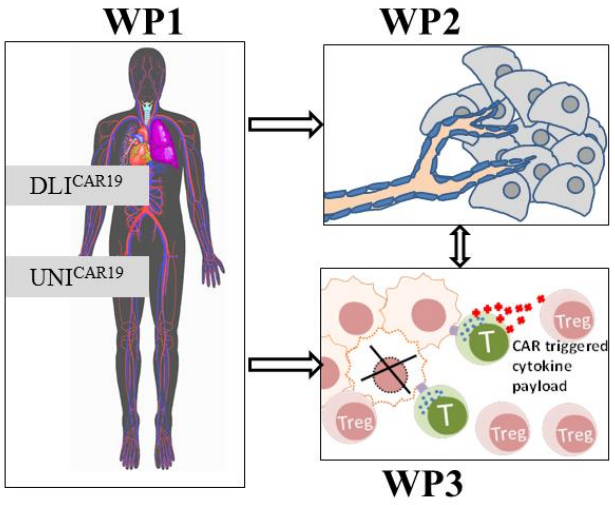
ATECT stands for "**A**dvanced **T**-cell **E**ngineering for **C**ancer **T**herapy".

We are an European Union FP7 funded consortium whose aims are to apply advanced genetic engineering methodologies to Cancer Therapy.

We will use a combination of technologies including Chimeric Antigen receptors, lentiviral vectors and genome editing tools to generate highly advanced therapeutic cellular products.



Scientific aims of ATECT



T-cell engineering strategies for Cancer therapy, either Chimeric Antigen Receptors (CARs) or TCR transfer holds promise to revolutionize cancer treatment. There are, however, considerable barriers to be overcome to take this form of therapy to a format that can benefit all EU citizens with a wide range of common cancers. The aim of this consortium is to exploit advances in T-cell engineering to allow the full potential of CAR therapy to be unleashed

At present, CAR therapy requires a bespoke autologous therapeutic product for each patient. This greatly limits practicality, scalability and commercialisation. The development of a strategy for creation of universal engineered T-cells is the first key aim of this consortium. There is an increased appreciation of the immunological hostilities (CAR) T-cells face in the tumour microenvironment, and prevention of this local immune suppressive effect will likely be critical in permitting effective tumour control

The second main aim of this proposal is therefore to engineer CAR T-cells to be resistant to the hostile microenvironment. CAR T-cells can only be effective if they can access the tumour site. Exploiting the fact that neo-angiogenesis is a hallmark of neoplastic progression, the third aim of the consortium is to utilise endothelial cues of neo-angiogenesis to direct CAR T-cell migration and activity.

Partners of ATECT

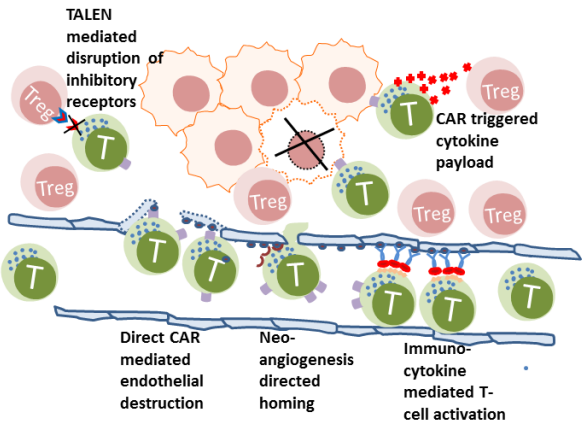
Partner 1: UCL | University College London : UCL is Europe's leading health research multidisciplinary university with the one of the world's major concentrations of biomedical researchers grouping 8,000 staff and 22,000 students. UCL has particular expertise in cell and Gene Therapy. Much of the work of the ATECT consortium will be executed at the UCL Cancer Institute.

Partner 2: CTX | Cellectis Therapeutics: Founded in France in 1999, the Cellectis Group is based on highly specific DNA engineering technologies. Cellectis is today one of the world leading companies in the field of genome engineering. The Group has a workforce of 230 employees working on 5 sites worldwide: Paris & Evry in France. Cellectis Therapeutics is a subsidiary of Cellectis. Its aim is innovative strategies for the development of wide-reaching therapeutic treatments based on core Cellectis technology.

Partner 3: PHL | Philogen: Philogen was founded in 1996 with the mission to develop new biopharmaceuticals for the treatment of angiogenesis-related disorders, such as cancer and rheumatoid arthritis. The company has been a pioneer in the isolation, engineering and clinical development of lead products capable of targeting angiogenesis in vivo and has been the first in the world to demonstrate that human monoclonal antibodies can efficiently and selectively target the tumor neo- vasculatur

Partner 4: NKI | Netherlands Cancer Institute: The Netherlands Cancer Institute was established in 1913. The Institute accommodates approximately 550 scientists and scientific support personnel, 53 medical specialists, 180 beds, an out-patient clinic that receives 183,000 patients each year, 5 operating theatres and 9 irradiation units. It is the only dedicated cancer centre in The Netherlands and maintains an important role as a national and international centre of scientific and clinical expertise, development and training.

Partner 5: UZH | University of Zurich: Founded in 1833, the University of Zurich is Switzerland's largest university, with a current enrolment of over 26,000 students. UZH is made up of seven faculties covering approximately 100 different subject areas. As a member of the "League of European Research Universities" (LERU), UZH is one of Europe's most prestigious research institutions.. Most of the ATECT will be carried out at the Institute of experimental Immunology at the University of Zurich. Read more about UZH, or the Institute of Experimental Immunology.



Key Concepts

We plan to develop strategies to engineer T-cells to be highly effective anti-cancer agents. A key part of this is to engineer them to be resistant to the hostile environment that tumour cells generate around them. A key part of this is to engineer the T-cell to secrete potent immune messages (called cytkones) into the tumour bed. Another strategy is to exploit the new vessels (neoangiogenesis) found in almost all tumours. We will engineer our therapeutic T-cells to attack and home to newly formed blood vessels indirectly targeting the tumours.

"Cancer Therapy is undergoing a revolution - from small molecules and proteins to heavily engineered T-cells"

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