Results:
CD4+CD28null - T cells in COPD Patients receiving a Lung-Transplantation
Chronic obstructive pulmonary disease (COPD)

Worldwide 600 million people suffer from COPD

200 000 – 300 000 people die every year in Europe of COPD

COPD is the third leading cause of death in the U.S. and the economic burden of COPD in the U.S. in 2007 was $42.6 billion in health care costs

Prediction show that in 2020 COPD will kill 6 million people every year and will become the third leading cause of death worldwide.
Background

T cell senescence and contraction of T cell repertoire diversity in patients with chronic obstructive pulmonary disease

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Several studies were able to show a strong correlation between tobacco abuse and the development of COPD but exact descriptions of the pathogenetic mechanisms remain vague.

Studies describing the role of CD8+CD28null lead to the hypothesis that a specific chronic inflammatory reaction of adaptive immune system is occurring in patients with COPD.

CD4+ T cells under chronic stress undergo multiple phenotypic and functional changes. One of the most described phenotypical changes is the loss of CD28. This was already described in diabetes mellitus, rheumatoid arthritis, multiple sclerosis, Wegener’s granulomatosis and Bekhterev syndrome.
Several of these functional features in CD4+CD28null T-cells are reminiscent of NK cells. Like NK cells, CD4+CD28null T cells are cytotoxic and can express NK cell receptors such as CD94 and CD158.
T cell senescence and contraction of T cell repertoire diversity in patients with chronic obstructive pulmonary disease.


Source
Department of Pulmonary Medicine, Medical University of Vienna, Vienna, Austria.

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AUC = 0.76
Patients with COPD show a profound change in the representation of functionally and phenotypically distinct subsets of CD4+ T cells.

The basic mechanisms causing replacement of other CD4+ T cells by CD4+CD28null clonotypes are incompletely understood.

Swhite blood cells derived from COPD GOLD I–II secreted augmented levels of IFN-g and TNF-a – cytokines that are known to increase macrophage and dendritic cell activity – compared with controls and severe COPD (GOLD III–IV), and causing tissue destruction
Methods

Flow cytometry – Blood
  CD4+ cells separated from lung tissue

Proliferation Assay - incubated with:
  Elastin soluble
  Elastin peptide
  Kollagen peptide
Lung Homogenization
Lung Homogenization

Cellcounter: 2-10 Mill/ml
Dynabeads separation

Cellcounter: 1 Mill/ml
CD28null Cells: Blood vs. Lung tissue
Proliferation Assay

Counts

- CD4+ + rad.PBMC + Elastin soluble
- CD4+ + rad.PBMC + elastin-peptid
- CD4+ + rad.PBMC + Kollagen peptid
- CD4+ + rad.PBMC + BSA

Counts
Psoriasin (S100A7) is a major \textit{Escherichia coli}-cidal factor of the female genital tract

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Empyema thoracis, defined as collection of pus in the pleural space, has been recognized since the time of Hippocrates and historically has been associated with high mortality.

The mortality rate from empyema thoracis remains high and it ranges between 6%–24%.

Pleural infection develops in 65,000 patients each year in the United States and the United Kingdom.

Bacteriology: Streptococcus pneumoniae, Staphylococcus aureus, Escherichia coli, Heamophilus influenza and Klesiella pneumoniae
Effect of Psoriasin on different bacteria

Antimicrobial psoriasin (S100A7) protects human skin from *Escherichia coli* infection

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