DANIEL MERTENS PRINCIPAL SCIENTISTS'S PROFILES

DANIEL MERTENS

PD Dr. rer. nat.,
Head of the cooperation unit „Mechanisms of Leukemogenesis“
DKFZ Heidelberg,
University Hospital Ulm

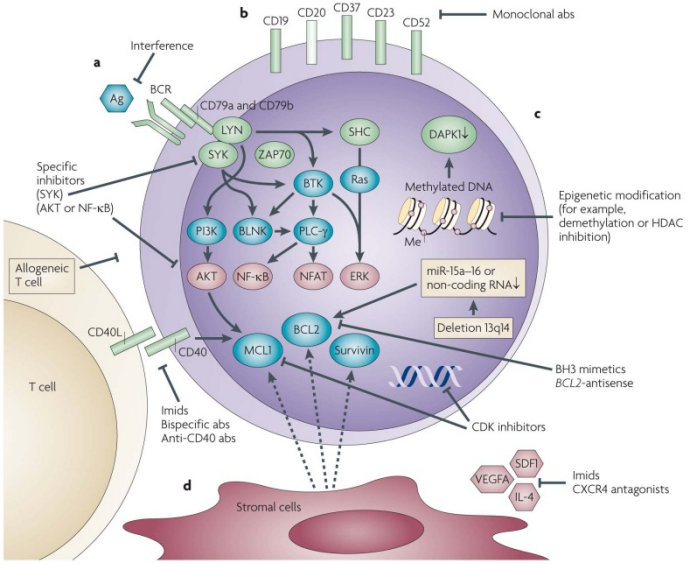
Daniel Mertens heads a junior group at the German Cancer Research Center (DKFZ) and a Max-Eder Group at the University Hospital Ulm.

RESEARCH AREAS

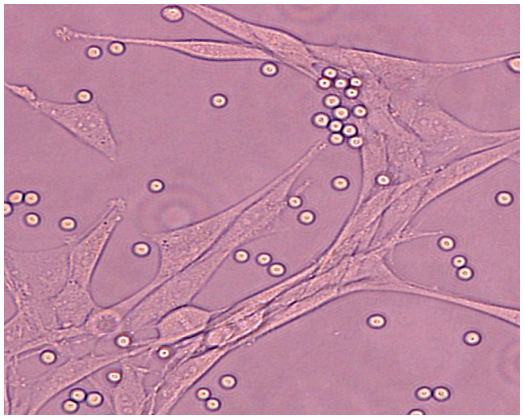
We are interested in the molecular mechanisms that cause leukemias and lymphomas. The scope of our research projects ranges from the elucidation and diagnostic applications of epigenetic oncomechanisms to the characteriziation of leukemia-specific signal transduction pathways in the malignant cells and the interaction with the non-malignant microenvironment.

Daniel Mertens has authored 53 publications that have been cited 1178 times (Thompson & Reuters, h-index 17). He was granted more than €3 Mio funding from third parties and currently coordinates two international research networks (cancerepisys.org and leukemia-resistance.de).

Modern oncology shifts in paradigm towards personalized medicine, where treatment is matched to the individual tumor. Such a targeted therapy requires understanding of the underlying pathomechanism of the disease entity: not only is the isolation of biomarkers needed in order to stratify single patients into prognostic subgroups, but also for the identification of central genes and pathways that can be targeted in therapies. The scientific focus of the cooperation unit is therefore to uncover the mechanisms of leukemogenesis and to translate this knowledge towards clinical application.



CLL pathogenic mechanisms and examples of targeted treatment options. Nature Reviews Cancer, (2010), **10**(1): 37-50.



Leukemic cells (small) need the support from non-malignant bystander cells. DIE ZEIT: Research in Baden Württemberg, 13.7.2013

The pathomechanism of malignant cells need also be viewed as an interplay of intracellular aberrations and their impact on the interaction of the tumor cells with their microenvironment. Inside the cell, genetic aberrations are complemented by epigenetic defects. These defects change the phenotype of the cell and its interaction with the surrounding non-malignant cells, the microenvironement. It is becoming increasingly clear how much the malignant cells form their microenvironmental niche that supports them. The dependency of the tumor cells on this niche for pro-survival support and protection makes this interaction a target that can be therapeutically exploited.