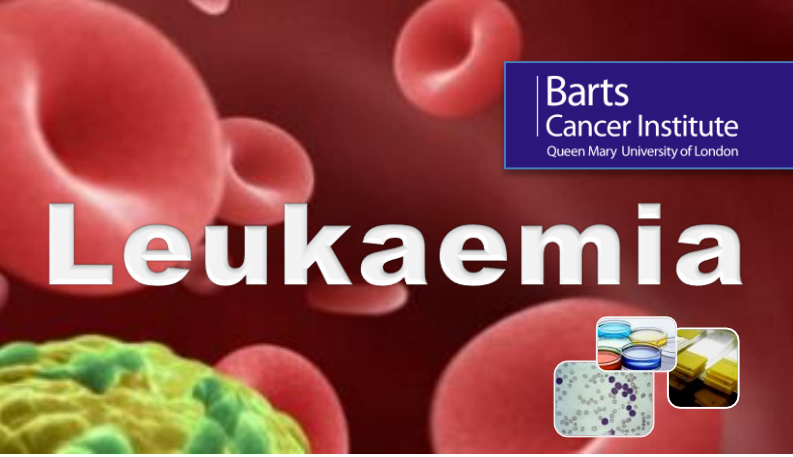
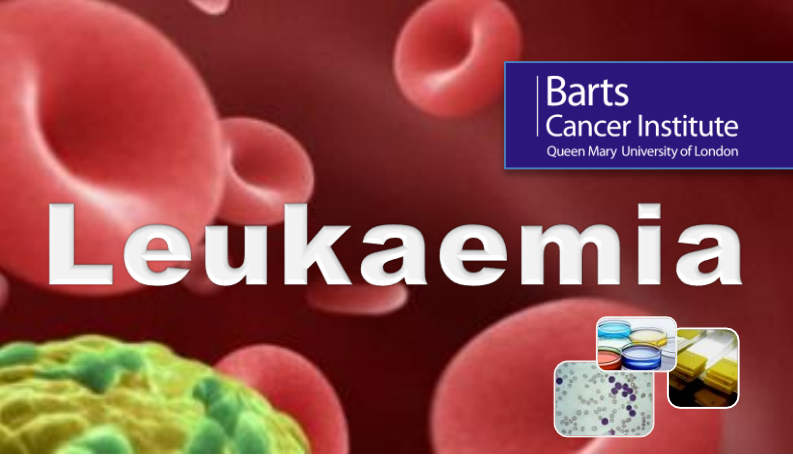


Why we focus on Leukaemia

Leukaemias can be acute or chronic and arise from the myeloid or lymphoid lineage. The treatment of chronic myeloid leukamia (CML) has been revolutionised by the use of agents such as Imatinib that target the molecular basis of the disease and this has become the paradigm to develop novel targeted agents for all leukaemias. Acute lymphoblastic leukaemia (ALL) is the most common cancer in children and is usually curable, however the outcome for adults is poor. Patients with ALL and high risk acute myeloid leukaemia (AML) are treated using intensive chemotherapy with stem cell transplant for those with an appropriate donor. However, many patients do not tolerate these therapies because of age and co-morbidity and the cure rate for those with poor risk disease is less than 10%. Therefore new approaches are desperately needed to improve patient outcome and to decrease the morbidity of treatment even in younger patients.

What we do

- We are characterising the nature of the leukaemic stem cells.
- We are investigating the interaction between normal and malignant stem cells. The focus of this work is to study how leukaemia out-competes normal haematopoietic stem cells to induce bone marrow failure.
- We are studying the molecular basis of familial leukaemia in order to understand the molecular evolution of disease.
- We are investigating the role and prognostic significance of sporadic mutations in disease, including analysis by whole genome sequencing approaches.
- We are studying the mechanisms whereby leukaemia induces immune suppression.
- We are investigating the nature of the graft versus leukaemia effect after allogeneic stem cell transplantation.



Key Publications

- Ramsay AG et al. Chronic lymphocytic leukemia cells induce defective LFA-1-directed T-cell motility by altering Rho GTPase signaling that is reversible with lenalidomide. Blood. 2013;121:2704-2714
- Riches, et al. T cells from CLL patients exhibit features of T-cell exhaustion but retain capacity for cytokine production. Blood. 2013;121:1612-1621.
- Taussig *et al* Leukemia-initiating cells from some AML patients with mutated nucleophosmin reside in the CD34(-) fraction. *Blood* 2010; 115: 1976-84.
- Le Dieu *et al.* Peripheral blood T cells in AML patients at diagnosis have abnormal phenotype and genotype and form defective immune synapses with AML blasts. *Blood* 2009; 114: 3909-16.

Who does the research

Prof. John Gribben	Allogeneic stem cell transplant, tumour microenvironment, immunotherapy
Dr. Jeff Davies	Immunotherapy, allogeneic stem cell transplantation
Dr. Jude Fitzgibbon	Molecular pathogenesis
Dr. Li Jia	Apoptosis in leukaemia
Dr. Rifca LeDieu	T cell response in leukaemia
Dr Pedro Cutillas	Proteomics
Dr Bela Wrench	Acute lymphomatic leukaemia
Dr Gabriella Ficz	Leukaemia stem cells

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- Cancer Research UK
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