XLP Symposium 2012

London 8th October

A conference for medical researchers on

X-Linked Lymphoproliferative Disease

DRAFT AGENDA

Introduction

0930–0940 David Hartley, The XLP Research Trust and Professor Bobby Gaspar, UCL Institute of Child Health, UK

Clinical Section

0940-1000 **Clinical manifestations of XLP1 and outcome**— Dr Claire Booth, Molecular Immunology Unit, UCL Institute of Child Health, United Kingdom

1000-1020 **X-linked dysgammaglobulin caused by hypomorphic *XIAP* mutation**, Dr Hirokazu Kanegane, Department of Pediatrics/University of Toyama, Japan

1020-1040 **Clinical and molecular comparison between CD27 and ITK deficiency—two fatal EBV Lymphoproliferative diseases in males and females**— Professor Ardt Borkhardt, Heinrich– Heince— University Dusseldorp, Germany

1040-1100 Break

XLP2/XIAP Pathogenesis

1100—1120 **Physiological activities of XIAP, the protein affected in type 2 XLP disease—**Dr Colin Duckett, University of Michigan, USA

1120- 1140 **Role of innate-like T cells and the NOD2 pathway in the pathophysiology of the X- Linked Lymphoproliferative syndrome type 2 (XLP2)** - Dr Sylvain Latour, INSERM 768, Hospital Necker– Enfants Malades, Paris, France

1140-1200 **An international survey of transplant outcomes for XIAP deficiency**—Dr Rebecca Marsh, Cincinnati Children's Hospital Medical Center, USA

1200-1310 Lunch

XLP1/SAP Pathogenesis

1310-1330 **Positive and negative signalling through SLAM receptors regulate synapse organisation and set thresholds of CD8-mediated cytolysis**—Dr Jennifer Cannons, National Human Genome Research Project, National Institute of Health, Bethesda, USA

1330-1350 **Expansion of somatically reverted memory CD8* T cells in XLP patients due to selective pressure from Epstein Barr virus**—Professor Stuart Tangye, Garvan Institute of Medical Research, Sydney, Australia

1350-1410 **Molecular function of SAP and NTB-A in promoting restimulation—induced cell death of activated T cells**—Dr Andrew Snow, Uniformed Services University of the Health Sciences, Bethesda, USA

1410-1430 Break

1430-1450 **Conditional elimination of the adaptor molecule SAP reveals an essential role in invariant NKT cell cytotoxicity *in vitro* and *in vivo***— Dr Kim Nichols, The Children's Hospital of Philadelphia, USA

1450-1510 **SAP-Mediated inhibition of diacylglycerol kinase alpha regulates TCR-induced diacylglycerol signalling**—Professor Gianluca Baldanzi, Universita del Piemonte Orientale Amedeo Avogadro, Novara, Italy

Gene Therapy

1510-1530 **Lentiviral vector mediated gene therapy for X-linked Lymphoproliferative disease restores humoral and cellular functions**—Dr Christine Rivat, UCL Institute of Child Health, UK

Summary

1530–1600 Professor Bobby Gaspar, UCL Institute of Child Health, London, UK