

Meeting abstract

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Dissecting the molecular pathogenesis of Burkitt lymphoma

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Burkitt lymphoma (BL) is a high grade B cell malignancy (Non-Hodgkin Lymphoma (NHL)) derived from germinal center B cells (GCB) that harbours a chromosomal translocation juxtaposing the protooncogene MYC next to the regulatory elements of one of the immunoglobulin loci. However, the precise contribution of Myc to the pathogenesis of this tumour is poorly understood. Based on the definition of a distinguishing gene expression signature for the molecular BL (mBL) with Myc as one hall-marking signature gene we are interested in getting answers to the questions (i) what are the target genes of Myc in primary human GCB cells and (ii) what is the functional significance of signature genes identified?

We describe a non-viral vector based approach (Vockerodt et al. 2008) to express Myc in primary human GCB cells. Comparative gene expression profiling was performed accompanied by qRT-PCR. In addition elucidation of the function of selected signature genes in BL is accomplished. In a representative cell line with a mBL signature RNAi directed inhibition of elements of the CD40 signaling cascade was conducted. After activating this particular signaling cascade in a BL cell line we analysed respective gene expression profiles of IKK α , IKK β , TRAF2, TRAF6, Jak3, BCL-3 and p38 deficient cells. Based on these different RNAi-mediated GE-profiles we reconstruct the topology of the respective signaling pathway by using the nested effects bioinformatic model, which has been described recently (Markowitz et al. 2005).

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