LYMPHOMA PATHOLOGY

P. Brousset, Toulouse (France), et al.

**DIAGNOSIS AND CLASSIFICATION ASSISTANCE FROM LYMPHOMA MICROSCOPIC IMAGES USING DEEP LEARNING**

Authors Conclusion from the abstract: These findings strongly suggest that DL models, even on very simple CNNs, can assist pathologists in the diagnosis and subclassification of lymphoma as recently shown is subsets of lung cancers.³

S.A. Pileri, Milan (Italy), et al.

**INTEGRATING TUMOR- AND MICROENVIRONMENT- REFLECTING GENES IN A UNIQUE AND ROUTINE- APPLICABLE ASSAY FOR ACCURATE RISK PREDICTION IN DLBCL.**

Authors Conclusion from the abstract: This study supports the idea that DLBCL heterogeneity involves both tumor and TME, resulting in diverse transcriptional subtypes with distinct outcomes and, putatively, diverse biology. Our integrative analysis prompts the development of a new survival categorization outperforming current prognostic risk-assessment. Moreover, the applicability of a unique Nanostring-based assay to routine biopsies may facilitate the stratification of patients at diagnosis and their inclusion in future trials exploring novel therapeutic approaches.

S. Balasubramanian, San Diego CA (USA), et al.

**CONCORDANCE BETWEEN IMMUNOHISTOCHEMISTRY AND GENE EXPRESSION PROFILING SUBTYPING FOR DIFFUSE LARGE B-CELL LYMPHOMA IN THE PHASE 3 PHOENIX TRIAL**

Authors Conclusion from the abstract: The overall concordance between non-GCB by Hans-based IHC and GEP using the EdgeSeq DLBCL COO Assay was 74-77% in the ITT population and age-related subgroups, even with centralized testing. Although only 75.9% of enrolled pts were the ABC subtype, the addition of ibr improved outcomes in pts.
**S. Araf, London (UK), et al.**

**LONGITUDINAL ANALYSES OF DIAGNOSTIC-RELAPSE BIOPSIES OF DIFFUSE LARGE B CELL LYMPHOMA SUGGEST THAT RELAPSE IS MEDIATED BY DISTINCT MECHANISMS IN ABC AND GCB LYMPHOMA**

Authors Conclusion from the abstract: The nature of the biological mechanisms responsible for DLBCL relapse has remained fairly elusive that may be inherent to the diagnostic tumor or acquired/enriched at disease relapse. Gene expression profiling of a series of DLBCL tumor pairs, resolved changes in gene expression that support distinct mechanisms of lymphoma relapse, based on a patient's COO, that parallel changes in the overall T-cell composition of the tumor microenvironment.

**S. Barrans, Leeds (UK), et al.**

**DEFINING BURKIT-LIKE LYMPHOMA WITH 11Q ABERRATION IN A SPECIALISED UK HAEMATOPATHOLOGY DIAGNOSTIC SERVICE**

Authors Conclusion from the abstract: These findings suggest that not all BLL-11q patients require high dose therapy, supporting the hypothesis that their biological features are more similar to DLBCL than BL. Further investigation into the molecular landscape of these patients is underway. Taken with data from the other molecular subgroups (DHL/MHG) this will direct standardised treatment decision making in routine practice, and help define when alternative therapies are warranted.

**L. Quintanilla-Martinez de Fend, Tübingen (Germany), et al.**

**GENOME WIDE-ANALYSIS OF T(14;18)-NEGATIVE FOLLICULAR LYMPHOMA**

Authors Conclusion from the abstract: