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| Clinical Utility of Molecular Testing in  Follicular Lymphoma  Diagnostic Utility  The canonical molecular abnormality in classical follicular lymphoma (FL) is t(14;18)(q32;q21) resulting in the *IGH::BCL2* fusion gene and overexpression of *BCL2*1.  Four variants of FL are recognised in the WHO revised 4th edition classification: *in situ* follicular neoplasia (ISFN), duodenal-type FL, testicular FL and the diffuse variant of FL. In addition, paediatric-type FL is a separate entity1. ISFN and duodenal-type FL are characterised by t(14;18)(q32;q21) and share genetic features with classic FL2,3. *BCL2* translocations are not typically observed in the diffuse variant of FL or testicular FL1.  Del(1p) (which contains *TNFRSF14)*, acquired copy neutral loss of heterozygosity and mutations in *TNFRSF14* are commonly seen across all variants of FL including paediatric-type FL1. These findings are not detected by this assay.  *BCL2* aberrant somatic hypermutation is frequently observed in FL and germinal centre type diffuse large B cell lymphoma4,5.  Mutations in chromatin-modifying genes are recurrently observed in classic FL and typically occur as early events. These include *KMT2D* (~80%), *CREBBP* (~65%), *EZH2* (~25%), *ARID1A* (~15%) and *EP300* (~10%) mutations6-8.  Other recurrently mutated genes in classic FL include *STAT6*, *FOXO1*, *RRAGC*, *TNFAIP3*, *IRF8* and *CARD119-11*.  The finding of 1p36/*TNFRSF14* abnormalities along with *STAT6*, *CREBBP* and/or *KMT2D* mutations supports a diagnosis of a t(14;18)-negative FL12,13.  Primary WHO diagnostic criteria for paediatric-type FL include the absence of *BCL2*, *BCL6*, *IRF4* and aberrant immunoglobulin gene rearrangements as well as not having *BCL2* amplification1. Additionally, mutations in epigenetic modifiers frequently observed in classic FL are typically absent such as *KMT2D, CREBBP* and *EZH2*. Instead *MAP2K1* mutations are identified in approximately 50% of cases14. The *IRF8* Lys66Arg has been described as a recurrent mutation in paediatric type FL14,15.  Prognostic utility  The number of additional cytogenetic abnormalities, mutational burden and degree of aberrant somatic hypermutation (aSHM) increases at histological transformation1,16.  Histological transformation is associated with biallelic loss of *CDKN2A*/*CDKN2B* and/or *TP53*, *MYC* translocation and mutations in NFKB signalling (*MYD88, TNFAIP3)*1,6,16,17.  Biomarkers of response to therapy  Activating *EZH2* mutations are the target of the EZH2 inhibitor class of therapeutics18.  References  **1.** Swerdlow S, et al. WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues (revised 4th edition). Lyon: IARC; 2017. **2.** Schmidt J, et al. 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