In this issue

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In this issue the miniseries on the pathology of follicular lymphoma is rounded up. The third paper in the series (DOI [10.1007/s00428-015-1840-6](https://doi.org/10.1007/s00428-015-1840-6)) addresses mantle cell lymphoma (MCL), which is characterized in most cases by the t(11;14) translocation and overexpression of cyclin D1. Patients with MCL usually present in an advanced stage and rapidly progress and historically it has been considered an aggressive disease. Nonetheless, it has been recognized more recently that some MCL have a long, clinically silent or remarkably indolent disease course and do not require immediate treatment and occasionally no treatment at all. Research results have provided further insight into the role of additional genetic alterations in the diverse pathogenesis and heterogeneous behavior of MCL. This paper addresses the diagnostic problems that variant morphologies, immunophenotypes and especially cyclin D1 negative MCL poses in daily practice. It presents the morphological and clinical characteristics of the leukemic non-nodal MCL variant and highlights the importance of and difficulties in interpretation of FISH analyses.

The final paper in the series (DOI [10.1007/s00428-015-1858-9](https://doi.org/10.1007/s00428-015-1858-9)) addresses the issue of plasmacytic differentiation in small B-cell lymphoma (SBL), which may be found in any SBL but the frequency of which varies according to further morphological characteristics. For a diagnosis of lymphoplasmacytic lymphoma

(LPL) some degree of plasmacytic differentiation is required. Plasmacytic differentiation is quite frequent in mantle zone lymphoma (MZL), but rather rare in follicular lymphoma, chronic lymphocytic leukemia and in particular in MCL. The differential diagnosis between these entities may be challenging and one must rely upon the morphological appearance of the small B-cell component and information on genetic abnormalities. The discovery of the MYD88 L265P mutation in a majority of LPL cases, even though not specific, has helped to better define the spectrum of morphological and phenotypic features of LPL, which is broader than previously thought. In LPL architectural destruction and follicular colonization may be more important than previously recognized. Most typical cases have a very monomorphic cytological appearance. Cases with polymorphic cytology most probably correspond to MZL. MCL used to be considered as lacking in plasmacytic differentiation but this is now recognized as being present in the subset of SOX11 negative cases.

But there is more in this issue. La Rosa et al. (DOI [10.1007/s00428-015-1882-9](https://doi.org/10.1007/s00428-015-1882-9)) report on *TP53* alterations in acinar cell carcinoma of the pancreas (ACC), a controversial issue as contradictory data have been published. The group collected a large number of primary and metastatic ACC samples, and looked for mutations and promoter methylation in the *TP53* gene, allelic loss by FISH and p53 protein expression by immunohistochemistry. They found *TP53* mutations more often in metastases of ACC than in primary tumors but when present in the primary the metastases had the same mutation. *TP53* allelic loss occurred in half of the cases while promoter methylation was rare. These molecular alterations were reflected in p53 immunohistochemical staining patterns. Cases with mutation of one allele and loss of the other showed worse survival. The fact that *TP53* mutations were more frequently found in metastases than in primary ACC suggests that *TP53* might be involved in tumor progression rather than in the early

The first two paragraphs introducing the papers on pathology of lymphoma were contributed by Leticia Quintanilla-Martinez, Steven L Swerdlow and Elias Campo.

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steps of tumorigenesis. The cover image is from this paper and illustrates a *TP53* mutated sequence in one of their cases.

Warth et al (DOI [10.1007/s00428-015-1892-7](https://doi.org/10.1007/s00428-015-1892-7)) report on mismatch repair competence as reflected in microsatellite instability (MSI-H) in a large series of pulmonary adenocarcinomas (ADC). They used a trusted mononucleotide marker panel to identify MSI-H cases

and confirmed loss of expression of mismatch repair genes by immunohistochemistry. MSI-H was rare (less than 1 % of cases), not accompanied by evidence of Lynch syndrome and associated with early stage and a history of smoking. They conclude that in spite of its rarity MSI-H ADC is a clinically relevant subgroup of non-small cell lung cancer.