

## In this issue

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This month's issue opens with an amazing new development: microscopy without a microscope! That is, without a microscope as we know it. Zhang et al. (DOI [10.1007/s00428-015-1782-z](https://doi.org/10.1007/s00428-015-1782-z)) reasoned that the conventional light microscope suffers from bulky size, lack of portability, and relatively high cost, which limit its use notably in low resource situations. As a solution, they developed a lens-free on-chip imaging platform. The image quality is quite comparable with that of a conventional microscope or a virtual slide prepared using a bulky scanner. Diagnostic performance (albeit on a limited set of samples) is equal to that of conventional microscopy. The lens-free computational microscopy platform holds high promise for applications in pathology, especially where resources are limited.

The cover image illustrates the setup of this lens-free imaging platform.

Lee and collaborators (DOI [10.007/s00428-015-1753-4](https://doi.org/10.007/s00428-015-1753-4)) delved into the details of involvement of the Wnt pathway in gastric neoplasia with a fundic gland phenotype, of which the chief cell predominant type is a recently identified new entity. The oncogenesis of the latter is still under investigation, notably as regards the existence of premalignant lesions and the transition from premalignant to invasive malignant lesions. They assessed Wnt pathway genes *CTNNB1*, *APC*, *AXIN*, and *PPP2R1A* for mutations and used  $\beta$ -catenin immunohistochemistry as a read-out for activation of the Wnt pathway. In a variety of the studied lesions, mutations of the different Wnt associated genes were found. These were not always accompanied by nuclear  $\beta$ -catenin staining, suggesting that not all Wnt pathway-associated gene mutations activate the pathway. The results also suggest a dysplasia-carcinoma sequence in chief cell predominant lesions, as in the

dysplastic chief cell lesions nuclear  $\beta$ -catenin staining was not found whereas this did occur in gastric cancer of the chief cell type.

The paper by Uguen et al. (DOI [10.007/s00428-015-1763-2](https://doi.org/10.007/s00428-015-1763-2)) addresses the issue of mitosis counting in malignancy grading. Mitotic counts on hematoxylin/eosin-stained sections have a reproducibility problem, and given their role in recurrence risk assessment in a variety of neoplastic lesions, including in gastrointestinal stromal tumors (GISTs), methodological improvement would be welcome. The group counted mitoses in GIST using immunohistochemical staining for phospho-histone H3, a protein exclusively present on condensed chromatin during mitosis, and compared this with immunostaining for Ki-67 and conventional histological staining. Not surprisingly, mitotic counts on immunohistochemically stained slides (phospho-histone H3 and Ki-67) went faster and had better intra- and interobserver reproducibility than those on conventionally stained slides. Phospho-histone H3 results were slightly higher than those on conventional stains but about one third of those for Ki-67, which stands to reason for a protein expressed during mitosis only. The authors propose to shift to immunohistochemically supported mitotic counting, which would require re-assessment of cutoff points for stratification of risk of recurrence.

We tend towards immediate rejection for case reports, unless such a report tells a real story with a message. The paper by Van der Horst et al. (DOI [10.007/s00428-015-1779-7](https://doi.org/10.007/s00428-015-1779-7)) is an example of a case report with an interesting message. The patient they describe had an inflamed eruption centered on the skin of his forehead and scalp, a biopsy of which showed primary cutaneous follicle center lymphoma. Local radical radiotherapy was successfully applied, but when the lesion relapsed, submandibular lymphadenopathy was found, which appeared to be an EBV-positive diffuse large B-cell lymphoma. The cutaneous lesion then also tested positive for EBV. The authors speculate that this EBV-positive lymphoma might have arisen as a consequence of age-related immune senescence. Their observation further expands the spectrum of age-related EBV associated B-cell proliferative lesions.

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