In this issue

Fred T. Bosman¹

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An at first glance a simple issue - deeper levels or not and if yes how many - is addressed by Schick et al. (DOI [10.1007/s00428-015-1866-9](https://doi.org/10.1007/s00428-015-1866-9)). They studied colonic biopsies from what the endoscopist had submitted as polyp but of which the first section(s) did not reveal any lesion that could explain the endoscopic impression. To this end they cut three to ten levels of consecutive 'negative' biopsies at 50 µm intervals. In about one out of every four biopsies a lesion was identified in the levels, 75 % of which a tubular adenoma. About half of these were identified already at the second level. The authors conclude that when initial sections reveal no lesion while endoscopically a lesion was found, deeper levels are necessary. We all know this and it stands to reason but the paper documents this nicely.

Du et al. (DOI [10.1007/s00428-015-1854-0](https://doi.org/10.1007/s00428-015-1854-0)) studied secretory immunoglobulin A (SIgA) in chronic obstructive pulmonary disease (COPD), a condition associated with deficiency of SIgA on the airway surface but not in sputum or bronchoalveolar lavage (BAL) fluid. To explain this discrepancy, the authors studied BAL fluid and lung tissue from patients with COPD and control subjects. Immunohistochemical analysis of large and small airways revealed less IgA on the airway surface but accumulation of IgA within luminal mucus plugs and ample immunoreactive protein in the submucosal glands. These observations led the authors to conclude that in

COPD increased SIgA production by hyperplastic submucosal glands does not compensate for reduced SIgA transcytosis by airway epithelial cells. As small airway SIgA deficiency is associated with COPD progression, targeting of this mechanism might provide a new therapeutic approach.

Geles et al. (DOI [10.1007/s00428-015-1852-2](https://doi.org/10.1007/s00428-015-1852-2)) report on pulmonary adenocarcinomas, notably on the question whether or not the mucinous type is more aggressive than the non-mucinous type. They also studied molecular marker patterns in association with prognosis. The bottom-line of the paper is that when corrected for T and N stage overall survival for mucinous adenocarcinomas is not different from that of non-mucinous adenocarcinomas. Goblet cell and colloid variants of mucinous adenocarcinoma often had a *KRAS* mutation but this was not associated with prognosis. Loss of p16 expression, however, correlated with worse outcome. Most important predictors of survival, therefore, are TNM stage and p16 loss.

Finally, Yilmaz et al. (DOI [10.1007/s00428-015-1851-3](https://doi.org/10.1007/s00428-015-1851-3)) looked into the mutational profile of malignant blue nevi. While *BRAF*, *NRAS* and *KIT* mutations are well documented in malignant melanoma they are rare in blue nevi, in which recently activating mutations in *GNAQ/GNA11* have been reported. The mutational landscape of malignant blue nevi remains unclear. The authors found *GNAQ* exon 5 mutations to be most frequent in 'blue' melanocytic lesions. A small proportion of malignant blue nevi had *BRAF* V600E mutations, in common with melanoma, and an even smaller proportion *GNA11* exon 5 mutations. In contrast, these were never found in the benign 'blue' melanocytic lesions. Further studies will be necessary to elucidate molecular pathogenesis of this rare lesion.

The cover image is a mechanism oriented schematic from the Nesi et al. (DOI [10.1007/s00428-015-1820-x](https://doi.org/10.1007/s00428-015-1820-x)) review paper on the role of chronic inflammation in bladder cancer.

✉ Fred T. Bosman
fred.bosman@citycable.ch

¹ Institute of Pathology, University Medical Center (CHUV) of Lausanne, Lausanne, Switzerland