

IN BRIEF

GENETICS

Gastric adenocarcinoma and proximal polyposis of the stomach (GAPPS): a new autosomal dominant syndrome

Worthley, D. L. *et al. Gut* doi:10.1136/gutjnl-2011-300348

The clinical and pathological features of a unique autosomal dominant gastric polyposis syndrome have been reported by Worthley and colleagues. Gastric adenocarcinoma and proximal polyposis of the stomach (GAPPS) has a histological appearance of predominantly dysplastic fundic gland polyposis, is limited exclusively to the gastric body and fundus, and confers a significant risk of gastric adenocarcinoma.

THERAPY

Haemoglobin decreases in NSAID users over time: an analysis of two large outcome trials

Goldstein, J. L. *et al. Aliment. Pharmacol. Ther.* doi:10.1111/j.1365-2036.2011.04790x

Analysis of data from the double-blind, prospective CLASS and CONDOR trials has confirmed that the NSAIDs celecoxib and diclofenac are associated with a clinically meaningful decrease in hemoglobin. A hemoglobin decrease of ≥ 2 g/dl was seen in the CLASS and CONDOR trials after 6 months in 1.9% and 2.0% of patients receiving celecoxib and 3.3% and 5.7% of patients receiving diclofenac, respectively.

MOTILITY

Pharmacogenetic trial of a cannabinoid agonist shows reduced fasting colonic motility in patients with non-constipated irritable bowel syndrome

Wong, B. S. *et al. Gastroenterology* doi:10.1053/j.gastro.2011.07.036

This study shows that dronabinol decreases the fasting proximal and distal left colonic motility index and increases colonic compliance in patients with IBS, when compared with placebo. However, dronabinol had its greatest effect in those with diarrhea-predominant or alternating IBS. The findings of this study also indicate that genetic variants in *FAAH* and *CNR1* may help determine the effect of dronabinol on colonic motility.

VIRAL HEPATITIS

Nucleotide change of codon 182 in the surface gene of hepatitis B virus genotype C leading to truncated surface protein is associated with progression of liver diseases

Lee, S.-A. *et al. J. Hepatol.* doi:10.1016/j.hp.2011.06.028

Reports suggest that mutations in the HBV surface gene contribute to the development of hepatocellular carcinoma (HCC) in patients infected with HBV genotype 3. Now, Lee *et al.* have shown that nucleotide change sW182* (which introduces a premature stop at codon 182) is significantly more prevalent in patients with HCC and liver cirrhosis than it is in HBV carriers and those with chronic hepatitis.