

IN BRIEF

PANCREATIC CANCER

Three pancreatic cancer susceptibility loci have been identified by Petersen *et al.* in their genome-wide association study. The researchers screened the genomes of 3,851 people with pancreatic cancer and 3,934 unaffected individuals as controls. After adjusting for factors such as age, ancestry and sex, the researchers identified eight single nucleotide polymorphisms that mapped to three genetic loci on chromosomes 13q22.1, 1q32.1 and 5p15.33. More than half of the polymorphisms mapped to *NR5A2* on 1q32.1, a single polymorphism mapped to a locus on 5p15.33 which is associated with multiple cancers, whilst the remainder mapped to a nongenic region on 13q22.1.

Original article Petersen, G. M. *et al.* A genome-wide association study identifies pancreatic cancer susceptibility loci on chromosomes 13q22.1, 1q32.1 and 5p15.33. *Nat. Genet.* **42**, 224–228 (2010)

CROHN'S DISEASE

Increased levels of interferon (IFN)- γ in the gut mucosa are associated with the symptoms of Crohn's disease. Reinisch and colleagues investigated the safety and efficacy of a humanized anti-IFN- γ antibody, fontolizumab, in patients with Crohn's disease. The researchers found that disease response rates after 29 days were comparable in all treatment groups. However, at later time points significantly more patients treated with a 1 mg/kg intravenous and up to three subsequent subcutaneous doses of fontolizumab attained clinical remission than control patients. The investigators demonstrated that fontolizumab was well tolerated and significantly decreased C-reactive protein levels in patients with Crohn's disease.

Original article Reinisch, W. *et al.* Fontolizumab in moderate to severe Crohn's disease: a phase 2, randomized, double-blind, placebo-controlled, multiple dose study. *Inflamm. Bowel Dis.* **16**, 233–242 (2010)

OBESITY

Extensive genome-wide association studies linked many single nucleotide polymorphisms with obesity, but only a small number of these contribute to the disease's heritability. Walters *et al.* investigated whether rare copy-number variants contribute to the development and heritability of obesity by screening the genomes of 31 people with an extreme obesity phenotype and mental retardation. The researchers identified a 593 kb deletion on chromosome 16p11.2 that correlated with a highly penetrant form of obesity in these individuals. The investigators then developed an algorithm to search data from previous genome studies for obesity genes and identified 19 similar deletions. The deletions were not present in healthy nonobese individuals and accounted for 0.7% of people who were morbidly obese.

Original article Walters, R. G. *et al.* A new highly penetrant form of obesity due to deletions on chromosome 16p11.2. *Nature* **463**, 671–675 (2010)