



## Referenties bij MAGMA 2-2011

### Klinische implicaties genetisch onderzoek, pag. 47–48

1. International Human Genome Sequencing Consortium *Nature* 409, 860-921. *Initial sequencing and analysis of the human genome*.
2. Fishel R., Lescoe M.K., Rao M.R., Copeland N.G., Jenkins N.A., Garber J., Kane M., Kolodner R. *Cell*. 1993; Dec 3;75(5):1027–38 The human mutator gene homolog MSH2 and its association with hereditary nonpolyposis colon cancer.
3. Neil Risch and Kathleen Merikangas. *Science* 1996; 13; September: Vol. 273 no. 5281 pp. 1516–1517. The Future of Genetic Studies of Complex Human Diseases.
4. Romanos J., Wijmenga C. Predicting Susceptibility to Celiac Disease by Genetic Risk Profiling. *Annals of Gastroenterology & Hepatology* 2010;1:11–18.
5. Dubois P.C. et al. Multiple common variants for celiac disease influencing immune gene expression. *Nat Genet*. 2010;42:295–302.
6. Franke A. et al. Genome-wide meta-analysis increases to 71 the number of confirmed Crohn's disease susceptibility loci. *Nat Genet*. 2010;42:1118–1125.
7. Anderson C.A. et al. Meta-analysis identifies 29 additional ulcerative colitis risk loci, increasing the number of confirmed associations to 47. *Nat Genet*. 2011;43:246–252.
8. Rioux J.D. et al. Genome-wide association study identifies new susceptibility loci for Crohn disease and implicates autophagy in disease pathogenesis. *Nat Genet*. 2007;39:596–604.
9. Festen E.A. et al. A meta-analysis of genome-wide association scans identifies IL18RAP, PTPN2, TAGAP, and PUS10 as shared risk loci for Crohn's disease and celiac disease. *PLoS Genet*. 2011;7:e1001283.
10. Hugot J.P., Chamaillard M., Zouali H. et al. Association of NOD2 leucine-rich repeat variants with susceptibility to Crohn's disease. *Nature* 2001;411:599–603.
11. Frank D.N., St Amand A.L., Feldman R.A., Boedeker E.C., Harpaz N., Pace N.R. Molecular-phylogenetic characterization of microbial community imbalances in human inflammatory bowel diseases. *Proc Natl Acad Sci USA* 2007;104:13780–13785
12. Daly A.K. et al. HLA-B\*5701 genotype is a major determinant of drug-induced liver injury due to flucloxacillin. *Nat. Genet*. 2009;41:816–819.
13. SEARCH Collaborative Group. SLCO1B1 variants and statin-induced myopathy – a genomewide study. *N Engl J Med*. 2008;359:789–799.

### Hepatitis-A-vaccinatie voor personeel MDL-endoscopie zeer gewenst, pag. 45

1. Man, dr. R.A. de, dr. H. van Driel, dr. A. Vossen, prof. dr. J.H. Richardus. Determinanten van de aanwezigheid van Hepatitis-A-antistoffen onder verpleegkundigen werkzaam op een endoscopieafdeling; implicaties voor HAV-vaccinatiebeleid in de zorg. *Ned Tijdschrift Medische Microbiologie* 2011;19(1)10-13.



**Casuïstiek – Hyper-Inflammatory Bowel Disease, pag. 59**

1. Meer J.W.M. van der, J.M. Vossen, J. Radl, J.A. van Nieuwkoop, C.J. Meyer, S. Lobatto, R. van Furth. Hyperimmunoglobulinaemia D and periodic fever: a new syndrome. *Lancet* 1984;1:1087-1090.
2. Hilst J.C.H. van der, E.J. Bodar, K.S. Barron, J. Frenkel, J.P.H. Drenth, J.W.M. van der Meer, A. Simon, and the International HIDS Study Group. Long-Term Follow-Up, Clinical Features, and Quality of Life in a Series of 103 Patients with Hyperimmunoglobulinemia D Syndrome. *Medicine (Baltimore)* 2008 nov;87(6):301-10.
3. Drenth, J.P.H., J.W. van der Meer, I. Kushner. Unstimulated peripheral blood mononuclear cells from patients with the hyper-IgD syndrome produce cytokines capable of potent induction of C-reactive protein and serum amyloid A in Hep3B cells. *J Immunol.* 1996; 157:400-404.
4. Drenth J.P.H., L. Cuisset, G. Grateau, C. Vasseur, S.D. van de Velde-Visser, J.G. de Jong, J.S. Beckmann, J.W.M. van der Meer, M. Delpech. Mutations in the gene encoding mevalonate kinase cause hyper-IgD and periodic fever syndrome. International Hyper-IgD Study Group. *Nat Genet.* 1999;22:178-181.