effects on disease phenotype may lead to the development of a gene-based classification system which assists in differentiating UC from colonic CD.

**Background & Aim:** IL10-deficient mice serve as a model for inflammatory bowel disease and in several autoimmune/inflammatory diseases. The region comprises the bacterial flora in the development of ulcerative colitis.

**Methods:** The association of the TNF-857C/T polymorphism with 1BD, CD and UC was examined in a cohort of 163 patients with a well-established diagnosis of UC and 136 healthy hospital workers were genotyped after informed consent for the TLR4 variant Asp299Gly and for the three CARD5 variants (Arg702Trp, Gly908Arg and Leu1007fs) using Taqman PCR and PCR-RFLPs, respectively. Clinical charts were reviewed for the following phenotypes: localisation (rectosigmoiditis, left colitis and pancolitis), surgery, extra-intestinal manifestations, familial disease, pANCA and smoking at diagnosis. Groups were compared using Chi-square test. Results: There were 31/163 (19.02%) UC patients carrying the TLR4 variant and 4/136 UC patients carried at least one CARD5 variant (p = 0.011). The mutated allele frequency was at least 10% higher in UC than in the control population (12.1% vs. 5.2%, p = 0.015). There were 2 UC patients homozygous for the mutant allele, compared to 1 control. Univariate analysis failed to show any significant association between TLR4 variant and UC susceptibility in this population. Conclusions: A positive association is observed between the TLR4 polymorphism and UC. The frequency of this mutant allele was twice as high in UC patients compared to healthy controls. This association may further help to understand the role of the bacterial flora in the development of ulcerative colitis.

**Candidate Genes for Experimental IBD: Microarray Analysis in Combination with Direct Association Tests (QTL) Mapping Data**

**Background:** The TNF-857T allele may be protective against early onset, arthritis and right colonic disease in both UC and CD. There was no correlation with CARD15 genotype. Results: The TNF-857T allele was over represented relative to controls in both German cases and German families for both BD (p = 0.024) and CD (p = 0.002). Allele frequencies were identical between cases and controls in a chronic acute CD cohort lacking certain complications (e.g. fistulization, stenosis, arthritis). No significance for UC was observed (p = 0.666). Identical results were obtained using the TDT test, although the TNF-857T allele was overrepresented in all cases. Logistic regression revealed that the TNF-857C/T polymorphism is independent of CARD15. Unless association is examined at the level of detailed clinical traits then studies may be conflicting because patient collections vary at this level. The TNF-857T allele may be protective against early onset, arthritis and smoking related IBD and may protect against persistent inflammatory conditions in general.