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Coeliac disease (CD) has an autoimmune component in genetically predisposed individuals triggered by environmental factor (gluten). The disease manifests in partial or total villous destruction of the small intestine with malabsorption and malnutrition. The main environmental triggering factor is a transglutaminated peptide within the gliadin component of gluten, found in wheat. CD has an established HLA component responsible to 35% of the genetic predisposition, rest being in the non-HLA region.

100 coeliac patients were recruited, having predominance of females over male coeliac patients (3:1, χ^2 = 25, p <0.001). The mean age at diagnosis for the whole group was 34 years (males 32 years, females 34 years, t = -0.65, N.S.). The predominant presenting symptoms were gastrointestinal related. A higher proportion of males reported a positive family history as compared to females (χ^2 = 5.44, p = <0.02).

Two polymorphisms found within the CTLA*4* gene were studied amongst a sample of coeliac patients and cord blood DNA samples (n = 187) that acted as the control group. Polymorphisms within the CTLA*4* gene have been associated with other autoimmune conditions and the gene plays a very important role in immunoregulatory function. The coeliac individuals and cord blood samples were genotyped for the -318 C/T and +49 A/G SNPs. No association of the single polymorphisms or the combined haplotypes with the coeliac condition was apparent amongst the coeliac patients under study. The -318 C allele and the +49 A allele were in linkage disequilibrium amongst the cord blood samples.