Table 2. Frequencies of HLA-DRB1 and -DQB1 alleles in H. pylori-positive patients and controls

HLA-DRB1	f patients N = 48	f controls N = 286	HLA-DQB1	f patients N = 48	f controls N = 286
***0101 - 6**	**0.1500**	0.0731	***0201-2**	0.1666	0.2098
***1501 - 8**	**0.1500**	0.1231	***0301**	0.1666	**0.2448**
***1601 - 8**	0.0333	0.0538	***0302**	0.1666	0.0559
***0301**	0.0666	0.0962	***0303**	0.0333	0.0419
***0302**	0.0166	0.0000	***0401**	0.0000	0.0070
***0401 - 32**	0.1333	0.1116	***0402**	0.0333	0.0385
***1101 - 35**	0.1000	**0.2038**	***0501**	0.1666	0.1119
***1201 - 6**	0.0000	0.0153	***0502**	0.0333	0.0559
***1301 - 2**	0.0666	0.0654	***0503**	0.0666	0.0280
***1303 - 4**	0.0000	0.0153	***0601**	0.0000	0.0035
***1305**	0.0166	0.0000	***0602**	0.1500	0.1014
***1306**	0.0166	0.0000	***0603**	0.0500	0.0839
***1401,- 4,-5,-7,-8**	0.0666	0.0231	***0604**	0.0666	0.0175
***1402,- 6,-9**	0.0000	0.0038			
***0701 - 4**	0.1166	**0.1423**			
***0801 - 21**	0.0333	0.0462			
***0901**	0.0000	0.0038			
***1001**	0.0333	0.0231			
Total	0.0000	0.9999		0.9995	1.0000

les were B*0702-8 (0.1333) and B*1801-5 (0.1166), and ultimately, the highest occurrence rate of Cw*0701-10 (0.3000) and Cw*0602, 4 (0.1875) out of all HLA-Cw alleles tested was observed. Comparing allele frequencies in the investigated group of patients to those in the healthy population, only one significant deviation was found – the increased frequency of HLA-Cw*0602, 4 (0.1875 vs. 0.0733; odds ratio: 2.913; two-sided P value: P = 0.0251).

The occurrence rates of HLA class II alleles in the investigated group of patients are shown in Table 2. The most frequent alleles were DRB1*1501-8 (0.1500), DRB1*0101-2 (0.1500), DQB1*0201 (0.1666), and DQB1*0501 (0.1666). No significant differences in allele frequencies were found by comparison with the healthy population.

Discussion

H. pylori is now recognized to be an important factor in the development of peptic ulcer disease and in gastric carcinogenesis (Fuchs and Mayer, 1995; Marshall, 1995). Studies have shown that *H. pylori* lipopolysaccharide (LPS) mimics the Lewis blood group in structure. Because Lewis antigens are present in the gastric mucosa, it is believed that LPS camouflages the organisms and protects them from elimination by effector mechanisms of the immune system. The pathogen does not invade the mucosa and there are autoimmune mechanisms which are ultimately responsible for the development of pathological symptoms (Negrini et al., 1991; Walker, 1998).

The sanctuary site where *H. pylori* evades antimicrobial therapy is unknown, but considerable data exist about its intracellular location (Engstrand et al., 1997). The major protective immune response against intracellular bacteria is cell-mediated immunity, which consists of two types of reactions: killing by macrophages and lysis of infected cells by MHC class I restricted $CD8^+$ T cells. Eighty percent of the intraepithelial T-lymphocytes express $CD8^+$ (Roitt et al., 1996; James 1998). It has been shown recently that these $CD8^+$ T cells have a repertoire of T-cell receptors biased towards bacterial antigens (Nanno et al. 1988; James, 1998), which suggests that they may play an important role in protecting mucosal surfaces of the body and that class I HLA antigens may influence the susceptibility or resistance to *H. pylori* infection. This is in agreement with the findings that infection with *H. pylori* is associated with T_H1 response (Sommer et al., 1998). T-cell clones isolated from antral biopsies taken from *H. pylori*-infected patients displayed a cytokine secretion profile typical of T_H1 cells: they produce IFN-γ, TNF and IL-12 (D'Elios et al., 1997).

Dermatological disorders, such as *urticaria*, *rosacea*, etc., are associated with *H. pylori* infection in some patients (Kolibášová et al., 1994; Tebbe et al., 1996). The reason is not known; cross-reactive antigens, circulating immune complexes, and other mechanisms may be involved. For instance, *H. pylori* heat shock proteins (HSP) share determinants with host tissues (Engstrand et al., 1991). These cross-reactive antigens may elicit an autoimmune process that results in dermatologic disorders, too.

HLA antigens are very well known markers of susceptibility to autoimmune diseases (for review see Buc 1997; Svejgaard et al., 1997). Our results on HLA-Cw6 association to dermatological disorders associated with the *H. pylori* infection seem to be in agreement with the role of the cell-mediated immunity in the course of the disease. However, other independent studies are needed to confirm this finding.

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