Psoriasis is a chronic inflammatory disease characterised by T-cell-mediated hyperproliferation of keratinocytes. Psoriasis is a heterogeneous disease with distinct but overlapping phenotypes. The most frequent ones are chronic plaque (psoriasis vulgaris) and guttate psoriasis. The pathogenesis of psoriasis is unknown, but its genetic background is undoubted. Genetic factors include HLA class I and II associations. Genes responsible for the disease have not been identified yet, but recent genetic linkage studies have shown evidence that one or more genes located very close to the C locus may be responsible for the development of psoriasis (Trembath et al. 1997). The transported associated with antigen processing (TAP) genes located in the HLA class II region might also be involved in psoriasis pathogenesis.

Results of a recent study from the northern Polish cohort have shown that the frequency of TAP1*D allele is significantly increased in psoriatic patients compared to a control population, which indicates that this molecule could lead to genetic susceptibility to psoriasis in Poles (Witkowski-Toboła et al. 2004).

So far HLA-Cw6 has been the most frequently described correlation confirmed in various ethnic groups of psoriatic patients (Enerback et al. 1997; Gudjonsson et al. 2002; Henseler et al. 1985; Swanbeck et al. 1995). In particular an association of the HLA-Cw6 antigen with psoriasis with the early onset has been well documented in various cohorts (Enerback et al. 1997; Gudjonsson et al. 2003; Henseler and Christophers. 1985). Data on correlation between the clinical expression of...
psoriasis and the HLA-Cw6 allele are inconsistent. Ikəheimo (1996) has failed to show any connection between the occurrence of Cw6 and the clinical disease phenotype. Another study has revealed no clinical differences between psoriasis with early and late onset (Swanbeck et al. 1995). In contrast, Henseler and Christophers (1985) have shown that Cw6-positive patients are more likely to have the widespread and recurrent disease. A recent study of a large Icelandic population of psoriatics suggests that patients with psoriasis have various clinical features, which depend on whether they are HLA-Cw6-negative or positive and that a more severe form of the disease is correlated with Cw6 expression. The most striking association was observed between Cw6 expression and acute guttate attacks or eruption of small papules around chronic plaque psoriatic lesions (Gudjonsson et al. 2002). The strong correlation of the Cw6 allele with early onset psoriasis vulgaris has also been proved in the Polish population (Luszczech et al. 2003; Szczerkowska-Dobosz et al. 1996). However, none of those studies analysed other clinical features of psoriasis in respect of Cw6 allele expression.

Hence in this report we studied the association of the HLA-Cw*06 allele (identified by DNA typing with sequence-specific primers), with chosen clinical parameters of psoriasis, its extent and severity. Seventy-eight patients with psoriasis vulgaris, attending the Dermatology Department, Medical University of Gdañsk participated in this study. The mean age of the group was 35 years (range 18-74). The mean duration of the disease was 8 years. A family history was obtained for each patient, and was considered positive if a first, second or third-degree relative had psoriasis. Patients were subdivided into two groups: with onset before the age of 40 (62 patients) and with onset after the age of 40 (16 patients). The patients represented two clinical patterns of the disease. Fifty patients had chronic, stable psoriasis with psoriatic plaques increasing slowly with time. The remaining 28 patients had an acute or eruptive psoriasis with an outbreak of red papules disseminated between chronic plaques. Patients with psoriatic arthritis and guttate psoriasis were not included in the study. A system of disease severity evaluation the PASI score (Psoriasis Area and Severity Index) was applied to each patient. This scoring system is based on the quantitative evaluation of three typical signs of psoriatic lesions: erythema (redness), infiltration (thickness) and desquamation (scaling) on a scale of 0 to 4, combined with the skin surface area involved. The area of erythema, induration and desquamation was scored in four anatomic sites: head, arms, trunk and legs. The average value from three evaluations was calculated. The control group consisted of 70 disease-free, unrelated individuals from the same ethnic background (northern Poland), whose blood was taken for paternity testing at the Department of Forensic Medicine, Medical University of Gdañsk. All patients and the control group were Caucasians. DNA was extracted from EDTA-treated blood by a non-organic method (Lahiri et al. 1991). The yield and purity of DNA were checked by measuring absorbance at 260 and 280 nm. HLA-Cw*06 alleles were recognised according to the HLA nomenclature from November 1999. The dynal classic SSP HLA Cw*06 standard kit (Deutsche Dynal GmbH, Germany) was used in the study. PCR was performed in a Masterecyler thermocycler (Eppendorf, Germany). The products were separated by 1.5% agarose gel electrophoresis in 1 x TBE buffer and stained with ethidium bromide. The HLA-Cw*06 alleles were identified by the presence of PCR products in UV illumination.

The frequency of the Cw*06 allele was significantly higher in psoriatic patients than in the control group. As many as 44 patients (56%) carried the HLA-Cw*06 allele, compared with 19% of the control group, giving a relative risk of 5.67 (\( p < 0.0001, \chi^2 \)). Table 1 presents a comparison of the chosen clinical parameters of psoriasis among Cw*06-positive and Cw*06-negative patients.

<table>
<thead>
<tr>
<th>Psoriasis type</th>
<th>Cw6 (+)</th>
<th>Cw6 (−)</th>
<th>P value (chi-square)</th>
</tr>
</thead>
<tbody>
<tr>
<td>early onset</td>
<td>39</td>
<td>23</td>
<td>0.0462</td>
</tr>
<tr>
<td>late onset</td>
<td>5</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>positive family history</td>
<td>32</td>
<td>10</td>
<td>0.0001</td>
</tr>
<tr>
<td>acute, eruptive chronic plaque</td>
<td>19</td>
<td>9</td>
<td>0.1271</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>25</td>
<td></td>
</tr>
</tbody>
</table>

The Cw*06 allele frequency was significantly higher in the early onset group of patients, compared to the late onset group (\( p < 0.05, \chi^2 \)). The HLA-Cw*06 was detected in 39 out of 62 patients with the early onset type of the disease (51%), and in 5 out of 16 patients with late onset (31%) and this difference is statistically signifi-
cant. In total, 42 out of 78 patients had a positive family history. Familial occurrence of psoriasis was significantly higher in Cw*06-positive patients than in Cw*06-negative patients (p < 0.001, \( \chi^2 \)). Comparison of Cw*06 allele frequency in two clinical subtypes of psoriasis (active, eruptive versus chronic, plaque psoriasis), revealed no statistically significant differences between the two groups. The Cw*06 allele was detected in 19 out of 28 patients with the active, eruptive type of the disease and in 25 out of 50 patients with chronic, plaque psoriasis. The PASI scores were significantly higher among Cw*06-positive patients than in the Cw*06-negative group (median values: 17.3 versus 9.7; p < 0.0001, median test).

There are several reports on Cw6 association with psoriasis vulgaris (Brenner et al. 1978; Economidou et al. 1985; Enerback et al. 1997; Gudjonsson et al. 2003; Ikáheimo et al. 1996; Tiilikainen et al. 1980; Szczerkowska-Dobosz et al. 1996). We found Cw*06 frequency to be significantly higher in psoriatic patients than in the control group, which is in agreement with other cohorts’ results, as well as with earlier studies of the northern Polish population (Szczerkowska-Dobosz et al. 1996). We found Cw*06 frequency to be significantly higher in psoriatic patients than in the control group, which is in agreement with other cohorts’ results, as well as with earlier studies of the northern Polish population (Szczerkowska-Dobosz et al. 1996). In this study for Cw*06 allele detection we used the PCR-SSP technique, which is more specific and sensitive than serologic methods. Serology is especially ineffective in HLA-C typing because up to 50% of the population carry serologically undetectable, undefined HLA-C alleles, i.e. so-called Cw “blank” alleles (Bunce et al. 1996). In the present study with the use of PCR methods we confirmed a significant correlation of the Cw*06 allele with the early onset type of psoriasis. Familial occurrence of psoriasis has been reported to range from 2% to 91% (Farber 1974). In our study the positive familial history was observed in 42 patients; the majority of them were Cw6 carriers. These results are in accordance with earlier studies, which pointed out a strong association between Cw6 expression and familial occurrence of psoriasis (Gudjonsson et al. 2002; Henseler and Christophers 1985).

In the present study we subdivided patients basing on the predominant character of skin changes into two clinical subgroups: with the chronic, plaque or the active, eruptive form of the disease. To the latter group, we enrolled patients, who according to Brenner’s definition (1978) of eruptive psoriasis had chronic plaques coexisting with disseminated red papules. Active, eruptive psoriasis was more frequent among Cw*06 carriers than among patients lacking this allele. However, probably due to the low number of patients, we failed to show a statistically significant correlation between Cw6 allele frequency in those two types of the disease. This could be also explained by the exclusion from the study of patients with classic guttate psoriasis, which is, by definition, an acute, widespread eruption of papules (with no plaques) associated with group A \( \beta \)-haemolytic streptococcal infections.

To assess the extent and severity of psoriasis, we used the Psoriasis Area and Severity Index (PASI), which is applied in routine clinical work (Fredriksson 1978). The PASI score is not an optimal general dermatological scoring system for psoriasis, but its application by an experienced physician can be helpful in assessment of disease severity (Van de Kerkhof 1997). The results of our study show that patients with Cw6 allele have a statistically higher PASI score, as compared with Cw6-negative patients. This suggests that the Cw6 molecule tends to be related to the more severe form of psoriasis.

In summary, our investigation showed that the Cw*06 molecule is positively associated with psoriasis vulgaris of the early type with familial occurrence and its more severe form. This confirms that the allele could be involved in the genetic background of this common disease.

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**REFERENCES**


