**SHORT COMMUNICATION**

**T-lymphocyte subsets in adult coeliac disease**

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**Summary**

1. As a defect of suppressor function has been hypothesized in the pathogenesis of coeliac disease, we measured, by monoclonal antibodies, the inducer/suppressor T-cell ratio in adult coeliac disease.

2. No statistical difference was observed between coeliac patients and healthy controls, irrespective of treatment and HLA status.

3. These results do not show an imbalance in the inducer/suppressor T-cell ratio in coeliac disease.

Key words: coeliac disease, HLA-B8, suppressor function, T-lymphocyte subsets.

**Introduction**

An imbalance among T-lymphocyte subsets has been considered in the pathogenesis of a number of immunological disorders [1]. In coeliac disease a defect of suppressor T-cell function has been proposed as the fundamental abnormality in the afferent limb of the immune response to gluten [2]; however, the proportion of various T-lymphocyte subsets has not been evaluated. Therefore we investigated, by T-cell typing with monoclonal antibodies, T-cell subpopulations in adult coeliac disease.

**Methods**

Thirty-three (12 untreated and 21 on a gluten-free diet) biopsy proven adult coeliac patients and 26 age-matched healthy controls were studied. Both patients and controls were unselected. As all the controls were shown to be HLA-B8 negative, five more healthy subjects known to be HLA-B8 positive were added to the study.

Mononuclear cells obtained on a Ficoll/Hypaque sedimentation gradient were stained for membrane antigens by indirect immunofluorescence with murine monoclonal antibodies (OKT4 reacting with T-cells of inducer type and OKT8 reacting with T-cells of cytotoxic/suppressor type; Ortho Pharmaceuticals, Raritan, NJ, U.S.A.) and then counted under fluorescence microscopy [3].

The HLA status was determined by a modification of the microlymphocytotoxic technique of Terasaki & McClelland [4].

The statistical analysis was done by Wilcoxon's rank sum test for unpaired data.

**Results**

The results expressed as inducer/suppressor cell ratio are shown in Fig. 1. No significant difference was observed between either treated (mean 1.5, range 0.5–4.5) or untreated (mean 1.5, range 0.7–3.0) coeliac patients and controls (mean 1.6, range 1.1–2.1), or between treated and untreated coeliac patients. Moreover, no significant difference was observed between HLA-B8 positive (mean 1.5, range 0.5–4.5) and HLA-B8 negative (mean 1.5, range 0.7–2.3) coeliac patients, or between HLA-B8 positive (mean 1.6, range 1.4–1.8) and HLA-B8 negative (mean 1.5, range 1.1–2.1) healthy controls. Only four coeliac patients showed an inducer/suppressor T-cell ratio above the upper limit of the control range, and, of these, three had an associated autoimmune diabetes.
exists in adult coeliac disease, it does not seem to be related to an imbalance of the inducer/suppressor lymphocytes with different phenotypic features, affairs. Therefore, if a defect of suppressor function immune diabetes is secondary to this state of condition in which an altered OKT4/OKT8 ratio three had an associated autoimmune diabetes, a associated with coeliac disease and that the autoimmune diabetes rather than coeliac disease per se. However, in our series the presence of HLA-B8 was not associated with an altered OKT4/OKT8 ratio, suggesting that in coeliac disease a quantitative imbalance of these lymphocyte subsets is not a feature of the immunological pattern of HLA-B8 positive subjects.

Discussion

An impairment of suppressor activity after stimulation with concanavalin A has been reported in adult coeliac patients on a gluten-free diet [5]. Our results, however, do not show any significant difference between coeliac patients and controls as far as the OKT4/OKT8 ratio is concerned. Only four coeliac patients showed a ratio above the upper limit of the control range and, of these, three had an associated autoimmune diabetes, a condition in which an altered OKT4/OKT8 ratio has been reported [6]. It is likely that in them diabetes rather than coeliac disease may be responsible for the observed heightened ratio; however, we cannot exclude that the heightened ratio is associated with coeliac disease and that the autoimmune diabetes is secondary to this state of affairs. Therefore, if a defect of suppressor function exists in adult coeliac disease, it does not seem to be related to an imbalance of the inducer/suppressor T-cell ratio. T-cell typing assesses and estimates lymphocytes with different phenotypic features, and stimulation with concanavalin A evaluates the overall suppressor function, where factors other than inducer/suppressor balance may play a role [7, 8]. It is not surprising then that conditions may exist in which suppressor function is not directly related to the OKT4/OKT8 ratio, as recently reported in chronic liver diseases [9].

Robertson et al. [5] found a significant association between low suppressor function and the presence of HLA-B8 in coeliac disease. This association has been found in Graves's disease [10] and myasthenia gravis [11], raising the possibility that abnormalities of suppressor function in coeliac disease are related to the high incidence of HLA-B8 rather than coeliac disease per se. However, in our series the presence of HLA-B8 was not associated with an altered OKT4/OKT8 ratio, suggesting that in coeliac disease a quantitative imbalance of these lymphocyte subsets is not a feature of the immunological pattern of HLA-B8 positive subjects.

References