Correspondence

Conjunctival epithelial cells infected with *Chlamydia trachomatis* express HLA-DR antigens

SIR, The major histocompatibility complex (MHC) class II products or HLA-DR antigens are cell surface glycoproteins concerned with antigen presentation and the elicitation of T lymphocyte-dependent immune responses. The class II products are predominantly expressed on immune competent cells such as B lymphocytes, activated T lymphocytes, macrophages, and Langerhans cells.[1] In addition a variety of epithelial cell types are now known to express HLA-DR antigens.[2]

In an immunopathological study of trachomatous conjunctivitis we recently observed that the superficial conjunctival epithelial cell layers express HLA-DR antigens.[3] In order to investigate in detail the topographical relationship between the distribution pattern of HLA-DR antigens on the one hand, and the presence of chlamydial antigens on the other, we performed a double staining technique. HLA-DR antigens were demonstrated with a three-step indirect immunoperoxidase procedure using TAL-1B_5 monoclonal antibody,[4] and chlamydial antigens were subsequently visualised in the same sections with fluorescein labelled monoclonal antibodies (BioMerieux, France). Chlamydial antigens were found in variable numbers of superficial epithelial cells, which constantly coexpressed HLA-DR antigens (Fig. 1).

Coexpression of infectious and HLA-DR antigens has previously been reported in human hepatitis[5] and in infections by murine hepatitis[6] and visna-maedi viruses.[7]

The mechanism by which the conjunctival epithelial cells are induced to synthesise these antigens is as yet unclear. Interferon-gamma, released by activated T lymphocytes in the epithelium, may be responsible, since this lymphokine has been shown to induce HLA-DR synthesis and expression in a variety of epithelial cells in vitro.[8,9] Alternatively, and by analogy with the murine hepatitis virus,[6] the chlamydial agent itself could play a causative role.

HLA-DR expression by the conjunctival epithelial cells may render these cells capable of acting as antigen-presenting cells for T lymphocytes, thereby enhancing the immune response and possibly inducing an autoimmune reaction.[10] Alternatively, expression of HLA-DR antigens may render the infected epithelial cells susceptible to

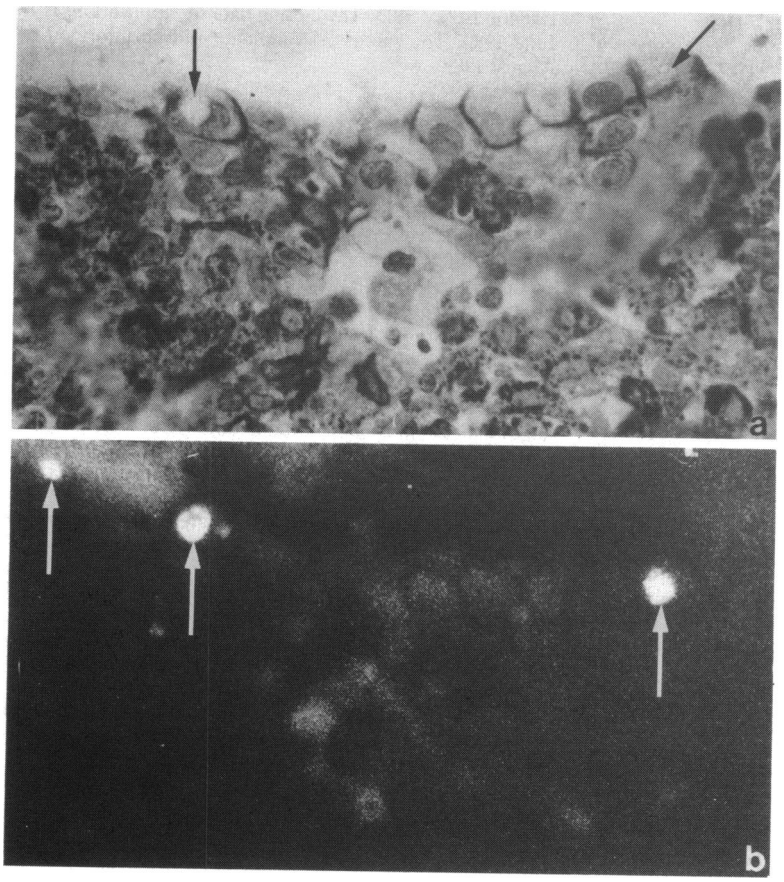

Fig. 1a: *TAL-1B_5 staining in trachomatous conjunctivitis. Many epithelial cells in the superficial layer are HLA-DR$^+$. Some of them contain Halberstaedter-Prowazek inclusion bodies seen as pale vacuoles (arrows).* b: *The same section, stained for chalmydial antigens, shows three brightly fluorescing inclusions, indicated with arrows, in the HLA-DR$^+$ epithelial cells. Faint fluorescence of the surrounding structures is due to the counterstaining. (Conjunctival specimen stained with a three-step indirect immunoperoxidase procedure for monoclonal antibody TAL-1B_5, and* (b) *subsequently incubated with fluorescein labelled antichlamydial monoclonal antibodies ×480.)*

immunological attack by HLA-DR-restricted cytotoxic T cells.[11]

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Notes

Research grants

The *David Cole travel fellowship*, instituted by Merck Sharp and Dohme in memory of Professor David Cole, will assist a visit to a hospital or research centre during the academic year starting 1 October 1989. The award will be equivalent to £2000. The purpose of the award is to enable the successful applicant to gain experience and knowledge in pursuit of a specific project related to glaucoma.

The Glaucoma Group Research grant, sponsored by International Glaucoma Association, will be available for a research project clinically orientated to glaucoma for 1989. The award will be equivalent to £2500. The grant may be used towards salary or project expenses or for buying equipment.

Both these awards are available to medical graduates and non medical scientists resident in the United Kingdom or Republic of Ireland. They may be held concurrently with other awards. Further details and application forms from Dr S Nagasubramanian, Secretary Glaucoma Group, Glaucoma Unit, Moorfields Eye Hospital, City Road, London EC1V 2PD. The closing date for applications is 15 June 1989. The successful candidate will be informed by August 1989.

Association for Eye Research

The thirtieth meeting of the AER will be held in La Grande Motte, near Montpellier, France, on 3–7 October 1989. Further information from the Programme Secretary, Dr John Tiffany, Nuffield Laboratory of Ophthalmology, Walton Street, Oxford OX2 6AW. The deadline for abstracts is 31 May 1989. All proceedings will be in English. The AER Publication Award will be made at this meeting for the best paper in any language, by either members or non-members of AER, published in the 12 months preceding the meeting. Eligible authors must be under the age of 35. In the case of multiauthored papers the award will be made to the main author, and the entry must be accompanied by a statement signed by each author, indicating who this is. The work described must relate to the eye and be original or based on a previously evolved technique or method. (If the paper is not in English, it must be accompanied by a translation of captions, results, and conclusions in sufficient detail to permit assessment.) Nominations (eight reprints or eight copies of the manuscript accepted for publication) must be made through a member of the AER to the General Secretary: Professor Martin Reim, Abtlg. Augenheilkunde, Med. Fakultät der RWTH Aachen, Klinikum, Pauwelsstrasse, D-5100 Aachen, West Germany, not later than 1 July 1989.