isolated from the cervix of nine of the forty pregnant women in group II (23%) and from the ejaculates of six of the twenty-three men in group III (26%). "Conventional" mycoplasmas, mostly *M. hominis*, were isolated only occasionally from cervical mucus or ejaculates. The difference in incidence of T-mycoplasmas between infertile (group I) and fertile patients (groups II and III) was found highly statistically significant ($P < 0.001$).

After an observation period of 3 months both husbands and wives in group I were treated with doxycycline in recommended dosage for 10 days between the seventh and the sixteenth day of the menstrual cycle in order to eradicate the T-mycoplasmas and to evaluate if this had any influence on the patients with regard to their fertility status or not. If no pregnancy ensued the treatment was repeated during the next two months at the same period of the menstrual cycle. During the fourth and fifth month the patients were treated with doxycycline in increased dosage: 200 mg daily from the seventh to the sixteenth day. If pregnancy was reported, no further treatment was given.

In the ten males and eleven females studied in detail the concentrations of doxycycline in serum varied between 0.4 and 1.35 μg ml.$^{-1}$ 12–14 h after the last dose of doxycycline, while those in ejaculates varied between 0.2 and 1.8 μg ml.$^{-1}$ (mean value 0.95 μg ml.$^{-1}$). Most of the isolated T-mycoplasmas were inhibited by doxycycline in concentrations below 0.1 μg ml.$^{-1}$ although some of the isolates had MIC:s up to 0.6 μg ml.$^{-1}$. The T-mycoplasmas disappeared from all ten males and from all but two of the eleven women studied in detail.

The patients were observed carefully for 5 months. After 3 months pregnancies had been reported in twelve couples. After 5 months another three pregnancies were reported.

It is suggestive that 29% of the women in group I became pregnant within a few months after the eradication of their T-mycoplasmas, after their earlier longstanding infertility. The results of this investigation therefore support the suggestion that at least some T-mycoplasmas may be of importance in some cases with reproductive failure where no other cause is detected.

These findings are now being further investigated.

HÅKAN GNARPE

Institute of Medical Microbiology,
University of Uppsala

JAN FRIBERG

Department of Obstetrics and Gynaecology,
University Hospital,
Uppsala

Received October 30; revised November 24, 1972.

1 Braun, P., Lee, Y.-H., Klein, J. O., Marcy, M., Klein, T. A., Charles, D., Levy, P., and Kass, E. H., *New Engl. J. Med.*, **284**, 167 (1971).
2 Kundsin, R. B., Driscoll, S. G., and Ming, P.-M., *Science*, **157**, 1573 (1967).
3 Kundsin, R. B., and Driscoll, S. G., *Ann. NY Acad. Sci.*, **174**, 794 (1970).
4 Kundsin, R. B., and Driscoll, S. G., *Surg. Gynecol. Obstet.*, **131**, 89 (1970).
5 Caspi, E., Herczeg, E., Solomon, F., and Sompolinsky, D., *Amer. J. Obstet. Gynecol.*, **111**, 1102 (1971).
6 Chanock, R. M., Hayflick, L., and Barlie, M. F., *Proc. US Nat. Acad. Sci.*, **48**, 41 (1962).
7 Shepard, M. C., and Lunceford, D. D., *Appl. Microbiol.*, **20**, 539 (1970).

Human Lymphocyte Antigen Association in Ankylosing Spondylitis

A STUDY of human lymphocyte phenotypes in unrelated Caucasian individuals with ankylosing spondylitis has revealed a striking similarity in their antigenic pattern. The lymphocytes of fifty such patients, when tested against a panel of twenty-six different specific typing sera, using a two stage lymphocytotoxicity micro-method, were shown to have in common either the antigen HL-A27 (96%) or the antigen W5 (4%). This remarkably high frequency of the antigen HL-A27 compares with the incidence of 5–6% of this antigen in random Caucasian populations.

Antigen HL-A27 has been shown to have a higher frequency among certain "isolated" communities such as the Pima Indians (10%), (ref. 1). Among these Indians, there is a higher incidence of ankylosing spondylitis than in a random Caucasian population[2], being 5.9% compared with 0.5% or less in the latter group. Likewise, there is a low frequency of this antigen among Negroid populations (0–1%) among whom there is a very low incidence of this disease[3]. These findings support the observation above, details of which will be reported later.

Significant associations between the human lymphocyte phenotype and groups of patients with various diseases involving the lymphatic system have been reported by some authors. The antigens HL-A5, W5 and W18 are found more frequently in patients with Hodgkin's disease[4–6]. Systemic lupus erythematosus is associated with HL-A8 and W15[7,8], adult coeliac disease with HL-A1 and HL-A8[9] and HL-A13 and possibly W17 with psoriasis[10]. HL-A9 and HL-A12 are more frequent in patients with chronic glomerulo-nephritis[11]. The results of two separate studies of patients with rheumatoid arthritis were, however, conflicting and inconclusive[12,13]. An increased frequency of HL-A27 (17%) has been noted in patients with lymphoblastic leukaemia[14]. This is perhaps of relevance to ankylosing spondylitic patients as some of them did develop leukaemia after radiotherapy.

It appears therefore that various HL-A phenotypes are associated with certain diseases. We also postulate that ankylosing spondylitis may be mediated partly by histocompatibility influenced immune responses.

We thank Drs Brewerton and Hart and their patients, and their registrars, Drs A. Nicholls and R. Sturrock. M. F. P. C. thanks the Governors of Westminster Hospital for a grant.

MAEVE F. P. CAFFREY
D. C. O. JAMES

Tissue Typing Laboratory,
Blood Transfusion Unit,
Westminster Hospital,
London SW1

Received January 12, 1973.

1 Amos, D. B., *Fifth International Histocompatibility Testing Workshop*, Evian, May 1972.
2 Gofton, J. P., Bennett, P. H., Smythe, H. A., and Decker, J. L., *Ann. Rheum. Dis.*, **31**, 474 (1972).
3 Baum, J., and Ziff, M., *Arthritis Rheumatism*, **13**, 305 A (1970).
4 Amiel, J. L., *Histocompatibility Testing 1967*, 79 (Munksgaard, Copenhagen, 1967).
5 Zervas, J. D., Delamore, I. W., and Israels, M. C. G., *Lancet*, ii, 634 (1970).
6 Falk, J., and Osoba, D., *Lancet*, ii, 1118 (1971).
7 McDevitt, H. O., and Bodmer, W. F., *Amer. J. Med.*, **52**, 1 (1972).
8 Walford, R. L., Smith, G. S., and Waters, H., *Transplant. Rev.*, **7**, 78 (1971).
9 Stokes, P. L., Asquith, P., Holmes, G. K. T., Mackintosh, P., and Cooke, W. T., *Lancet*, ii, 162 (1972).
10 Russell, T. J., Lorain, M. D., Schults, M., and Kuban, D. J., *New England J. Med.*, **287**, 738 (1972).
11 Mickey, M. R., Kreisler, M., and Terasaki, P. I., *Histocompatibility Testing 1970*, 237 (Munksgaard, Copenhagen, 1970).
12 Kueppers, F., Brackertz, D., and Mueller-Eckhardt, C. H., *Lancet*, ii, 1425 (1972).
13 Seignalet, J., Clot, J., Sany, J., and Serre, H., *Vox Sanguinis*, **23**, 468 (1972).
14 Lawler, S. D., and Klouda, P. T., *Brit. J. Haematol.*, **21**, 595 (1971).