products' in ankylosing spondylitis denotes complement consumption and may represent an altered immune response to a persistent antigen in this disease.

References

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—, HOBBS, J. R., AND HOLT, P. J. L. (1973) *Ann. rheum. Dis.*, 32, 557 (Complement metabolism in rheumatoid arthritis. I. Longitudinal studies)

Lymphocyte proliferation to artery antigen as a positive diagnostic test in polymyalgia rheumatica. By B. L. HAZLEMAN, I. C. M. MACLENNAN, and M. M. ESIRI (Oxford). Published in full in the *Annals*, 1975, 34, 122.

Evidence of impaired cell mediated immunity in the sero-negative arthritides. By R. D. STURROCK, K. FROEBEL, R. N. M. MACSWEEN, and W. C. DICK (Centre for Rheumatic Diseases, Royal Infirmary and Department of Pathology, Western Infirmary, Glasgow)

Lymphocyte responses to plant mitogens have previously been reported to be depressed in ankylosing spondylitis (Escanilla, Alepa, and Reeve, 1970). In view of this, lymphocyte responses to phytohaemagglutinin (PHA), Concanavilin A (Con A), and poke-weed mitogen (PWM) have been studied in twenty patients with ankylosing spondylitis, twelve patients with Reiter's disease, and eleven patients with psoriatic arthritis*. The results were compared with a group of fourteen normal controls and twenty patients with osteoarthritis. Skin testing was performed using 40 units streptokinase-streptodornase varidase and dinitrochlorobenzene sensitization. The Table summarizes the effect of the plant mitogens on lymphocytes from the various groups. PHA responses at a submaximal dose were significantly depressed in the ankylosing spondylitic and Reiter's groups and occurred particularly in those patients with clinically severe disease. An increased response occurred in the psoriatic group at higher doses of PHA and a similar effect was observed in response to PWM among the Reiter's patients. No correlation was found between *in vivo* skin testing and *in vitro* lymphocyte response, and the pattern of skin

* None of these patients were on anti-inflammatory treatment at the time.

reaction to dinitrochlorobenzene and streptokinase-streptodornase varidase was similar in all groups tested.

It is concluded that the impaired lymphocyte responsiveness to the plant mitogens tested may reflect an alteration of lymphocyte function in this group of diseases. Further work is necessary to establish whether this is a quantitative or qualitative defect.

Reference

ESCANILLA, F., ALEPA, P. F., AND REEVE, W. (1970) *Arthr. and Rheum.*, 13, 314

A serial study of eosinophilia and raised IgE antibodies during gold therapy. By P. DAVIS and G. R. V. HUGHES (Post-graduate Medical School, Hammersmith Hospital, London)

Gold therapy is known to be effective therapy for rheumatoid arthritis (RA) but to be associated with a high incidence of mucocutaneous side effects (Empire Rheumatism Council, 1961). Little is known about the mechanism of gold reactions nor is there a satisfactory method of monitoring gold reactions. A recent report has shown that patients with gold reactions also have raised levels of IgE antibodies (Davis and others, 1973) and that this was usually associated with raised eosinophil counts. The aim of this study was to perform serial IgE levels and eosinophil counts in patients receiving gold for arthritis and to correlate raised levels with gold reactions.

Case and methods 47 patients with RA and 3 patients with psoriatic arthritis have been studied. All patients were receiving gold in a standard regimen and had normal eosinophil counts and IgE levels before starting their therapy. Patients with a known atopic history were excluded. Eosinophil counts were measured by routine full blood counts and IgE levels by the radioimmuno-sorbent technique.

Results 14 patients developed a reaction to their gold therapy, i.e. rash, pruritus, or mouth ulcers. 11 of these patients had concurrent eosinophilia which preceded the side effect in 6 instances. In addition, a further 9 patients had eosinophilia without clinical side effect. IgE levels were raised in 22 of the 50 patients receiving gold therapy, the relationship to eosinophilia and clinical side effect is shown in the Table.

Table Lymphocyte responses to PHA, PWM, and Con A found to be significantly different ($P < 0.05$) from normal

	Ankylosing spondylitis	Psoriatic arthritis	Reiter's	Osteoarthritis	Normals
PHA (2.5 µg/ml)*	$\bar{x} = 15928 \pm 3479$ $\dagger t = 2.84$ $P < 0.05$		$\bar{x} = 19473 \pm 4007$ $t = 2.098$ $P < 0.05$		$\bar{x} = 31370 \pm 4175$
PHA (500 µg/ml)*		$\bar{x} = 32921 \pm 4461$ $t = 5.12$ $P < 0.005$	$\bar{x} = 33268 \pm 3991$ $t = 2.947$ $P < 0.01$		$\bar{x} = 21397 \pm 1604$
Con A (250 µg/ml)*				$\bar{x} = 13641 \pm 2099$ $t = 2.416$ $P < 0.005$	$\bar{x} = 30130 \pm 6847$
PWM (1:10)*			$\bar{x} = 28138 \pm 3601$ $t = 3.218$ $P < 0.01$		$\bar{x} = 16015 \pm 1632$

* The concentration of the stock solution of mitogen. The final concentration in the culture medium is 10% of the stock solution.
† The mean dpm per culture for the group $\pm$ SEM.
‡ The values of t and P are derived using Student's 't' test.

	<i>Eosinophilia alone</i>	<i>Eosinophilia + side effect</i>	<i>Side effect alone</i>	<i>No side effect or eosinophilia</i>
Raised IgE	9	8	2	3
Normal IgE	0	3	1	24

Resolution of side effect and fall of eosinophil counts and IgE levels were monitored in all patients on stopping gold. In 4 patients where gold therapy was reinstituted after a reaction, eosinophilia and raised IgE levels reappeared. Serial serum gold levels performed on all patients throughout the study confirmed the finding (Jessop and Johns, 1973 that these did not correlate therapeutic affect or the development of side effects to gold therapy.

It is concluded that these results support the concept that dermatological reactions to gold salts are mediated by a type 1 hypersensitivity response and that serial measurements of eosinophil counts and IgE levels may be a useful therapeutic guide to their development.

References

Empire Rheumatism Council (1961) *Ann. rheum. Dis.*, 20, 335
DAVIS, P., EZEKOE, A., MUNRO, J., HOBBS, J. R., AND HUGHES, G. R. V.
(1973) *Brit. Med. J.* 3, 679
JESSOP, J. D., AND JOHNS, R. G. S. (1973) *Ann. rheum. Dis.*, 32, 228

November 28–29 Annual General Meeting, Royal College of Physicians, London.

Notes

Articular Cartilage Symposium

Imperial College, London, September 2–6, 1974

The symposium was organized by Drs. G. E. Kempson, A. Maroudas, and B. O. Weightman for the Biomechanics Unit, Imperial College. There were 80 participants in the six sessions on the structure of articular cartilage, chemistry of matrix, metabolism, enzymes and degradation, functional properties, and pathogenesis of osteoarthritis. The symposium will be published by the *Annals* as Supplement 2 to Volume 34.

Heberden Society

Programme, 1975

May 23 Clinical Meeting, Taplow, Berks.
June 1–6 8th European Rheumatology Congress, Helsinki, Finland.
October 3–4 Joint meeting with the Spanish Society of Rheumatology, Madrid.

Immunoglobulin levels during rubella in children suffering from Still's disease. By A. HOWARD, B. M. ANSELL, and R. POWELL (*The MRC Rheumatism Unit, The Canadian Red Cross Memorial Hospital, Taplow, Maidenhead, Berkshire*)

Previous work has shown that in children suffering from active Still's disease there is an increase in all the immunoglobulin levels, particularly IgG and IgA, which are significantly higher than in the control healthy children.

Ten inpatients, 9 girls and 1 boy, with active Still's disease developed rubella with a typical rash and significant rise in antibody titre. In all the immunoglobulin levels were known before the infection; further samples were obtained during the rubella and approximately weekly afterwards for up to 12 weeks. All the children showed an increase in IgM which began at day one or two in three of them, and in the remainder was evident by the end of the first week; it was maximum between the first and second weeks, rising to about three times normal and then fell steadily over the next 5 weeks. The IgG levels rose steadily from the infection reaching a maximum at 5 to 6 weeks, about the time the IgM was falling away. By 12 weeks the IgM was at preinfection levels in all and the IgG had returned to their previous levels in all except three.

From this small study, it appears that infection with the rubella virus in children suffering from Still's disease causes the same antibody response as found in healthy persons.